Proceedings

9th VETERINARY PARAVETERINARY & SASVEPM CONGRESS 2017

24-27 JULY 2017

BIRCHWOOD HOTEL & OR TAMBO CONFERENCE CENTRE

SAVA
South African Veterinary Association
Suid-Afrikaanse Veterinêre Vereniging

Southern African Society for Veterinary Epidemiology & Preventive Medicine

South African Association of Veterinary Technologists

VVSA VNASA

9th VETERINARY PARAVETERINARY & SASVEPM CONGRESS 2017

PROCEEDINGS OF THE 9TH VETERINARY, PARAVETERINARY AND SASVEPM CONGRESS 24-27 JULY 2017 BIRCHWOOD HOTEL & OR TAMBO CONFERENCE CENTRE

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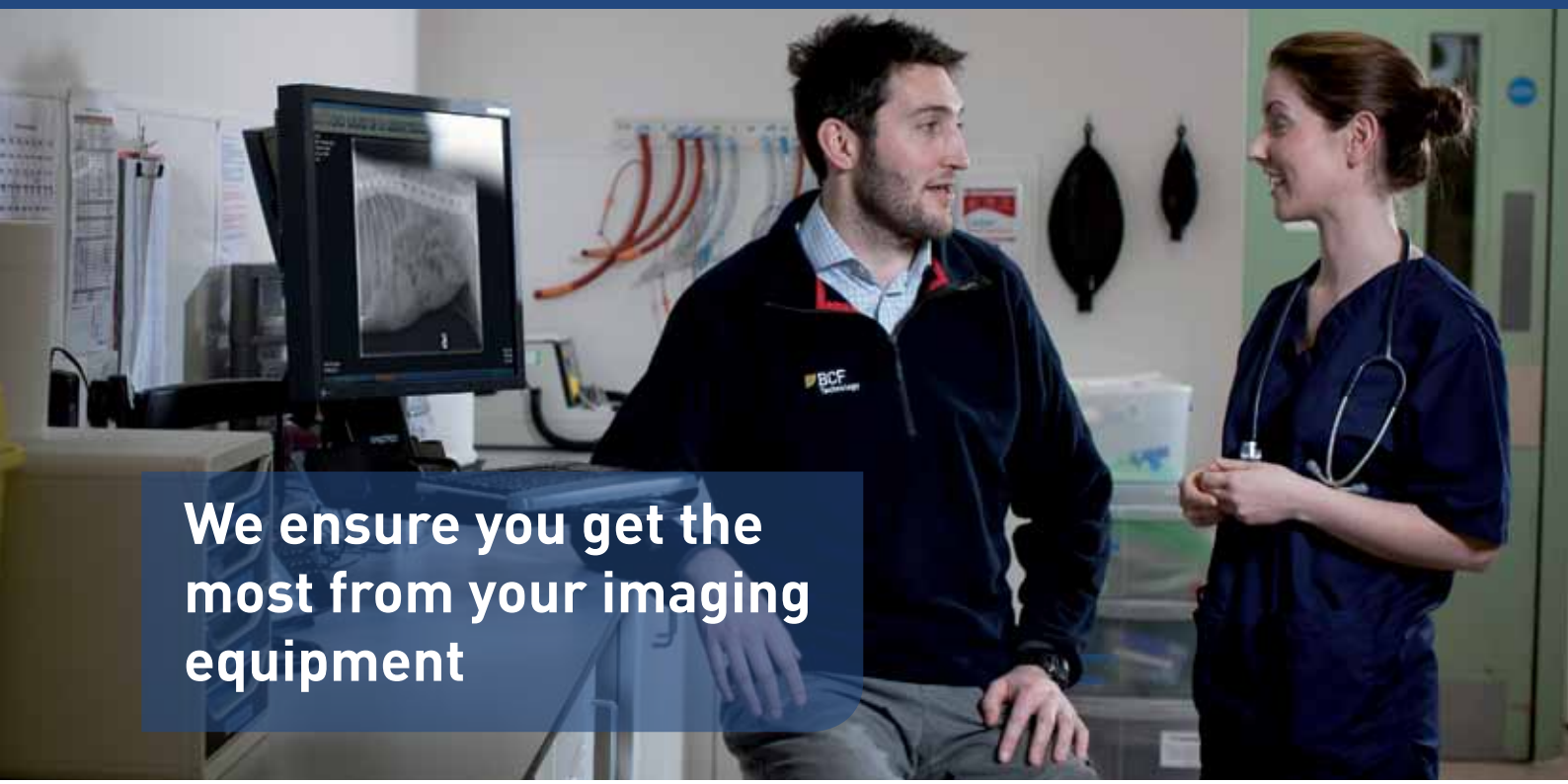

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PROGRAMME

Monday 24 July 2017

16:00-19:00	Congress Registration from 16:00-19:00
18:00	Opening of exhibition and cocktail function Explicitly for registered 9th SA Veterinary and Paraveterinary and SASVEPM 2017 Congress delegates

Tuesday 25 July 2017

Time	Small Animal Medicine	Critical Care	Small Animal Surgery	Production Animals	Wellness & Practice Management	Animal Welfare	Behaviour	Nurses	SASVEPM	Vet Techs/ Technicians/ SAALAS
Venue	Auditorium	Cape Town International	Macro Polo	Grand Central	Wonderboom	Virginia	Waterkloof	Barcelona	King Shaka	Charles de Gaulle
8.30-9.10	Recognising endocrine disease ♦ (Remo Lobetti)	Fluid therapy (Kenneth Joubert)	Basics of fracture repair (Greg Irvine Smith)	An unusual case of rabies in sheep in the North West Province, South Africa (Ken Pettey)	Practice Management 101: Finances ♦ (Henry Annandale)	Legal protection of animals (Bonita Meyersfeld)	Canine aggression: Interdog aggression (Frederique Hurly)	Delving deeper into bite wounds (Dr Ross Elliot)	Private-Public partnerships in veterinary services (Botlhe Michael Modisane)	African horse sickness virus evolutionary dynamics (A van Schalkwyk) Serotype- Specific RT- PCR and sequencing for discriminating between vaccine and field African horse sickness viruses (A van Schalkwyk)
9.15-10.00	Interpretation of endocrine tests (Johan Schoeman)	Fluid therapy – continued (Kenneth Joubert)	Basics of external skeletal fixation (Fanie Naude)	Creating rabies awareness in a rural community (Quixi Sonntag)	"Sleep Deprivation" and the debilitating impact on optimal performance among veterinarians – A practical guide to understanding sleep architecture, sleep disorders and remedial interventions♦ (Hermann Liebenberg)	NSPCA and animal protection legislation (Marcelle Meredith)	Canine aggression – Part 2: Human directed aggression and risk assessment (Frederique Hurly)	To suture or not to suture? (Dr Ross Elliot)	A framework for targeted allocation of resources (Bruce Gummow) Clinical presentation of cases in veterinary behaviour practices in South Africa and association with breed (Tinashe Zangure)	Salivary gland transcriptome of *Rhipicephalus (Boophilus) microplus* (S Genu) African swine fever virus maintenance and transmission dynamics in the sylvatic *Ornithodoros* vector (CI Boshoff)
10.00-10.30	Tea									
10.30-12.00	Plenary session: Day-one Skills for Veterinarians									
	Product showcase Bayer Animal Health (20 min) – Dr Clint Austin									
12.00-13.00	Lunch									

♦ These talks are earmarked for veterinarians that qualified in the last 5 years

Time	Small Animal Medicine	Critical Care	Small Animal Surgery	Production Animals	Wellness & Practice Management	Animal Welfare	Behaviour	Nurses	SASVEPM	Vet Techs/Technicians/SAALAS
Venue	**Auditorium**	**Cape Town International**	**Macro Polo**	**Grand Central**	**Wonderboom**	**Virginia**	**Waterkloof**	**Barcelona**	**King Shaka**	**Charles de Gaulle**
13:00-13:40	Euthyroid sick syndrome revisited (Johan Schoeman)	Toxicities: treatment and principles (Vanessa McClure)	Approach to front limb lameness ♦ (Greg Irvine Smith)	Brucella abortus – a frustrating herd disease (Ken Pettey)	Practice Management 101: Personnel ♦ (Henry Annandale)	Performing Animals Protection Amendment Act (Deryn Petty)	Canine compulsive disorders (Frederique Hurly)	Bite wounds wrapping it up! Bandages, dressings & drains (Sr Tamarin Fisher)	An evaluation of syndromic data from rural poultry farmers as a viable disease reporting tool using eastern Zambia as a model (Chrisborn Mubamba) A Livestock Field Census carried out in Gauteng province – Lessons learnt, Gauteng, South Africa, 2016 (Peter Geertsma)	African swine fever outbreak in South Africa, 2016 (R Malesa) The distribution of African Horse Sickness vectors in the protection and surveillance zones of the Western Cape Province, South Africa (K Labuschagne)
13:45-14:30	Feline hyperthyroidism (Joanne McLean)	Critical Care nutrition (Martin de Scally)	Approach to hind limb lameness (Fanie Naude)	Brucellosis knowledge, attitudes and practices of cattle keepers in a rural community in the Eastern Cape (Alicia Cloete)	Conflict management as encountered by veterinarians – focus on dealing with eg. difficult clients, basic Practice management and ethical considerations ♦ (Hermann Liebenberg)	Brachycephalics – An animal welfare disaster! (Dale Neves)	Canine noise phobia (Frederique Hurly)	Identifying the gold mine in your practice	Investigation of seasonal prevalence of low pathogenic avian influenza in a heterogenous wild waterfowl population in Pretoria (Thandeka Phiri Seasonal occurrence of Theileria parva infections and control practices amongst pastoralist communities in Monduli district, Northern Tanzania (Esther Gwae Kimaro)	Antimicrobial activity of silver, nano silver and antibiotics on selected mastitis causing organisms (Khanyi, M.S.F.) Presence and distribution of *Listeria monocytogenes* in South African meat and meat products (I Matle)
14:35:-15:15	Update on Addison's disease (Johan Schoeman)	Critical Care nutrition – continued (Martin de Scally)	Techniques for repair of cranial cruciate ligament disease (Greg Irvine-Smith)	Economic success farming with sheep and carnations (Ken Pettey)	Practice Management 101: Inventory ♦ (Henry Annandale)	The donkey skin trade (Morgane James)	Stress of the pet in the veterinary practice, how can we make the visit more pleasant for the pet ♦ (Frederique Hurly)	Happy snaps Radiology in young animals (Dr Nicky Cassel)	Zoonotic epidemiology of bovine brucellosis in Gauteng, South Africa, 2016 (Krpasha Govindasamy) Prevalence study of Brucella canis in parts of the Theewaterskloof and Overstrand municipalities of the Western Cape Province in South Africa (Werner Gouws)	Vaccination of on-farm cattle against heartwater using an attenuated tissue culture vaccine (R D Marumo) Three techniques confirming separate species status within the *Culicoides brucei* species group (K Labuschagne)
15:15-15.45	**Tea**									

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Time	Small Animal Medicine	Critical Care	Small Animal Surgery	Production Animals	Wellness & Practice Management	Animal Welfare	Behaviour	Nurses	SASVEPM	Vet Techs/Technicians/SAALAS
Venue	Auditorium	Cape Town International	Macro Polo	Grand Central	Wonderboom	Virginia	Waterkloof	Barcelona	King Shaka	Charles de Gaulle
15.45-16.25	Management of canine Cushing's disease (Varaidzo Mukorera)	Veterinary nosocomial infections (Neil Forbes)	Surgical options for repair of hip dysplasia (Fanie Naude)	Differential expression of tick resistance related genes following artificial infestation with R. microplus and R. decoloratus ticks (Chris Marufu)	"Brainspotting" for veterinarians – A new and revolutionary Neurophysiological evidence based psychotherapeutic breakthrough in dealing with stress, burnout, trauma and various other emotional challenges♦ (Hermann Liebenberg)	Monitoring physiological indicators of stress during transrectal palpation in mares used for teaching (Elize van Vollenhoven)	-	Feeding for recovery & long term health (Dr Guy Fyvie) 	Spatial planning, implementation, monitoring and evaluation of dog rabies vaccination campaigns in the Bushbuckridge Municipality, Mpumalanga Province, South Africa (Bjorn Reininghaus) Overview of the perceived risk of transboundary pig diseases in South Africa (Japtha Mokoele)	Diagnostic testing at PVVD, ARC-OVI (Matthee, O) Evaluation of African horse sickness cases to *Culicoides* numbers and climatic variables (K Labuschagne)
16:30-17:10	Diabetic ketoacidosis and hyperglycemic hyperosmolar syndrome (Amie Koenig)	Rational antibiotic use: should you really be using that? (Wilco Botha)	Patella luxation as a sign of an angular limb deformity (Ross Elliott)	Comparison of immunological responses in ancient and modern tick-host interactions (Chris Marufu)	Medici – The future of the doctor-patient relationship (Chris Ellis and Steve van der Watt)	-	-	Heading home, the best way to recovery (Dr Vanessa McClure) 	Seroprevalence and risk factors for Rift Valley fever in domestic ruminants in the Free state and Northern Cape, 2015-2016 (Yusuf Ngoshe) Projected numbers of historical human Rift Valley Fever and Crimean-Congo Heamorrhagic fever cases in South Africa (Veerle Msimang)	-
	Happy Hour									
17:30	**SAVA AGM – Venue: Cape Town International**									

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Wednesday 26 July 2017

Time	Critical Care	Small Animal Surgery	Small Animal Medicine	Wellness & Practice Management	Animal Ethics	Wildlife	Exotics	Nurses	SASVEPM	Vet Techs/ Technicians/ SAALAS
Venue	**Auditorium**	**Marco Polo**	**Cape Town International**	**Wonderboom**	**Grand Central**	**Lanseria**	**Waterkloof**	**Barcelona**	**King Shaka**	**Charles de Gaulle**
8.30-9.10	Recognizing shock, categories, treatment (Amie Koenig)	Surgical management of TL intervertebral disc disease (Sara Boyd)	Oncology cases of dogs and cats treated with electron radiation therapy (Georgina Crewe)	"Sleep Deprivation" and the debilitating impact on optimal performance among veterinarians – A practical guide to understanding sleep architecture, sleep disorders and remedial interventions♦ (Hermann Liebenberg)	Animal sentience, animal ethics and the veterinary profession (John Austin)	Wildlife antelope digestion – basic and practical review (Andri Garrett)	-	Perils for puppies & kittens – talking toxicities (Dr Wilco Botha) MSD Animal Health The Science of Healthier Animals®	The interpretation of laboratory diagnostic test results for disease diagnosis (Joule Kangumba)	**Workshop to be announced**
9.15-10.00	Heatstroke (Amie Koenig)	Surgical management of lumbar disc disease (Ross Elliott)	Understanding the basics of cancer and the various methods of treatments (Georgina Crewe)	Practice Management 101: Finances ♦ (Henry Annandale)	Why the veterinary profession cannot ignore the rights of non-human animals (Michele Pickover)	Treating rhino trauma, orphans and translocation (Peter Rogers)	-	VNASA AGM (Sr Retha Pansegrouw)	Detection and distribution of bovine trypanosomiasis in Malawi (Elizabeth Chimera) Molecular epidemiology of bovine trypanosomiasis amongst pastoralist cattle: a case of Monduli District, Northern Tanzania (Esther Gwae Kimaro)	**Workshop to be announced**
10.00-10.30	**Tea**									
10.30-12.00	**Opening/Plenary session: Antimicrobials – Dr Niel Homer-Forbes**									
	Product show Health and Hygiene (20 min) – Dr Niel Homer-Forbes									

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12.00-13.00	Lunch								
Time	**Critical Care**	**Small Animal Surgery**	**Wellness & Practice Management**	**Animal Ethics**	**Wildlife**	**Exotics**	**Nurses**	**SASVEPM**	**Vet Techs/ Technicians/ SAALAS**
Venue	**Auditorium**	**Marco Polo**	**Wonderboom**	**Grand Central**	**Lanseria**	**Waterkloof**	**Barcelona**	**King Shaka**	**Charles de Gaulle**
13:00-13:40	Sepsis (Amie Koenig)	Surgical treatment of cervical disc disease ventral slot vs distraction stabilisation (Sara Boyd)	Conflict management as encountered by veterinarians – focus on dealing with eg. difficult clients, basic Practice management and ethical considerations♦ (Hermann Liebenberg)	Happiness – The basis of animal welfare assessment for the veterinarian (Quixi Sonntag)	Kruger National Park anti-rhino poaching K9 units (Johan de Beer)	-	Fluid therapy "the good, the bad & the ugly" (Dr Lynette Bester)	The coordinate confusion (Hannes Pienaar) Prevalence and risk factors for antimicrobial resistant Staphylococcus aureus isolates from poultry meat products in South Africa, 2015-2016 (Vashnee Govender)	**Workshop to be announced**
Time	**Critical Care**	**Physiotherapy**	**Wellness & Practice Management**	**Animal Ethics**	**Wildlife**	**Exotics**	**Nurses**	**SASVEPM**	**Vet Techs/ Technicians/ SAALAS**
Venue	**Auditorium**	**Marco Polo**	**Wonderboom**	**Grand Central**	**Lanseria**	**Waterkloof**	**Barcelona**	**King Shaka**	**Charles de Gaulle**
13:45-14:30	Recognizing Respiratory Patterns in the Dyspnoeic Patient (Amie Koenig)	What is it all about and how can it contribute (Marinette Teeling)	Practice Management 101: Personnel ♦ (Henry Annandale)	Better science from fewer animals. Where science and animal welfare meet (Bert Mohr)	Overview of diseases in African rhinos (Michele Miller)	Avian emergencies, triage and stabilization (Dorianne Elliott)	"Back to school arrhythmic" Drug & infusion calculations (Dr Lynette Bester)	Prevalence of Salmonella spp. In slaughter cattle, the abattoir environment and meat sold at retail outlets in Gauteng (Ayanda Manqele) Antimicrobial resistance profiles of Listeria monocytogenes isolates from raw meat, processed meat products and ready to eat meat products in South Africa (Itumeleng Matle)	**Workshop to be announced**
14:35:-15:15	Monitoring ICU patients (Kenneth Joubert)	Review of the science behind the modalities used (Marinette Teeling)	"Brainspotting" for veterinarians – A new and revolutionary Neuro-physiological evidence based psychotherapeutic breakthrough in dealing with stress, burnout, trauma and various other emotional challenges♦ (Hermann Liebenberg)	Tail docking in dogs: 10 years on (Quixi Sonntag)	Cheetah veterinary management and lessons learnt (Peter Rogers)	Recent advances In avian orthopaedic surgery (Neil Forbes)	Why is paediatric care in icu important? (Dr Vanessa Mcclure) 	Frequency of occurrence and antimicrobial resistance patterns of Escherichia coli O157 and non-O157 E. coli isolated from meat and meat products in South Africa (Keneiloe Malokotsa) Mycobacterium bovis infection in cattle at the wildlife/livestock interface in northern KwaZulu-Natal province, South Africa (Petronillah Sichewo)	**Workshop to be announced**

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15:15-15.45	Tea								
Time	**Dermatology (Virbac)**	**Physiotherapy**	**Wellness & Practice Management**	**Animal Ethics**	**Wildlife**	**Exotics**	**Nurses**	**SASVEPM**	**Vet Techs/ Technicians/ SAALAS**
Venue	**Auditorium**	**Marco Polo**	**Wonderboom**	**Grand Central**	**Lanseria**	**Waterkloof**	**Barcelona**	**King Shaka**	**Charles de Gaulle**
15.45-16.25	Diagnosis and management of canine atopy (Andrew Leisewitz)	The surgical patient (Marinette Teeling)	Practice Management 101: Inventory ♦ (Henry Annandale)	-	Common and emerging infectious diseases of farmed sable antelope (Jacques O'Dell)	Approach to the anorexic rabbit (Dorianne Elliott)	Opening a can of worms – the 3 terrors (Dr Mats Abatzidis) MSD Animal Health The Science of Healthier Animals®	**SASVEPM AGm**	**VET TECHS AGM**
16:30-17:10	Overview of cutaneous auto-immunities (Andrew Leisewitz)	The neurological patient (Marinette Teeling)	Medici – The future of the doctor-patient relationship (Chris Ellis and Steve van der Watt)	-	First aid and immobilization drug exposure (Jacques O'Dell)	Basic reptile husbandry and medicine (Dorianne Elliott)	Lifetime nutrition – start at the very beginning (Dr Guy Fyvie) Hills	**SASVEPM AGm**	**VET TECHS AGM**
Time	**Dermatology (Virbac)**	**Physiotherapy**	**Wellness & Practice Management**	**Animal Ethics**	**Wildlife**	**Exotics**	**Nurses**	**SASVEPM**	**Vet Techs/ Technicians/ SAALAS**
Venue	**Auditorium**	**Marco Polo**	**Wonderboom**	**Grand Central**	**Lanseria**	**Waterkloof**	**Barcelona**	**King Shaka**	**Charles de Gaulle**
17:15 -17:55	Diagnosis and treatment of Otitis externa (Heidi Schroeder)	The ICU Patient (Marinette Teeling)	-	-	What future for African vultures? (Neil Forbes Triage and What first aid for injured free-living African vultures? (Neil Forbes)	How not to kill your exotic patients (Dorianne Elliott)	-	-	-
19:30	**Gala Dinner**								

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Thursday 27 July 2017

Time	Small Animal Medicine	Small Animal Surgery	Production Animals	Equine	Wellness & Practice Management	Nurses	SASVEPM
Venue	**Auditorium**	**Marco Polo**	**Grand Central**	**Lanseria**	**Virginia**	**Barcelona**	**King Shaka**
8.30-9.10	Pancreatitis: demystifying diagnosis and treatment (Zandri Whitehead)	Dealing with the obstructed ureter (Ross Elliott)	Uses and abuses of antibiotics and antibiotic resistance (Adél de Haast)	Practical application of special radiographic views in equine practice (Nicolene Hoepner)	Practice Management 101: Finances ♦ (Henry Annandale)	It wasn't me! Accepting accountability (Des Rees) 	Collection and packaging of diagnostic samples (Didi Janse van Rensburg & Laura Lopez)
9.15-10.00	My patient is anaemic and it's not *Babesia*...now what? (Anri Celliers)	Artificial Urethral Sphincter placement for urinary incontinence (Ross Elliott)	Methicillin-resistant Staphylococcus aureus in pig farming (Shani van Lochem)	Standard equine radiographic views, their worth & basic radiological anatomy (Nicolene Hoepner)	"Sleep Deprivation" and the debilitating impact on optimal performance among veterinarians – A practical guide to understanding sleep architecture, sleep disorders and remedial interventions ♦ (Hermann Liebenberg)	Breaking the cycle of burnout♦ (Des Rees) 	Zoonotic disease awareness of one health stakeholders, Gauteng, 2016 (Krpasha Govindasamy) Brucella melitensis – combatting an outbreak in the field (Bennie Grobler)
10.00-10.30	**Tea**						
10.30-12.00	**Plenary session: Remuneration for Vets**						
	Product show case Virbac Animal Health (20 min) – Sr Tracey Phillips						
12.00-13.00	**Lunch**						
Time	**Small Animal Medicine**	**Small Animal Surgery**	**Production Animals**	**Equine**	**Practice Management**	**Nurses**	**SASVEPM**
13:00-13:40	Approach to PuPd (Chad Berman)	Surgery of the hepatobillary system (Charlie Boucher)	Cryptosporidium: Update on diagnostics, treatment, and control in South Africa (Luke Arnot)	Current Deworming protocols for horses Dentistry for Dummies (Ingrid Cilliers)	Practice Management 101: Personnel ♦ (Henry Annandale)	**#superbugs-mustfall** Antimicrobial Resistance (Dr Neil Forbes) 	Comparison of individual and herd seroprevalence of bovine brucellosis in North West Province (Cheryl McCrindle) Risk factors for bovine brucellosis in KwaZulu-Natal (Thami Nogwebela)
13:45-14:30	Acute Phase Proteins in diagnosis with emphasis on CRP (Fred Reyers)	How to ensure the best possible outcome in intestinal surgery (Charlie Boucher)	Technologies in the farming industry and their potential to help veterinarians (Emiliano Raffrenato)	Current surgical techniques for removing fractured molars (Ingrid Cilliers)	Conflict management as encountered by veterinarians – focus on dealing with eg. difficult clients, basic Practice management and ethical considerations ♦ (Hermann Liebenberg)	Paediatrics vs geriatrics (Sr Tania Serfontein)	Serological analysis of Brucella serum and milk samples with in-house iELISA converted on luminex xmap technology (Maphuti Betty Ledwaba) A comprehensive next-generation sequencing strategy for whole genome analysis of SAT1 and SAT2 foot and mouth disease viruses (Danica van der Merwe)

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14:35:-15:15	Update on point of care tests for the diagnosis of CPV, CDV, FIV, and FeLV (Denis York)	Approach to the dyspnoeic brachycephalic patient (Sara Boyd)	The influence of dam nutrition on offspring's performance (Giulia Esposito)	Current therapies available for joints/ tendons (Ingrid Cilliers)	Practice Management 101: Inventory ♦ (Henry Annandale)	"Anaesthesia preparation" Minimising the side effects (Sr Tania Serfontein)	Isolation and whole genome analysis of a lytic bacteriophage infected Bacillus anthracis isolate from Pafuri, South Africa (Ayesha Hassim) Comparing immunogenicity of non-living anthrax vaccine candidates in combination with simultaneous antibiotic treatment in goats and using passive mouse protection model (Henriette van Heerden)
15:15-15.45	**Tea**						
Time	**Small Animal Medicine**	**Small Animal Surgery**	**Production Animals**	**Equine**	**Practice Management**	**Nurses**	**SASVEPM**
Venue	**Auditorium**	**Marco Polo**	**Grand Central**	**Lanseria**	**Virginia**	**Barcelona**	**King Shaka**
15.45-16.25	Outbreak of canine distemper in dogs owned by low-income families in Mozambique (Custodio Bila)	Technique for emergency tracheostomy (Charlie Boucher)	Heifer and calf nutrition: much more than growth (Emiliano Raffrenato)	Enhancing Wound Healing in Horse – Part 1 (Johan Marais)	"Brainspotting" for veterinarians – A new and revolutionary Neuro-physiological evidence based psychotherapeutic breakthrough in dealing with stress, burnout, trauma and various other emotional challenges ♦ (Hermann Liebenberg)	Creating fabulous fur kids, setting young animals up for success (Dr Quixi Sonntag) MSD Animal Health The Science of Healthier Animals®	Clinical expression of African horse sickness in South African horses (Megan Riddin) A field investigation of the African horse sickness outbreak in the controlled area of South Africa in 2016 (John Grewar)
16:30-17:10	Unique Compounded products applicable to small animal practice (Ockert Botha)	Closure of large skin wounds (Ross Elliott)	Review of the Downer cow syndrome management (Chris Marufu)	Enhancing Wound Healing in Horse – Part 2 (Johan Marais)	-	The ticking time bomb! Tools for time management ♦ (Des Rees) Hills	Seroprevalence and associated risk factors of West Nile virus in equine populations in South Africa (Rebecca Jeal) The design and field implementation of a digital identification system for horses (Melanie Scholtz)
17:15 -17:55	Veterinary Compounding demystified – Laws, Regulations, Rules and Quality (Estelle Botha & Jacques Lubbe)	-	-	TBA	**SASVEPM POSTER PRESENTATIONS:** • *Seroprevalence of leptospirosis from abattoirs slaughtered animals in Gauteng province, South Africa* (Banenat Bajehson Dogonyaro) • *Retrospective data analysis on Salmonella serotypes in animals and animal products in South Africa from 2007 to 2014* (Awoke Gelaw) • *Mycobacterium tuberculosis infection in cattle from the Eastern Cape Province of South Africa* (Tiny Hlokwe) • *Prevalence, serotypes and virulence characteristics of Shiga toxin-producing Escherichia coli (STEC) from cow-calf operations in the Gauteng and North West Provinces of South Africa* (Musafiri Karama) • *Sero-prevalence of brucellosis in slaughter animals in Gauteng province abattoirs, South Africa and assessment of risk factors posed to abattoir workers* (Francis Kolo) • *Prevalence and characterisation of Shiga toxin-producing Escherichia coli in beef carcasses and beef products in Gauteng province* (Libby Onyeka) • *Development of real time PCR assays to improve the accuracy of bovine and porcine cysticercosis diagnosis* (Ana Mbokeleng Tsotetsi-Khambule)			

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MY PATIENT IS ANAEMIC AND IT'S NOT BABESIA…NOW WHAT?

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ABSTRACT

Anaemia is a decrease in the erythrocyte mass in the body and one of the most commonly identified haematological abnormalities seen in veterinary practice. It is not a primary diagnosis and the underlying cause should be investigated in most cases. Confirmation of anaemia is an essential first step in the diagnostic process. The decision whether to continue with an investigation into the cause of anaemia, is determined by the severity of the anaemia and clinical presentation of the patient. Thereafter a systematic approach, starting with classifying the anaemia as regenerative or non-regenerative, can be followed to identify the causal pathomechanism. Each category has a specific list of differentials as well as diagnostic tests to perform, which can be used to narrow down the search for the underlying cause for anaemia.

INTRODUCTION

Anaemia is a decrease in the erythrocyte mass in the body. It is reflected as a haemoglobin concentration, red blood cell (RBC) count and haematocrit (Ht) or packed cell volume (PCV) below the reference interval set to include 95% of the population[3]. The search for the underlying cause of anaemia can be daunting due to the numerous potential differentials. The causal pathomechanism, with the appropriate differentials and diagnostic tests to perform, can be uncovered with an organized approach. This will aid in the correct management of these often, frustrating cases.

CONFIRMATION OF ANAEMIA

Not all patients with pale mucous membranes are anaemic. Pallor can be caused by any condition that causes hypoperfusion such as: hypovolaemia, shock, dehydration, pulmonary thromboembolism, pulmonary hypertension and cardiac tamponade[3]. The different reasons for pallor can be distinguished during the hands-on physical examination of the pulse quality and capillary refill time (CRT)[3]. Even though a CRT in patients with pale mucous membranes can be difficult to determine, it is important to note whether the mucous membrane colour is 'white on yellow' or 'white on pink'. To confirm anaemia a PCV or Ht is ultimately required.

IS A WORK-UP REALLY NEEDED?

After establishing anaemia, the next decision is whether this case requires a complete work-up or if a wait-and-see approach can be adopted. This will depend on the severity of anaemia and whether the patient's vital parameters are stable. For a stable patient with a Ht > 30%, a recheck in 3 – 7 days are in order. If by that time, the patient decompensates or the Ht remains the same or lower, a complete work-up is warranted[3].

It is also necessary to take the patients' signalment and reproductive status into account when diagnosing anaemia[3]. Puppies, pregnant bitches and patients that are sedated, under general anaesthesia or overhydrated will have a mildly decreased haematocrit[5]. Certain breeds, like sighthounds, normally have a Ht > 50%[5]. In these breeds a Ht less than that, even if it's still within the reference interval, can be classified as anaemic.

Specific questions to ask the owner to aid in finding the cause of anaemia should concentrate on recent medication, parasite control, recent vaccinations, travel history, blood loss noted (epistaxis, haematemesis, haemoptysis, bleeding wounds), toxin ingestion (onions, garlic, acetaminophen, zinc), bee stings and snakebites.

It is important to remember that anaemia is not a primary diagnosis and an underlying cause needs to be determined.

CLASSIFICATION OF ANAEMIA

Anaemia can be classified according to the pathophysiological mechanism (RBC loss, RBC destruction or decreased RBC production), bone marrow response (regenerative or non-regenerative), RBC indices (mean corpuscular volume (MCV) and mean corpuscular haemoglobin concentration (MCHC)) and severity together with duration (acute or chronic; mild, moderate or severe). All 4 categories are used in the description of anaemia[5]. Determination whether a regenerative or non-regenerative anaemia is present, is a key first step in the diagnosis. Regenerative anaemias are only caused by haemorrhagic or haemolytic conditions and non-regenerative anaemias mainly involve ineffective erythropoiesis by the bone marrow[4]. Specific differentials and diagnostic tests exist for both main categories.

With haemorrhagic anaemia, blood loss can either be internal as seen with haemoperitoneum and haemothorax, or external via the gastro-intestinal tract, pulmonary system, genitourinary system and skin. Possible differentials include: trauma, rupture of neoplasic masses, coagulopathy (rodenticide toxicity, thrombocytopenia or thrombocytopathia) and severe tick, flea or worm burdens[3; 4]. Chronic blood loss anaemia can become non-regenerative due to iron deficiency, but the RBC indices will most often differ from other non-regenerative causes.

Haemolytic anaemia can be distinguished into intravascular haemolysis (rupture of RBC in circulation) or extravascular haemolysis (phagocytosis of RBC by macrophages and removal by the liver, spleen and lymph nodes). These conditions can occur separately or in combination. Signs of haemolysis are: icterus, yellow discoloured faeces and red discoloured urine with haemoglobinuria and haemoglobinaemia seen mainly with intravascular haemolysis[5]. Acquired non-immune mediated conditions, haemophagocytic syndromes and immune mediated conditions can all cause haemolytic anaemia. Differentials for acquired non-immune mediated conditions include: toxins (bee stings, snake envenomation, onions, garlic, acetaminophen, zinc, vitamin K and methylene blue), infectious (*Babesia*), hereditary (erythrocytes enzyme deficiencies such as: pyruvate kinase deficiency and phosphofructokinase deficiency, increased osmotic fragility) and severe hypophosphataemia. RBC fragmentation syndromes are caused by conditions that alter the blood flow and movement of RBC through narrowed blood vessels (disseminated intravascular coagulation (DIC), vasculitis, haemangiosarcoma, cardiac and liver diseases, splenic torsion and haemolytic uraemic syndrome)[4].

Differentials for immune mediated haemolysis are: idiopathic immune mediated haemolytic anaemia (IMHA) or secondary IMHA caused by infections, toxins, medication or neoplastic conditions.

As a general guideline, Ht and total serum protein (TSP) will be decreased with haemorrhagic anaemia, while with haemolytic anaemia, Ht will be decreased while TSP will be normal or increased[4]. Characteristic changes seen on the blood smear and on the RBC morphology in cases with haemolysis are: spherocytes, acanthocytes, eccentrocytes, Heinz bodies, keratocytes, schistocytes, siderocytes and agglutination[3].

Non-regenerative anaemia is appreciated in more chronic conditions seeing that the average lifespan of an erythrocyte is about 100 days in the dog. It can be suspected when the reticulocyte count is still < 60 000 cells/µL after an adequate time of 3 -7 days has been allowed for regeneration to take place after a hypoxic event[3]. This time frame should also be kept in mind with peracute bleeding, which will initially appear non-regenerative. Ineffective erythropoiesis is either due to inadequate stimulation of the bone marrow by erythropoietin, the bone marrow being unable to respond to the erythropoietin stimulus or a maturation defect of the RBC precursor cells[1].

Causes of non-regenerative anaemia can broadly be classified into extramedullary and medullary categories. Differentials for extramedullary disease include: anaemia of inflammatory disease (AID), renal disease, hypothyroidism, hypoadrenocorticism and neoplasia. AID is a commonly encountered cause of non-regenerative anaemia due to increased cytokine production, abnormal iron regulation and the increased production of hepcidin during times of inflammation[2], and usually characterized by a mild normocytic, normochromic anaemia. Chronic kidney disease causes a decreased production

of erythropoietin that together with uraemic toxins, lead to non-regenerative anaemia. Thyroid hormones and cortisol stimulate erythropoiesis[3; 4].

Medullary disease can be caused by increased intramedullary cell death, abnormal cell production with intramedullary cell death, damage or destruction of the RBC precursors, decreased Hb production, decreased or abnormal DNA production and infiltrative neoplasia[3].

Causes of intramedullary cell death include: non-regenerative IMHA (diagnosis by exclusion), hormones (oestrogen), toxins, histiocytic disorders, infections (*Ehrlichia*) and drugs. The most common drugs involved are: chemotherapy agents, acetaminophen, trimethoprim sulfadiazine, phenobarbital, azathioprine, cephalosporins, carprofen, metronidazole, albendazole, fenbendazole and recombinant human erythropoietin. Myelodysplastic syndromes are implicated in abnormal cell production with increased cell death[1; 4].

Differentials for damage or destruction of RBC precursors are: pure red cell aplasia (PRCA), Parvo virus, *Ehrlichia*, drugs and toxins[3]. With PRCA a normocytic, normochromic anaemia with erythroid hypoplasia/aplasia is seen. The leukocytes and thrombocytes are usually still within normal limits.

Decreased Hb production due to iron deficiency is often caused by chronic blood loss, but portosystemic shunting and inflammatory disease can also lead to hypoferremia[2]. RBC indices often only become microcytic and hypochromic later in the disease process, because of the relatively long lifespan of RBC in circulation[7]. Iron deficiency anaemia is a well-recognized cause of anaemia in humans, especially amongst the elderly population, due to iron-deficient diets and gastrointestinal blood loss[2]. Similar discoveries have recently been made in studies on healthy geriatric canine populations[6]. Obesity in humans may also be linked to iron deficiency due to the presence of chronic subclinical inflammation[2]. Humans with *Helicobacter pylori* infections are prone to iron deficiency due to decreased absorption of iron, decreased bioavailability of vitamin C and the formation of micro ulcerations[2]. The same might hold true for veterinary patients.

Vitamin B12 deficiency impairs DNA production in the erythroblasts by preventing purine and thymidylate synthesis. Deficiencies may be seen with hereditary selective cobalamin malabsorption and other malabsorption syndromes[3].

Acute and chronic leukaemia, lymphoma, multiple myeloma and malignant histiocytosis are the most common infiltrative bone marrow neoplasia's encountered[1].

Aplastic anaemia effect 2 – 3 cells lines causing a bi- or pancytopenia, with 95% of the bone marrow being replaced by adipose tissue. Myelonecrosis can involve focal or multifocal areas of coagulative necrosis and can be caused by sepsis, lymphoma, IMHA, systemic lupus erythematosus (SLE) and drugs. Eventually myelonecrosis will lead to myelofibrosis due to replacement of bone marrow with fibroblasts and collagen[1].

Purulent or granulomatous inflammation in the bone marrow can result in bone pain and lameness with non-regenerative IMHA and sepsis. It is usually associated with a left shift neutrophilia with toxic changes[1].

DIAGNOSTIC TESTS

A minimum database including a history, physical examination, blood smear, urinalysis and faecal examination should be done when anaemia is confirmed. If an obvious reason for the anaemia does not present itself at this stage, a complete blood count with a reticulocyte count and a biochemistry panel is required. A reticulocyte count more than 60 000 cells/µL is required to confirm regeneration[3].

If a regenerative anaemia is diagnosed, an extended data base to include parasite testing for *Ehrlichia*, thoracic and abdominal radiographs, abdominal ultrasound, in-saline agglutination (ISA) and Coombs to test for IMHA and anti-nuclear antibody (ANA) testing for Systemic Lupus Erythematosus (SLE), can be performed to further determine the underlying cause.

With non-regenerative anaemia, expanding of the data base might be needed to also include an ACTH stimulation test, thyroid hormone levels combined with TSH, vitamin B12, folate and copper

levels, an iron panel and lastly bone marrow cytology and core biopsy with histopathology. Bone marrow biopsies can lead to a clinical diagnosis, in which case treatment can be instituted without further testing, or a pathological diagnosis, which will necessitate further testing before appropriate therapy can be started.

CONCLUSION

The underlying cause of anaemia is often multifactorial which can complicate the diagnostic process to reach a final diagnosis. Once anaemia has been confirmed, a methodical approach can be followed to identify the underlying pathomechanism involved, perform the appropriate diagnostic tests and to ultimately reach a diagnosis to be able to institute corrective measures and treatment.

Table 1. Summary of the main causes of anaemia

Regenerative anaemia			Non-regenerative anaemia
Haemorrhagic	Haemolytic		Defective erythropoeisis
Trauma	Non-immune mediated	Immune mediated	Anaemia of inflammatory disease
Rupture of neoplastic masses	Onions	Idiopathic IMHA	Iron deficiency
Coagulopathy	Garlic	Secondary IMHA	Renal disease
Severe parasite burdens	Zinc		Hepatic disease
GIT ulcerations	Acetaminophen		*Ehrlichia*
Wounds	Bee stings		Drugs
Genitourinary	Snake envenomation		Estrogen
Respiratory	Vitamin K		Non-regenerative IMHA
Surgery	Methylene blue		PRCA
Rupture of non-splenic neoplasms	*Babesia*		Aplastic anaemia
	Erythrocyte enzyme deficiencies		Infiltrative neoplasia
	Erythrocyte fragmentation syndromes		Parvo virus
	Severe hypophosphataemia		Cobalamin deficiency
	Heatstroke		Endocrine disorders
			Radiation

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APPROACH TO POLYURIA AND POLYDIPSIA

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ABSTRACT

Polyuria (PU) and polydipsia (PD) are common presenting complaints in small animal practice. It is something which is not worked up methodically and often it is thought that Diabetes mellitus (DM) is the underlying problem, however there are numerous different differentials. The cause of PU/PD can usually be determined from the first examination. This includes an adequate history, physical examination as well as appropriate routine laboratory tests. An understanding of the physiology of water metabolism is essential to understand why the animal has PU/PD. This together with a systematic approach to cases will allow the clinician to determine the cause of the problem while managing patients appropriately.

PHYSIOLOGY OF WATER METABOLISM

Water consumption and urine production are controlled by interactions between plasma osmolality, fluid volume in the vascular compartment, the thirst centre, the kidneys, the pituitary gland and the hypothalamus. Imbalance or dysfunction in any of these can result in abnormalities of water metabolism. This results in PU and usually a compensatory PD. The exception is a psychogenic PD with a compensatory PU to excrete the excess water. Water balance is controlled by complex a system that involves regulation of water intake via thirst mechanisms and control of water loss via the kidneys, influenced by the action of antidiuretic hormone (ADH) and water lost through the respiratory and gastrointestinal tracts.

ADH is synthesized in the hypothalamus and secreted by the posterior pituitary gland. Both increasing plasma osmolality and hypovolaemia are stimuli for the hormones release. ADH promotes water conservation and increases plasma volume. Central diabetes insipidus (CDI) occurs if the body cannot produce ADH and nephrogenic diabetes insipidus (NDI) occurs if the kidneys can no longer respond to ADH. The cause of CDI is usually idiopathic but can result from trauma, neoplasia or inflammatory disease. Primary NDI is very rare but secondary NDI is extremely common. Solute diuresis results in an increased volume of fluid reaching the distal tubules. Low plasma concentrations of sodium, chloride and urea reduces the ability to maintain interstitial osmolality.

HISTORY

A detailed history is an important first step in the diagnostic work up. A good history can allow differentiation between urinary incontinence, nocturia and pollakuria. The signalment, clinical signs and physical examination can aid in differentiation. For example urinary incontinence is expected in middle aged spayed bitches who passively leak urine when lying down or sleeping whereas nocturia is seen more commonly in older dogs due to senile related changes. Further history should involve enquiring about the patients appetite, on any medication, is the animal spayed as well as general questions regarding the overall health of the patient. Important questions to ask include the severity of the PU/PD. In CDI the PU/PD is usually severe in comparison to other polyuric disorders; the PU/PD is usually moderate. Furthermore the onset of PU/PD is usually very sudden in CDI but can also be seen in iatrogenic Cushing's. Furthermore asking questions about recent changes in the house environment, lack of exercise or discipline may increase suspicion of psychogenic polydipsia (PP).

CLINICAL EXAMINATION

A thorough physical examination is essential to any animal with PU/PD. Palpating the thyroid area, peripheral lymph nodes, mammary glands and anal sacs for any evidence of enlargement which will increase suspicion for malignancy as a possible cause. An enlarged abdomen and hepatomegaly may give indication for Cushing's disease. The clinical signs are also essential as a bright and alert dog is less likely to be suffering with pyometra or Addison's then an animal who is systemically

unwell. A proper fundus examination for cataracts and papilledema. Dermatological changes like symmetrical alopecia, comedones and skin thinning may suggest hyperadrenocorticism. Palpation of the kidneys may also increase suspicion of certain diseases like chronic kidneys disease (CKD). Ascertaining if the patient is losing weight may indicate DM or even malignancy.

DIAGNOSTIC APPROACH

Once confirming from the history (as well as from the urine) that the animal is PU/PD, the most important diagnostic step is to determine the urine specific gravity (SG). It is essential to interpret the urine SG together with the hydration status of the patient as the urine SG of healthy animals can vary depending on physiological circumstances. An animal that is dehydrated or hypovolaemic, the appropriate response would be to produce concentrated urine with a SG of 1.030 in dogs and 1.035 in cats. In comparison to an azotaemic animal with a urine SG of less than 1.030 indicates that the patient has some impaired urine concentrating abilities. Concentrated urine (SG>1.030) without glucose may indicate a lower urinary tract infection, bladder calculi, bladder mass, behavioural, neurogenic or anatomical abnormalities. Concentrated urine with glucose can often indicate diabetes mellitus (DM). In cats stress may result in glucosuria and thus a home sample and fructosamine may be needed. Cushing's disease can result in glucosuria and hyperglycaemia in both dogs and cats. Furthermore Acromegaly will lead to DM in most cats. In the rare situation where the blood glucose is normal a congenital tubular defect should be suspected. Hyposthenuric urine (SG< 1.008) is suspicious of CDI, NDI and PP, however it also indicates the renal tubules have retained the ability to dilute the glomerular filtrate and are thus functioning. Isosthenuria (SG 1.008-1.012) implies a urine SG that is the same as serum or plasma. Isosthenuria can be seen in many PU conditions but a persistent isosthenuria is often consistent with chronic kidney disease. A urine SG of 1.020 can be seen in many conditions including pyometra, hypercalcaemia, hepatic disease, hyper/hypoadrenocorticism, pylonephritis, hypokalaemia, hyperthyroidism, iatrogenic and post obstructive diuresis. From here appropriate tests which can include a full blood count, biochemical profile, ACTH stimulation test, abdominal radiographs as well as abdominal ultrasound may be needed to rule in or out many of the above conditions.

When all of the above conditions are ruled out the following 3 conditions to consider are CDI, NDI and PP. At this point a modified water deprivation test can be performed. It is a time consuming and expensive test that does pose a risk to the patient. The purpose of this test is to see if the patient can produce ADH and if the kidneys can respond to it. This test should always be done on an inpatient basis and is contraindicated in animals with CKD and close monitoring of hydration status and body weight is important. If the patient can concentrate urine then PP is suspected. If however the urine is still not concentrated ADH can be administered. Furthermore if the urine concentrates then CDI is suspected but if it doesn't the patient most likely has NDI. One can also measure the serum osmolality (normal around 300mosm/l). Dogs with CDI tend to have high serum osmolality since the primary stimulus for PD is hypovolaemia and increased plasma osmolality due to losses of free water. Conversely dogs with PP tend to have normal or low serum osmolality since excessive water intake results in hypervolaemia and reduced plasma osmolality. A trial therapy with synthetic ADH is also a safe way of making a diagnosis where CDI should respond. If PP is suspected a behaviourialist should be consulted but if CDI is high up on the differential list then further imaging studies like a MRI should be considered.

Thus PU/PD can be divided into a few categories which include primary polydipsia, primary polyuria which involves osmotic diuresis, CDI, primary NDI and secondary NDI. There are numerous different differentials for each of these.

- Primary PD: behavioural, fever, encephalopathy, pain and a neurological disorder.
- Osmotic diuresis: DM, Fanconi's syndrome, post-obstructive diuresis and primary renal glucosuria.
- CDI: Congenital, neoplastic, trauma and a renal insensitivity to ADH.
- Primary NDI
- Secondary NDI: Chronic renal failure, renal medullary washout, pyelonephritis, pyometra, liver disease, hyper/hypoadrenocorticism, hypercalcaemia, hypokalaemia, hyoerviscosity, high salt diet as well as certain drugs.

CONCLUSION

y following a logical and systemic approach to PU/PD patients the underlying cause should be identified. The history and physical examination is critical to any diagnostic work up. This will aid in ruling in/out many differentials. Thereafter a urine analysis should be performed and appropriate tests should be chosen based on what would aid in making the diagnosis, for example using an ACTH stimulation test when Cushing's disease is suspected and not as a screening test. Most causes of PU/PD should be determined by following this methodical approach. When the diagnosis is still eluding the clinician a modified water deprivation test can then be performed in order to differentiate between CDI, PP and NDI. CDI is uncommon and PP and NDI are both extremely rare.

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UNDERSTANDING THE BASICS OF CANCER AND THE VARIOUS METHODS OF TREATMENTS

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A brief incomplete introduction (revision) to the basics of cell anatomy, mitosis, types of tissues and their normal mitotic rates. DNA, genes, the tumour suppressor gene (TSG) controlling the rate of mitosis, damage to tumour suppressor gene resulting in cancer (uncontrolled growth of damaged cells). The life cycle of the normal cell and apoptosis (cell suicide).The life cycle of the cancer cell and avoidance of apoptosis. The basics of surgery, chemo and radiation therapy the advantages and disadvantages of each and why they are used in combination often very successfully. Finally a brief discussion on metronomic therapy and palliative treatment.

CRP AS A TEST FOR ASSESSING AND MONITORING SYSTEMIC INFLAMMATION

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ABSTRACT

Inflammation caused by a number of disparate mechanisms, cause the release of Interleukins by leukocytes and some of these (such as IL-6) promote the release of Acute-phase proteins by the liver. One of these is C-reactive protein (CRP). The use of CRP in the detection and monitoring of inflammatory disease, in human medicine has been well established for over 30 years and many specific applications have been published.
For many years validate methods were not available for the assay of CRP in veterinary medicine (such as for dogs) and in-house developed methods were used in the earlier publications on CRP in dogs. More recently, some methods designed for use in humans were validated for canine serum CRP and very recently, a number of immunologically based methods, specific for canine serum CRP were developed. This has led to the publication of numerous articles demonstrating its clinical usefulness in certain settings. In order to derive maximum utility of the now widely available test, the author has suggested four clinical scenarios to be used as templates to guide the clinician in using this test.

- Scenario 1: Vague clinical presentation (such as anorexia) where the test is used to determine if significant inflammatory pathology is present.
- Scenario 2: Differentiating between inflammatory and non-inflammatory causes of clinically evident abnormal signs (such as differentiating inflammatory from non-inflammatory causes of lameness)
- Scenario 3: In the presence of diagnostically established inflammatory disease (such as burns), determining the degree of inflammation and setting a prognosis.
- Scenario 4: In the presence of diagnostically established inflammatory disease (such as pneumonia), determining the response to treatment.

INTRODUCTION

Inflammation can be produced by a number of tissue insults, including trauma, heat, infections, immune reactions and various types of neoplasia. During inflammation, T-lymphocytes and monocytes/macrophages (principally) produce a number of “communication molecules” called Interleukins and, in particular, Interleukin-6 (IL-6). In turn, IL-6 induces the production of a range of compounds by the hepatocytes, which are called “Acute Phase Reactants”, most of which are proteins and hence the name “Acute Phase Proteins” (APP) is generally used. Different APPs respond to differing degrees (in terms of their blood serum concentration) in different species. For instance, in humans and dogs C-reactive protein (CRP) has a profound response (up to a 1000-fold increase over resting levels), whereas in horses and cats it does not. In those species Serum Amyloid A (SAA) and Alpha-1 Acid Glycoprotein (AGP), respectively show a greater increase. APPs are produced as part of the innate immune response and play different roles in this response. CRP, for instance, *inter-alia* promotes the binding of Complement to bacteria, enhancing phagocytosis, induces the production of other cytokines, and inhibits neutrophil chemotaxis.

CRP tends to rise rapidly in serum after the onset of inflammation and can peak in 24-48 hours. It has a relatively short half life and therefore, when the inflammation abates (or is successfully treated) CRP levels decline rapidly.

USE OF SERUM CRP IN HUMAN MEDICINE

A test for CRP has been available in human medicine for well over 30 years and it has been shown to be a useful aid in the diagnosis of various forms of inflammatory joint disease, rheumatic fever, pancreatitis, sepsis, pneumonia, appendicitis, bacterial endocarditis, post-operative infection, Crohn’s disease, cystitis, differentiating between inflammatory and non-inflammatory thyrotoxicosis, post-operative monitoring of metritis, risk of death in chronic dialysis patients, identifying neonatal infections, urinary

been used to assess response to treatment in a number of conditions and it is a standard practice in the treatment of pneumonia.

It is important to note that the determination of CRP, in human medicine, is not, in itself diagnostic of any one particular disease – just the presence of inflammation. However, it has been shown that the magnitude of the rise in CRP can be used as a rough guide to the cause of inflammation with viral infections eliciting a smaller response than bacterial infections and certain immune-mediated diseases, with the highest levels seen in sepsis and burns. In some disease situations CRP has been used to distinguish between two or more potential causes. For instance, in inflammatory bowel disease CRP is either not increased or only minimally increased in ulcerative colitis but Crohn's disease shows a strong CRP response. Against expectations, CRP is not usually elevated in Systemic Lupus (possibly due to high IFN-alpha in SLE inhibiting CRP production) but if an SLE patient has a significant increase in CRP, then it may point to secondary infection.

CRP has also entered the field of prognosis in human cancer. A study showed that CRP was useful in determining prognosis in non-Hodgkins lymphoma. Elevated CRP levels are identified with poor prognosis in patients with solid cancers. Raised CRP has been shown to be associated with poor outcome/prognosis in small-cell lung cancer.

Recently, a new, more sensitive CRP test has been developed for use in human medicine, namely "High-sensitivity CRP". This test is rapidly gaining acceptance in the field of cardiovascular disease investigation in that persistent production of low levels of CRP appear to be responsible for angiopathy.

USE OF SERUM CRP IN VETERINARY MEDICINE

In general, CRP in canine serum (based on immunological tests) does not cross-react with most methods designed for humans until quite recently when a few validation studies, using a human method, were published.

Reports of the use of CRP in veterinary medicine have been around since the 1980s but very sporadic until recently. The earlier literature refers to studies that were conducted with in-house/research-linked/developed methods. This situation has now changed quite profoundly recently. First of all, it was established that some methods for human CRP cross-reacted sufficiently well with canine CRP to be validated as clinically useful and, very recently, several canine-specific CRP methods have been published and put into practice – one of which will now become available to users of the Idexx Catalyst biochemistry analyser.

So, more recently (2013-2016 literature), a number of publications have appeared that show the utility of determining serum CRP in dogs, mostly using canine-validated/specific tests:

- Cystitis in dogs – although full urinalysis will reveal the same information, a single serum test, in suspected cystitis can be very useful.
- CRP has been shown to be a much more sensitive marker of inflammation than leukocyte count in canine babesiosis.
- CRP has been shown to be associated with treatment success in canine leishmaniosis.
- Differentiating between suppurative and non-suppurative arthritis has been shown to be facilitated by serum CRP measurement.
- In suspected pyometra in bitches, serum CRP was found to be a more reliable indicator than leukocyte count.
- Elevated CRP was found to be a useful test to confirm the diagnosis and monitor response to treatment in dogs with various forms of cancer.
- In fracture healing of dogs it was found that low CRP levels were associated with cases stabilized with closed reduction.
- Post-surgical inflammatory complications in dogs, was identified by raised serum CRP.
- Measurement of CRP was found to be a useful parameter to assess the post-treatment complications in adulticide treatment of heartworm in dogs.
- Also, serum CRP can be used as a marker of endothelial arteritis and hypertension in dogs with heartworm.
- Major increases in CRP were found to be a marker of disease severity in canine parvoviral

- In pancreatitis, disease severity was positively correlated with serum CRP levels in dogs.
- In canine spirocercosis, serum CRP levels were associated with the presence of secondary neoplasia.
- Plasma CRP was found to be a useful marker for the presence of Immune-Mediated Polyarthropathy in dogs.
- Dogs with bacterial pneumonia were found to have higher CRP levels compared with dogs with other respiratory diseases.
- In canine babesiosis, serum CRP levels were high before treatment and remained high for three days after initiation of treatment.
- Serum CRP was found to be a reliable indicator of the degree of surgical trauma in soft tissue surgery.
- Serum CRP was found to be high in SRMA in a dog.
- In ovariohysterectomy by experienced and inexperienced surgeons, serum CRP was an indicator of the level of inexperience.
- In GDV, serum CRP concentrations were frequently increased.

There is, therefore, sufficient recent evidence that serum CRP has a place in the laboratory workup of canine clinical cases. However, CRP is NOT specific for any one disease and it is important to grasp the role that CRP should play in diagnostics. To this end, there are four scenarios described, below, which are not exhaustive but can be used as a model to suit the needs of the veterinary diagnostician.

SCENARIOS FOR OPTIMIZING THE USE OF CRP IN THE CLINICAL SETTING

Scenario 1

a) Problem: The dog “is not him/herself”/ is lethargic / is anorexic / has unexplained weight loss, without any overt clinical signs.
b) Role of CRP assay: To determine if there is a significant inflammatory process/pathology present somewhere in the body.

The sensitivity of CRP in this scenario is not very high as there are many diseases that can present with the above vague findings that do not have inflammatory disease. However, the specificity (for detecting significant inflammation/pathology) is quite substantial and if CRP is abnormally high, it suggests that other diagnostic approaches and procedures are warranted.

Scenario 2

a) Problem: There is a clinical finding that can have more than one cause, one (or more) of which is/are inflammatory. For example: lameness, abdominal effusion, coughing.
b) Role of CRP assay: To determine if the abnormal clinical finding has an inflammatory or non-inflammatory cause.

Again, sensitivity is not very high but specificity is good. If CRP is raised, in the presence of the clinical abnormality, then the diagnostic options are a very small focus group.

Scenario 3

a) Problem: There is an established diagnosis of inflammatory disease. For instance: Trauma (fight or HBC), snakebite, burns.
b) Role of CRP assay: To determine the severity of the pathology and possibly set a prognosis.

Scenario 4

a) Problem: Largely an extension of Scenario 3. There is an established inflammatory disease present and treatment has been initiated. For instance: canine babesiosis, pneumonia, post-operative (as in pyometra), trauma.
b) Role of CRP assay: To determine whether the treatment is/has been effective. This may require several sequential determinations.

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OUTBREAK OF CANINE DISTEMPER IN DOGS OWNED BY LOW-INCOME FAMILIES IN MOZAMBIQUE

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Although significant animal suffering caused by preventable diseases is frequently seen in developing countries, reports of this are scarce. This report describes avoidable animal suffering owing to a suspected canine distemper (CD) outbreak in unvaccinated dogs owned by low-income families in Mozambique that killed approximately 200 animals. Affected dogs exhibited clinical signs, and gross and microscopic lesions compatible with CD. Immunohistochemical staining confirmed the presence of canine distemper virus (CDV) in the kidney of one dog from the cohort. This brief communication again illustrates that large outbreaks of CDV in unvaccinated dogs occur and that large-scale avoidable suffering and threats to the health of dogs and wild canines continue. Mass vaccination supported by government and non-government organisations is recommended.

VETERINARY NOSOCOMIAL INFECTIONS

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Veterinary hospital acquired infections are a matter of fact, with an incidence of 4-9% (Mielke, 2010), compared to a human level of 5-10% (Burke, 2003). Hospitals are occupied by sick patients. Nosocomial infection outbreaks, with differing aetiologies have been documented, a significant percentage of which have been zoonotic infections (Milton et al 2015). A nosocomial (hospital acquired), infections are contracted in a hospital environment, arising between 48 hours following admission, up until 30 days following discharge. Common nosocomial infections in the veterinary hospital include urinary tract infections, surgical wound infections and infectious diarrhoea. Historically efforts focused on the control of infectious diseases such as canine parvovirus, more recently it is the control of infectious zoonotic diseases such as MRSA, *C. difficile* and MRSP that have taken precedent. It is accepted that such infections are endemic within veterinary hospitals. The longer a patient is hospitalised, the more invasive the procedure (e.g. i/v or urinary catheters), the greater the risk.

With the now necessary reduction in the use of prophylactic antibiosis for hospitalised surgical and medical cases, all practices must re-harness the old adage 'Cleanliness is next to Godliness', returning to the days of Florence Nightingale, when cleanliness and infection control was taken very seriously in all hospitals, knowing that a lack of infection control was a matter of life and death.

This presentation will tackle practical aspects of cleaning, disinfection, fomite identification and most importantly measuring the efficiency of biosecurity management using Adenine Triphosphate testing to verify cleanliness.

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PANCREATITIS: DEMYSTIFYING DIAGNOSIS AND TREATMENT

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ABSTRACT

Pancreatitis is the most common disorder of the exocrine pancreas in dogs and cats but ante-mortem diagnosis of pancreatitis can be challenging. The currently used gold standard for the diagnosis of pancreatitis is pancreatic histopathology. Limitations of this gold standard include finding clinically insignificant pancreatic lesions in dogs and cats and the possibility of missing focal lesions in the pancreas in clinically affected dogs. The accuracy of other tests for pancreatitis is evaluated against this imperfect gold standard. The Spec cPL® and Spec fPL® (IDEXX Laboratories) is the considered the most sensitive and specific test for the diagnosis of pancreatitis in dogs and cats, respectively. An in-clinic SNAP cPL and SNAP fPL test cannot confirm a diagnosis of pancreatitis, but can be used to rule pancreatitis out. DGGR lipase is a newer pancreatic lipase test with good agreement with Spec cPL® and Spec fPL® results. A diagnosis of pancreatitis relies on exclusion of conditions with similar clinical signs and using a combination of physical examination findings, pancreatic lipase tests, diagnostic imaging and pancreatic cytology. The basic principles in treatment of acute pancreatitis include replacing intravenous fluid losses, controlling nausea (using maropitant as first-line drug), providing analgesia and providing enteral nutrition.

DIAGNOSIS OF PANCREATITIS

Pancreatitis is the most common disorder of the exocrine pancreas in dogs and cats but ante-mortem diagnosis of pancreatitis can be challenging.[1] The term “pancreatitis” not only refers to inflammation (i.e. cellular infiltration) of the pancreas but also necrosis (necrotising pancreatitis) and irreversible structural changes such as fibrosis (chronic pancreatitis).[1]

Pancreatic histopathology is the current gold standard for diagnosing pancreatitis and for differentiating acute and chronic pancreatitis.[1] Neutrophilic inflammation in the absence of fibrosis or atrophy is indicative of acute pancreatitis and the presence of fibrosis, atrophy and lymphocytic inflammation is compatible with chronic pancreatitis.[1]

Histopathology is however not an ideal gold standard due to several limitations:

- Histopathological criteria for pancreatitis have not been universally standardised.[1]
- Distinction between acute and chronic pancreatitis is not always clear and many animals have evidence of both acute and chronic pancreatitis.[1]
- Histopathological lesions of pancreatitis might be clinically insignificant and can lead to a false diagnosis of pancreatitis.
    - 64% of 73 dogs undergoing necropsy for various reasons had microscopic evidence of pancreatitis.
    - 67% of all cats undergoing necropsy had histopathological lesions of pancreatitis, including 45% of healthy cats.
- Pancreatitis cannot be excluded based on histopathology as the inflammatory lesions in the pancreas can be highly localised and easily missed. It is not feasible to evaluate multiple sections of pancreas ante-mortally to increase the likelihood of finding microscopic lesions.
- Pancreatic biopsy requires invasive and expensive procedures that are potentially detrimental in patients that may be haemodynamically unstable.[1]

The ability of other diagnostic modalities to identify pancreatitis when it is present (sensitivity of a test) or exclude pancreatitis if it is not present (specificity of a test) is measured against the inadequate gold standard of histopathology. This further complicates the correct diagnosis of pancreatitis.

Signalment and risk factors

Dogs of any age, breed or sex can develop pancreatitis.[1] In the USA, Miniature Schnauzers and Terrier Breeds (esp. Yorkshire terriers) are considered to be at an increased risk to develop pancreatitis and in the UK, Cocker Spaniels, Cavalier King Charles Spaniels, Border collies and Boxers are reported to have an increased risk for chronic pancreatitis.[1]

Potential risk factors for pancreatitis in dogs include being overweight, endocrine disease, hypertriglyceridemia, prior surgery, infections, dietary factors, use of azathioprine and use of potassium bromide with phenobarbitone.[1,2] The presence of these risk factors should raise the suspicion for pancreatitis.[1] In cats the risk factors are less clear but a strong association between pancreatitis, inflammatory bowel disease and cholangitis ("triaditis") has been shown.[3]

Clinical presentation

Clinical presentation of pancreatitis varies greatly and no clinical sign or combination of signs are pathognomonic.[1] Signs of concurrent diseases may also be misleading.[1] Dogs with severe acute pancreatitis can show one or more of the following signs; anorexia, weakness, vomiting, diarrhoea and abdominal pain.[1] Severe acute pancreatitis cases might present with cardiovascular shock, DIC and multi-organ failure.[1] The majority of cats with pancreatitis present for anorexia and/or lethargy and gastro-intestinal signs are less common.[3] Dogs and cats with chronic pancreatitis can have subclinical disease or may only show mild non-specific clinical signs such as intermittent anorexia and weakness with no gastro-intestinal signs. The diagnosis of pancreatitis is then often missed because of the low suspicion of pancreatitis.[1]

Routine clinical pathology

Haematological and serum biochemical findings in dogs and cats with pancreatitis are non-specific and therefore non-diagnostic.[1] These tests should however always be performed as they are useful in the diagnosis or exclusion of other diseases with similar signs. Also, findings provide important information regarding potential systemic complications and help to guide treatment.[1]

Pancreas-specific biochemical diagnostic tests

As mentioned previously, the evaluation of the diagnostic accuracy of diagnostic tests are predicated upon having an acceptable gold standard, which currently doesn't exist for pancreatitis.[1] Results of studies of pancreatic diagnostic tests should thus be interpreted with caution. The reported sensitivity and specificity of diagnostic tests for pancreatitis varies upon the type of studies performed, the criteria used to diagnose pancreatitis (i.e. histopathological confirmation, ultrasonographic findings or overall clinical information), the type of pancreatitis (i.e. acute, chronic, mild or severe) and cut-off values chosen. Direct comparison of results among different studies is often challenging.[1]

Pancreatic lipase immunoreactivity

Pancreatic lipase immunoreactivity (PLI) is considered the most sensitive and specific serum test for diagnosis of pancreatitis in dogs and cats. It is the only assay that can measure exclusively lipase of pancreatic origin.[1] The originally developed canine PLI (cPLI) and feline PLI (fPLI) has been replaced with the Spec cPL® (IDEXX Laboratories) for dogs and Spec fPL® (IDEXX Laboratories) for cats.

Interpretation of the Spec cPL® and Spec fPL®:

	Reference Interval	*Grey zone* *	*Highly suggestive of pancreatitis*
DOGS Spec cPL®	0 – 200 ug/L	201 – 399 ug/L	≥ 400 ug/L
CATS Spec fPL®	0 – 3.5 ug/L	3.6 – 5.3 ug/L	≥ 5.4 ug/L

**** Values in the grey zone are non-diagnostic and require further testing or re-testing***

The reported sensitivity of the Spec cPL varies from 21 to 94% and the specificity varies from 66 to 100%.[1,4] The reported sensitivity of the Spec fPL varies from 54 to 100% and specificity varies from 67 to 100%.[1] In general, the sensitivity of cPLI and fPLI is lower for detecting chronic pancreatitis than for acute pancreatitis.[1] Lesions associated with chronic pancreatitis (pancreatic fibrosis and atrophy) are not expected to be associated with leakage of pancreatic enzymes. In cases with mild or chronic pancreatitis, a false negative PLI test is thus possible, but the question is also raised whether mild chronic pancreatitis is clinically significant.[1]

Canine and feline SNAP pancreatic lipase
A rapid, in-clinic, semi-quantitative, visually read test for the estimation of pancreatic lipase in serum is available for both dogs and cats.[1] A reference spot correlates with the upper limit of the reference interval and a sample spot is compared with the reference spot. Results are either normal (sample spot less intense than the reference spot) or abnormal (equally or more intense than the reference spot).[1] An "abnormal" result indicates that the actual PLI could be in the grey zone (200-400 ug/L for dogs and 3.6 – 5.3 ug/L in cats) or consistent with the diagnosis of pancreatitis (> 400 ug/L for dogs and >5.3 ug/L in cats). A diagnosis of pancreatitis cannot be based on the result of the SNAP cPL alone.[1] Further testing with the quantitative Spec cPL or Spec fPL is thus required in cases with a positive SNAP test.[1] The main use of the SNAP fPL and SNAP cPL is to rule pancreatitis OUT (i.e. a normal result makes the diagnosis of pancreatitis very unlikely).[1]

There is 90-100% agreement between the SNAP cPL and the reference Spec cPL.[1] The sensitivity of the SNAP cPL is reported between 91 – 94% and specificity 71-78%.[1] Studies on validation and clinical performance of SNAP fPL have not been reported in the literature but the manufacturer indicates that the test has 82-92% agreement with the fPLI. Theoretically the sensitivity will thus be high and similar to the Spec fPL.[1]

Trypsin-like immunoreactivity (TLI)
TLI assays are immunoassays that measure trypsinogen and trypsin concentrations in serum and is of limited usefulness in the diagnosis of canine and feline pancreatitis.[1] Raised TLI levels decrease to within the reference interval as early as 3 days in dogs and 48h in cats after the induction of pancreatitis.[1] The sensitivity for diagnosis of pancreatitis is thus low; cTLI (36-47%) and fTLI (28 – 64%).[1]

Serum lipase and amylase measured by traditional methods
The serum activities of these two enzymes are of little value in the diagnosis of pancreatitis due to their low sensitivity and specificity.[1] Many other tissues synthesise amylases and lipases (including gastric mucosa, renal, hepatic parenchyma and many others).[1]

New lipase activity assay: DGGR LIPASE
This assay uses 1,2-*o*-dilauryl-rac-glycero glutaric acid-(6' methyl resofurin)-ester (DGGR) as a substrate and has been validated for use in dogs.[1] In both dogs and cats, there is substantial agreement between the DGGR lipase and the Spec cPL and Spec fPL, respectively.[5-7] The lipase DGGR shows promise as a test to aid in the diagnosis of canine and feline pancreatitis. [1]

Diagnostic imaging
Radiographs
Abdominal radiographs are of no value for the diagnosis of canine and feline pancreatitis but remains a logical initial approach for diagnosis or exclusion of other diseases that may cause similar clinical signs.[1]

Ultrasound
Like abdominal radiographs, abdominal ultrasound is helpful in the diagnosis or exclusion of other diseases that cause similar signs to pancreatitis.[1] The ultrasonographic features of pancreatitis include hypoechoic areas within the pancreas (necrosis or fluid accumulation), increased echogenicity of the surrounding mesentery (necrosis of peripancreatic fat), enlargement and irregularity of the pancreas, dilation of the pancreatic or biliary duct and abdominal effusion.[1] Hyperechoic areas of the pancreas could indicate pancreatic fibrosis.[1] In cats, the presence of a thick left limb of the pancreas, severely irregular pancreatic margins and hyperechoic peri-pancreatic fat is supportive of pancreatitis (together with appropriate clinical signs and elevated fPL).[8]

Reported sensitivity of abdominal ultrasound is 68% in dogs and 11 - 67% in cats with pancreatitis but largely depends on the expertise of the ultrasonographer and the quality of the equipment used.[1,3] A normal pancreas on ultrasound examination is not sufficient to rule out pancreatitis in either dogs or cats. This is particularly true in mild or chronic cases where the changes are mild.[1] It is also important to not over-interpret ultrasonographic findings because many other diseases of the pancreas (hyperplastic nodules, oedema etc.) can display similar ultrasonographic findings.[1]

Pancreatic cytology

FNA of the pancreas is minimally invasive and is used increasingly for the diagnosis of pancreatitis in dogs and cats.[1] There are no studies evaluating the sensitivity and specificity of this diagnostic modality.[1] Pancreatic acinar cells constitute the majority of the cells found in normal pancreas.[1] In acute pancreatitis, cytological samples are hypercellular with the presence of entire and degenerate neutrophils and degenerate pancreatic acinar cells. Chronic pancreatitis is characterised by small numbers of lymphocytes and neutrophils and low cellularity due to replacement of normal pancreatic tissue by fibrotic tissue.[1]

As for histopathology, highly localised lesions may be missed and negative FNA results are not sufficient to rule out pancreatitis.[1]

The safety of pancreatic FNA has not been evaluated in dogs and cats with pancreatic diseases. But it is considered relatively innocuous. In healthy dogs, pancreatic FNA or surgical biopsy did not cause increases in serum cPLI or clinically detectable pancreatitis.[1]

Summary

- No diagnostic modality has 100% reliability for the diagnosis of canine or feline pancreatitis.[1]
- Exclude other diseases that have similar clinical signs.
- Maintain a high level of suspicion for pancreatitis, especially in animals that are presented with mild, non-specific clinical signs.
- Use a combination of history, physical examination findings, routine clinical pathology findings, pancreatic lipase tests, diagnostic imaging and cytology.[1]
- A combination of the clinical picture of the patient, serum PLI concentrations and abdominal ultrasound is considered the most practical and reliable methods for an accurate diagnosis or exclusion of pancreatitis.[1]

TREATMENT OF PANCREATITIS

No randomised control trials or cohort studies in different populations of animals (level A evidence) have been published to provide evidence for the use of different treatment modalities in pancreatitis.[9] The best available evidence for treatment of pancreatitis in dogs is level B evidence (retrospective cohorts, experimental cohorts of the same species and case-control studies) for the use of maropitant as first-line anti-emetic.[9] Treatment recommendations are thus largely extrapolated from experimental rodent models or general critical care principles.[9]

The basic principles in treatment of pancreatitis include

- Replacing intravenous fluid losses and correction of electrolyte abnormalities
- Controlling nausea
- Providing pain relief

Intravenous fluid therapy

Aggressive fluid therapy is the mainstay of treatment in dogs with severe pancreatitis. Vomiting and anorexia result in dehydration that generally requires intravenous fluid therapy. The pancreas is also susceptible to disturbed microcirculation as a result of increased vascular permeability due to the release of inflammatory cytokines.[9] Colloids may be beneficial if perfusion cannot be maintained with crystalloids.[9]

The is no evidence for the use of fresh frozen plasma in pancreatitis despite the purported benefits of supplying circulating α-macroglobulins, coagulation factors and anti-inflammatory factors. Fresh frozen plasma should be reserved for dogs with documented coagulation abnormalities.[9]

Anti-emetic therapy

Maropitant blocks neurokinin-1 receptors and substance P production and is an effective anti-emetic agent. Substance P contributes to the develop of visceral pain and increased capillary permeability. Maropitant is thus considered the preferred first-line anti-emetic in pancreatitis.[9] Additional anti-emetics (i.e. serotonergic agents such as ondansetron) should be used as required.[9] Even dogs that are not showing overt signs of nausea or vomiting should be treated with an anti-emetic to encourage voluntary eating.[9]

Analgesia
Inflamed and enlarged pancreas itself causes pain. Intense and prolonged pain input may result in activation of NMDA receptors and central sensitisation. Even if animals do not display typical signs of pain, it is sensible to assume that there exists a degree of pain in all dogs with acute pancreatitis. Pain scales should be used to determine the appropriate level of pain to facilitate appropriate levels of analgesia:

- Mild pain: buprenorphine or methadone at high dose-end and frequency.
- Moderate pain: buprenorphine or methadone PLUS lidocaine and ketamine infusion
- Severe to excruciating pain: epidural morphine or fentanyl infusion PLUS ketamine/lidocaine infusion.[9]

Because of the presence of hypovolaemia and dehydration in the majority of dogs with acute pancreatitis, NSAIDs and α2-adrenoreceptor agonists are not recommended.[9]

Nutrition
Evidence for early enteral nutrition in dogs with pancreatitis is not supported by robust clinical trials to date but in humans there is evidence that it reduces hospitalisation times. The premise for early enteral feeding is to improve the health of the intestinal tract. Unhealthy enterocytes are thought to perpetuate systemic inflammation.[9] Nutrition delivered directly to the intestine decrease villous atrophy, reduce bacterial translocation and decrease pancreatic inflammation.[9] Starvation can be detrimental in feline patients due to the possibility of concomitant hepatic lipidosis. Feeding should thus be instigated as soon as possible.[3] There are no current recommendations on the type of food to be administered. Avoidance of high fat-diets in dogs is logical as many of the animals are hyperlipidaemic.[9] Due to the peculiarities of feline metabolism and lack of evidence that a low-fat diet is beneficial in cats, it is generally suggested that a low-carbohydrate, high protein diet is fed to prevent the development of hepatic lipidosis.[3]

Antibiotics
Dogs with pancreatitis rarely have bacterial infectious complications and the routine use of prophylactic antibiotics should be avoided due to the risk of causing increased community resistance to antibiotics.[9] The use of antibiotics should be limited to dogs with documented or strongly suspected bacterial infection (i.e. pancreatic necrosis or aspiration pneumonia).[9]

Gastric acid suppression and nasogastric suctioning
It has been proposed that reduction of gastric acid production could decrease pancreatic exocrine stimulation.[9] Pancreatitis predisposes to the development of gastric ulceration due to hypovolaemia and local peritonitis. There is currently no evidence that reducing gastric acidity improves outcome in dogs with acute pancreatitis but if there is clinical evidence of gastric ulceration (haematemesis, malaena) or oesophagitis, gastric acid suppression is indicated. Omeprazole (2.5 mg/kg per day in divided doses) is the most effective drug at increasing gastric pH for the longest duration of time.[9]

Corticosteroids
The routine use of corticosteroids in pancreatitis is not recommended yet.[9]

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AN UPDATE ON POINT OF CARE TESTS FOR THE DIAGNOSIS OF CPV AND CDV IN DOGS AND FIV AND FELV IN CATS

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Rapid point of care (POC) tests have been available to the veterinarian for decades. However, some recent interesting findings need to be noted.

The objective of this paper is to highlight the merits and limitations of some of the more popular point of care tests used in the diagnosis of Canine parvovirus (CPV), Canine distemper virus (CDV), Feline immunodeficiency virus (FIV) and Feline leukemia virus (FeLV).

The CDV and CPV semi-quantitative antibody POC tests can be used to monitor the immune status of a vaccinated dog. The tests should be done 4 weeks after the initial vaccination program to confirm a successful vaccination. The rapid antibody tests can also be used to assist in the prognosis of a CDV and CPV infection and to determine when to administer the next vaccine booster. The CDV and CPV antigen tests use faeces or conjunctiva as the preferred diagnostic sample. A recent study revealed that the Anigen POC test does not detect the vaccine within 9 days of vaccination implying that a positive POC result is not due to the vaccine.

In the case of FIV infection there is a strong antibody response following infection. The Bionote/Anigen FIV antibody POC test uses both blood and saliva as diagnostic sample. The assay is both sensitive and specific and able to distinguish an FIV infected from vaccinated uninfected cat. However not all rapid antibody tests performed the same. The Anigen/Bionote FIV antibody test was able to identify a cat infected with FIV irrespective of whether the cat had been vaccinated or not 100% of the time as long as testing was not performed within 6 month following the primary vaccination (Westman, 2016). Saliva proved to be equally sensitive and specific to blood and may have advantages over blood in certain situations.

A recent study investigated the use of the Anigen/Bionote FeLV antigen POC test on blood and saliva samples from cats progressively, regressively and negatively infected with FeLV. The assay had a sensitivity of 57% and 54% when blood and saliva were used, respectively. Specificity was 98% and 100% for blood and saliva, respectively (Westman et al 2017). The authors justify the use of the POC test using whole blood to test for FeLV however they recommend that all FeLV positive POC results be confirmed using a real time proviral PCR which is regarded as the gold standard for FeLV testing. Because of the lower sensitivity, cats that test negative, but that have been exposed to infected cats, should also be tested by PCR. If PCR is not available two different FeLV POC kits can be used to improve the diagnosis. However more studies need to be performed before saliva will be recommended as diagnostic sample for FeLV antigen POC testing.

The merits and limitations of the 4 rapid POC tests will be discussed with special reference to the test, the best sample to use and when PCR should be used to confirm a POC result.

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A GALLERY AND DISCUSSION OF INTERESTING ONCOLOGY CASES OF DOGS AND CATS TREATED WITH ELECTRON RADIATION THERAPY

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A brief explanation of how electron radiation works to eliminate cancer cells followed by in depth discussion of the cases presented. The cases will cover squamous cell carcinomas caused by excessive solar exposure and other SCC's of different origin. Methods of controlling the solar exposure in both dogs and cats. Cutaneous Mast Cell Tumours their origin and use of radiation before surgery. Mesenchymal Cell Tumours (spindle/soft tissue cell tumours) and the advantage of electron radiation therapy before and after surgery.

RATIONAL ANTIBIOTIC USE: SHOULD YOU REALLY BE USING THAT?

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ABSTRACT

Antimicrobial resistance is one of the fastest developing and far-reaching issues of our time. Small animal veterinary practice is not untouched and has an important role to play in the prevention of antimicrobial resistance development. Most veterinarians in South Africa prescribe antibiotics empirically prior to doing investigative diagnostics. To be able to make rational antibiotic choices there is a need for geographical epidemiological data on resistance patterns of common isolates, which is lacking for small animal veterinary practice in South Africa. Other principles such as considering the true need of antimicrobials, de-escalating therapy and treating for the shortest possible duration may further improve responsible antimicrobial use. The owner, veterinarian, local bacteriology laboratory and other relevant health services all have equally vital roles to play in the success of prudent and responsible antimicrobial use.

RATIONAL ANTIBIOTIC USE

The development of antimicrobial resistance is one of the largest contemporary challenges faced by all involved in animal and human health. Antimicrobial resistance not only complicates daily clinical practice, but has far reaching effects in medicine and agriculture. The most common factors linked to the development of antimicrobial resistance relates to the misuse of antimicrobials such as under dosing, off label usage and misdiagnosis[13; 15]. The misuse of antimicrobials by companion animal veterinarians can promote the failure of the newer treatment options by selecting for resistant bacteria which also has the potential to spread to the humans sharing the animals environment[8]. A recently published paper on the antimicrobial usage patterns by small animal veterinarians in South Africa indicated that 91.16% of participating veterinarians prescribed antimicrobials empirically prior to doing laboratory tests. Participating veterinarians reported that this method of practice is influenced mainly by financial constraints of the owner. They also reported that an distressingly large number of owners present the animal only after initiating antimicrobial therapy at home themselves, to save on consultation costs[4].

Similar to the worldwide trend, South Africa has experienced concerning changes in the resistance patterns of nosocomial infections in humans. Increase in extended-spectrum beta-lactamase production, emergence and increases in carbapenem resistance, increase in multidrug-resistant *Escherichia coli* and emerging resistance in Gram-positive isolates have been reported[3]. In a study by Chipangura et al., 2017, the monitored prevalence from 1712 isolates presented over 6 years to a veterinary bacteriology laboratory was over 10% and the multiple drug resistance over 50%. In the study investigating the usage patterns of antimicrobial agents by South African small animal veterinarians it was found that 73.48% of participants did not choose antibiotics based on organ system nor did they make use of any other selection protocol to guide antimicrobial choice[4].The same study reported that 81.3% of participants indicated that they were seeing an increased patient return rate due to treatment failure, suggesting incorrect empirical antimicrobial choices[4]. Several risk factors for imprudent antibiotic use has been identified including not using local epidemiological and antibiotic susceptibility data, using broad-spectrum antibiotics when not absolutely necessary, treating contamination or colonisation rather than invasive infections, inappropriate surgical prophylaxis and excessive use of antibiotics[3]. The lack of availability of local epidemiological data, high rate of empirical antibiotic use and increase in treatment failures support that the development of antimicrobial resistance is a ubiquitous issue in small animal veterinary practice in South Africa that beseechs attention.

There are three general recommended approaches to limit the development of antimicrobial resistance namely, preventing disease occurrence, reducing overall antimicrobial drug use and prudent antimicrobial use[19]. Preventing disease occurrence relies heavily on good animal husbandry, appropriate use of efficacious vaccines and the use of infection control measures in veterinary

hospitals[19]. Prudent use of antimicrobial agents is broadly defined as the optimal selection of drug, dose and duration of antimicrobial treatment along with reduction in inappropriate and excessive use, as a means of achieving the best clinical outcome while minimising the emergence of antimicrobial resistance[14]. Improving the practice of prudent antimicrobial use may follow several recommendations[3].

Firstly, the true need for antimicrobial therapy must be logically considered and justified in every case. Viral infections, immune-mediated diseases and inflammatory conditions such as pancreatitis and neoplasia can cause pyrexia and other signs often ascribed to bacterial infection. The use of early diagnostic investigation instead of empirical antibiotics may aid in distinguishing these conditions from bacterial infections. With that said, not all bacterial infections also necessarily require treatment with systemic, or indeed any, antimicrobials. For example, incision and drainage is the preferred method for treatment of localised abscesses, with no evidence that concurrent antimicrobial therapy is necessary for resolution[19]. Several other studies have recently been published providing good evidence to question the use of antibiotics in certain conditions in which systemic antibiotics were the norm.

Superficial bacterial folliculitis is the principal reason for antimicrobial prescription in small animal practice[9]. However, the use of a topical shampoo containing 4% digluconate chlorhexidine has been shown to be at least as effective as systemic amoxycillin-clavulanic acid at 25mg/kg twice daily in a prospective, randomized and controlled study[2]. This supports the current recommendation of empirically treating canine superficial bacterial folliculitis topically in all patients in which owner and patient factors allows such practice[9]. The routine use of even topical antimicrobials were brought into question when an ear cleanser containing 2.5% lactic acid and 0.1% salicylic acid used twice daily in dogs with otitis externa due to yeast or bacterial infections was shown to resolve infection in 67.7% of cases[6].

Idiopathic acute haemorrhagic diarrhoea syndrome (AHDS; previously known as idiopathic haemorrhagic gastroenteritis) is a syndrome where serious loss of the intestinal mucosal barrier integrity occurs. Thus, empirical antibiotics were often considered part of the treatment plan for these cases. A prospective study investigating the incidence of bacterial translocation in dogs with AHDS indicated that there was no significance difference in the bacteraemia in patients with AHDS and controls. The presence of bacteraemia also did not affect disease severity, laboratory parameters, duration of hospitalisation or mortality. This study concluded that bacteraemia does not influence the clinical course or survival of cases suffering from AHDS and antibiotic therapy is not indicated to prevent sepsis[16]. An in-practice study evaluating the outcome or time to recovery in dogs with AHDS treated with or without empirical antimicrobials showed no difference further devaluing the use of antimicrobials in this condition[17].

Both feline and canine upper respiratory tract disease is self-limiting and most cases experience spontaneous resolution within 10 days without antimicrobial therapy[10]. Current recommendations are to withhold antimicrobial therapy for a 10-day observation period and only consider antibiotics if patient experience pyrexia, lethargy, or anorexia concurrently with a mucopurulent nasal discharge[10]. Treating potential pathogens in the absence of clinical disease may promote colonisation or infection with resistant species. In particular, asymptomatic bacteriuria can be found in 2 – 9% of dogs and due to lack of evidence it is recommended that these animals should not be treated[20]. For this reason the use of screening cultures is discouraged in the absence of clinical signs[19].

The second recommendation states that early and appropriate empirical therapy may reduce mortality (See Table 1). Recently the International Society for Companion Animal Infectious Diseases have published guidelines to aid small animal veterinarians in their antimicrobial choices for canine superficial bacterial folliculitis, and infections of the urinary and respiratory tracts[9; 10; 20]. Many other individual publications have also provided valuable information regarding drug dosages, course length and formulation options for different organ systems that may be preferable to alleviate selection pressure as well as offering valuable epidemiological data applicable to South Africa in some publications [1; 4; 11; 21]. The use of antibiotics according to the label is not always consistent with the principles of prudent antimicrobial use as the labelled dose and dosing interval are not always consistent with current principles of antimicrobial drug use[19].

Indication	Most common isolate	Empirical antimicrobial	Reference
Bacterial cystitis	*Escherichia coli*	Amoxycillin or trimethoprim-sulphonamide	[20]
Pyelonephritis	Culture and sensitivity is recommended	Fluoroquinolone	[19]
Feline upper respiratory tract disease	*Staphylococcus* spp. but often a mixed population	Doxycycline	[4; 10]
Canine infectious respiratory disease complex	*Staphylococcus* spp. but often a mixed population	Doxycycline	[4; 10]
Bacterial bronchitis	Culture and sensitivity is recommended	Only in severe cases whilst culture is pending, doxycycline	[10]
Pneumonia	Culture and sensitivity is recommended	Mild cases: doxycycline No evidence of sepsis: beta-lactam antimicrobial Sepsis: Fluoroquinolone combined with ampicillin or clindamycin with de-escalation once culture and sensitivity available	[10]
Pyothorax	Most commonly polybacterial and culture and sensitivity is advised	Fluoroquinolone combined with a penicillin or clindamycin with therapeutic drainage with or without lavage	[10]
Dog bite wounds	*Pasteurella canis* and pyogenic streptococci	*Only indicated in severe cases with cytologic evidence of infection Potentiated penicillin or potentiated sulphonamide	[11]
Hepatobiliary	*Escherichia coli*	Fluoroquinolone and penicillin with de-escalation once culture and sensitivity available	[18]

Table 1: Examples of currently recommended empirical antimicrobial choices for selected conditions.

Thirdly, de-escalating or tailoring antibiotics to the isolate or known susceptibility patterns. De-escalating therapy is commonly practiced in human medicine where the initial broad-spectrum empirical antibiotic choice is changed to a drug with a narrower spectrum matching the susceptibility pattern of the isolate once the culture and sensitivity results are available[19]. In human medicine, the use of de-escalation therapy has not been associated with a negative clinical impact or improved patient outcome, including for life-threatening conditions such as sepsis[7]. This is now also recommended in veterinary medicine where a broad spectrum empirical choice should be changed to a narrower spectrum drug once the culture and sensitivity results become available[10; 19].

Lastly, a shorter duration of therapy is currently recommended. It is a common misconception that there is a minimum duration or course that an antimicrobial drug should be used for to reduce the emergence of resistance. Although there is limited evidence to guide the duration of therapy for most conditions in animals recent randomised trials in humans have supported the use of shorter treatment durations in many infectious syndromes[19]. Support for shorter treatment durations in veterinary medicine have also been reported where for example three days of trimethoprim-sulfamethoxazole for the treatment of uncomplicated bacterial cystitis did not differ in cure rate from cephalexin treatment for ten days[5]. Shorter treatment durations are beneficial in that there is reduced exposure of commensal bacteria to antimicrobial drugs, improved owner compliance, reduced cost and inconvenience, and a lesser chance of adverse drug effects[19]. It is uncommon for most types of bacterial infections to occur without a predisposing condition and repeated courses of antibiotics is not only clinically futile for disease cure but leads to increased risk of antimicrobial resistance[19]. Attention to the underlying cause alone might lead to resolution of a secondary bacterial infection without the need for antimicrobial drugs[19]. Periodic antimicrobial dosage regimens such as prescribed for

recurrent lower urinary tract infections and superficial bacterial folliculitis are discouraged due to a failure to meet sound pharmacokinetic-pharmacodynamic principles, lack of evidence for efficacy and the impact on antimicrobial resistance[19].

In general, several other principles may apply for prudent antimicrobial use. It is not recommended to use human generic drugs when a licensed veterinary drug is available, especially when efficacy and bioavailability data for the generic drug is lacking[19]. Rules restricting the use of compounded products by veterinarians when registered veterinary products are available have also recently been brought about[4]. When compounded drugs are used specific attention should be paid to the concentration of the active ingredients, pH stability and quality of the raw starting materials[12]. Some antibiotics are used for their nonantimicrobial properties such as anti-inflammatory, immunomodulatory and prokinetic properties. However, this should be discouraged due to the lack of compelling evidence of efficacy, little data pertaining to possible adverse effects and many alternative drug options[19].

Resistance patterns differ geographically and the knowledge of the resistance patterns present in a specific area may aid in development of recommendations regarding good empirical antibiotic choices for different infections[4]. Unfortunately, this kind of epidemiological data pertaining to small animal veterinary practice in South Africa is lacking. It is recommended that veterinarians and their local bacteriology laboratory collaborate and share information regarding common isolates, susceptibility patterns and the emergence of resistance when identified. Special attention must be paid to species such as *Staphylococcus pseudintermedius*, Campylobacter, Salmonella and *Escherichia coli*[8]. This will equip either parties to combat the development of antimicrobial resistance and practice successfully with guidance in appropriate empirical antimicrobial choices per organ system or condition.

Besides making rational choices regarding the use of antimicrobials, owner education is imperative in the struggle against antimicrobial resistance. It must be explained to the owner in layman's language what the antimicrobial is, why it is being prescribed or is deemed unnecessary, how antimicrobial develops and what the impact is on public health.

Antimicrobial resistance is an emerging and contentious issue faced by all health professionals, including small animal veterinarians today. Limiting the use of antimicrobials as far as possible and making rational choices when antimicrobials are indicated may aid in preventing or at least slowing the development of antimicrobial resistance. The owner, veterinarian, local bacteriology laboratory and other relevant health services all have a vital role to play in the success of prudent and responsible antimicrobial use.

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CRITICAL CARE NUTRITION IN SMALL ANIMALS PART I

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ABSTRACT

The basics of critical care nutrition apply throughout all disease states. These basics include, an initial nutritional assessment, prescribing an appropriate diet for that specific condition, taking the patients present state in mind, setting goals for the nutritional intervention, and finally assessing whether these goals are in fact being met. The goals of critical care nutrition are to meet resting energy requirements as well as to supply sufficient essential and conditionally essential amino acids and all other micro and macro nutrient needs. A plethora of methods are available to us to achieve this, including a variety of diet and tube options for the still preferred enteral nutrition route as well as multiple solution options and peripheral and central line options for parenteral nutrition. In some cases, partial parenteral and enteral nutrition best supplies the patient's nutritional needs. Nutritional assessment and monitoring, and methods, indications and complications of critical care nutritional interventions are all discussed in this lecture.

INTRODUCTION

Adequate caloric intake has been positively associated with time to hospital discharge **Error! Reference source not found.**, **Error! Reference source not found.**. Malnutrition on the other hand is associated with increased morbidity and mortality **Error! Reference source not found.**, **Error! Reference source not found.**. Only a minority of dogs and cats ever achieve adequate nutritional intake voluntarily in hospital **Error! Reference source not found.**. Excess calories can also impact negatively on survival **Error! Reference source not found.**, **Error! Reference source not found.**, **Error! Reference source not found.**. This means that planned, monitored and revaluated nutrition interventions are necessary for each intensive care patient.

NUTRITIONAL ASSESSMENT

A nutritional assessment of a critical care patient includes the following, the degree of weight loss over a defined period, body condition scoring, muscle condition scoring and a full dietary history **Error! Reference source not found.**. Further testing may include blood glucose, haematology, serum albumin, transferrin, Na, K, Cl, PO_4^{2-}, Ca, Mg, serum chemistries to check for organ disease, creatinine kinase, adrenal function and thyroid function tests depending on the needs of each individual case **Error! Reference source not found.**,. In one study baseline admission serum albumin, but not BCS and lymphocyte count, correlated with risk of mortality in critically ill dogs **Error! Reference source not found.**. In cats decreased CK activity resolved after adequate introduction of food indicating its possible use as a marker of malnutrition in this species **Error! Reference source not found.**. In another study both hypoalbuminemia and low serum transferrin values in dogs both on admission and in the case of transferrin, also after feeding, correlated with a negative outcome **Error! Reference source not found.**.

Subjective global assessment (SGA)
In humans, a SGA has been found to be a useful tool to assess nutritional status and the need for intervention **Error! Reference source not found.**. The following questions are asked. Is nutrient assimilation restricted by, decreased intake, maldigestion or malabsorption? What are the effects of malnutrition on organ function and body composition? What is the effect of the disease process on nutrient requirements? The assessment conclusions may be that the patient is well nourished, moderately well-nourished or at risk of becoming malnourished, or severely malnourished.

An adapted SGA for dogs and cats has been suggested which includes diet composition history, food intake history, weight loss vs time, changes body composition (BCS/MCS), GI signs, presence of oedema or ascites, hair coat changes, exercise tolerance, physical activity and metabolic demands of the underlying disease state. There are many similarities between this modified SGA and a CCECAI score explained below. A modified CCECAI score is currently been investigated for use in small animals.

Modified Canine Chronic Enteropathy Clinical Activity Index (CCECAI)

The CCECAI is specifically designed for inflammatory bowel disease (IBD); however, it encompasses most factors that would initiate a nutritional intervention. If slightly modified from its use in IBD, it enables the application of a clinical score to an animal's current ability to sustain an adequate nutritional status [Error! Reference source not found.]. The higher the score, or an increasing score, the more likely a nutritional intervention is necessary, as mentioned this score is busy undergoing clinical evaluation.

1. Attitude or activity where: 0=normal, 1=slightly↓, 2=moderately↓, 3=severely↓
2. Appetite levels where: 0=normal, 1=slightly↓, 2=moderately ↓, 3=severely↓
3. Vomiting where: 0=normal, 1=mild change (1/week), 2=moderate change (2-3/week), 3=severe change (>3/week)
4. Stool consistency where: 0=normal, 1=slightly soft faeces, 2=very soft faeces, 3=watery diarrhoea
5. Stool frequency where: 0=normal, 1=slightly↑ (2–3/day) or faecal blood, mucus, or both, 2=moderately↑ (4-5/day), 3=severely↑ (>5/day)
6. Weight loss where: 0=none, 1=mild (<5%), 2=moderate (5-10%), 3=severe (>10%)
7. BCS (9 piont scale) where: 0=BCS 4-5, 1=BCS 3or6 2=BCS 2or7 3=BCS 1or>7
8. MCS where: 0=(MCS3) 1=(MCS2) 2=(MCS1) 3=(MCS0)
9. Serum albumin levels: 0=>26g/l, 3=< 26g/l or serum transferrin levels 0=>180mg/dl, 3=<180mg/dl Nakajima 2014
10. Ascites, pleural effusion or peripheral oedema: 0=normal, 1=mild, 2=moderate, 3=severe
11. Presence of skin coat abnormalities: 0=no skin coat changes, 1= mild skin coat changes, 2= moderate skin coat changes, 3= severe skin coat changes.

The changes from the original CCECAI are found in criteria number 7, 8, 9 and 11.

The scores of these 9 variables are summated together to give the index where:

- 0-3 likely to sustain nutritional status without assisted nutrition, feed
- 4-5=assisted nutrition possibly required, feed and monitor closely
- > 6=assisted nutrition required
- >10=more aggressive nutritional intervention required

GOALS OF INTENSIVE CARE NUTRITION

These are to supply sufficient calories to prevent protein catabolism, supply sufficient essential and conditionally essential amino acids to aid healing, organ function, immune function, drug metabolism and prevent metabolic complications. Restore and / or sustain electrolytes such as Na, K, Cl, Ca, PHOS and Mg. Restore and sustain micro-nutrients such as Vit-B1, Vit-B12, Vit-K and Vit-D. Avoid complications of overfeeding such as hyperglycaemia, vomiting and diarrhoea. Avoid complications of over hydration.

FEED TO RESTING ENERGY REQUIREMENTS

Resting energy requirements (RER) = calories needed to maintain homeostasis at rest in a thermoneutral environment during a post absorptive state. It is a predictive energy equations based on calorimetric studies:

For patients < 2 and > 30 kg **RER=70 x (current body weight in kg)$^{0.75}$**

For patients between 2 and 30 kg **RER=(30 x current body weight in kg) +70**

RER adjustments in disease

Most disease states only require meeting RER. This is because of decreased activity, hospitalisation, sedation, in house temperature control, vs increased disease utilisation' [Error! Reference source not found., Error! Reference source not found., Error! Reference source not found.]. Serial nutritional monitoring allows for adjustments from a RER baseline. In rare cases, such as severe burn wounds RER can be adjusted. The recommended RER for burn wounds may be as much as 2 X RER [Error! Reference source not found.].

ROUTES OF NUTRITIONAL SUPPORT

Nutritional support can be supplied by EN, PN feeding, or a combination of both. EN routes include assisted feeding and naso-oesophageal, nasogastric, oesophagostomy, gastrotomy (PEG or Laparotomy), and jejunal tubes. The various tubes can also be placed post pyloric using endoscopic techniques. Parenteral nutrition can be supplied by a peripheral or a central line depending on the osmolality of the food.

ENTERAL NUTRITION (EN)

Indications and benefits of enteral nutrition

For a patient to be considered for enteral feeding the attending clinician must first establish that it is necessary, the patient must be conscious and have a gag reflex; unless a jejunostomy or post pyloric tube is to be considered. The patient must also have a functional bowel and clotting system; and must be able to undergo chemical constraint or GA depending on the type of nutritional intervention required.

Benefits enteral nutrition (EN) over total parenteral nutrition (TPN)

EN stimulates splanchnic blood flow which reduced intestinal mucosal atrophy. It stimulates secretory IgA which reduces pathogenic bacterial adherence to mucosa. Helps support commensal bacterial populations and stimulates intestinal motility. EN maintains structural and functional intestinal integrity which reduced GIT bacteria translocation **Error! Reference source not found.**. There are overall fewer complications than TPN.

Complications of EN

Complications of EN include aspiration pneumonia, fluid overload and nutritional overload causing, hyperglycaemia, excessive nitrogenous waste, vomiting, regurgitation and diarrhoea **Error! Reference source not found.**.

Naso-oesophageal tube feeding (NETF)

NETF is simple and does not require general anaesthesia. Requires correct technique (a video will be shown in the lecture). Usually used for < 5 days of nutrition. It is inexpensive, requires gag reflex and is complicated by vomiting, bleeding, and rhinitis. Thin tube size limits formula consistency to liquid, 3-5F, or liquidised thin gruel, 6-10F. Continuous or intermittent feeding is possible using pumps but no difference in outcome has been noted. Usually requires a collar

Oesophagostomy tube feeding (EGTF)

EGTF is quick and simple but requires GA and correct technique (a video will be shown in the lecture). Used for > 5 days and up to 4 weeks. Improves comfort and tube size compared to NETF. Patients are prone to oesophagostomy site infection. Complicated by vomiting and aspiration

Gastrotomy tube feeding (GTF)

Requires GA. Can be placed percutaneous or via laparotomy. GTF bypasses the oesophagus. Requires correct technique and can be used for > 4 weeks to 2 years. It improves comfort and tube size compared to nasal tubes. Patients are prone to gastrotomy site infection and life threatening intra-abdominal leakage especially with premature removal, < 14 days. May delay gastric emptying for up to 5 days post insertion **Error! Reference source not found.**.

Post pyloric feeding tubes (PPFT)

These tubes bypass oesophagus and the stomach. They can be placed using flouroscopic or endoscopic techniques. They include nasojejunal, oesophagojejunal, gastrojejunal. Require GA and an adequate clotting system. Their indications include upper GIT functional abnormalities with risk of aspiration and protracted central, hepatic, renal, gastric, duodenal, peritoneal, or pancreatic induced vomiting. They are also used if a jejunostomy tube is contra-indicated.

Jejunostomy tube feeding tube

Requires GA. Usually placed via laparotomy but endoscopic techniques are described. It bypasses oesophagus and stomach and requires correct technique. Indications include high risk of aspiration, gastric outlet obstruction and pancreatitis. They are prone to jejunostomy site infection (up to 40% complications). Complicated by premature removal, < 14 days, which may include life threatening intra-abdominal leakage.

Basic tube placements will be described during the lecture.

Selecting formulas for enteral nutrition

Tube size dictates diet consistency that can be used. Nutritional state and disease state determine type of diet. Irrespective the diet needs to be calorie dense (1-2 kcal/ml) and meet all the nutritional requirements of the patient. Osmolality does not appear to be NB for enteral feeding **Error! Reference source not found.**. Remember to select quantity based on % RER elected to feed. **The fluid portion of the diet must be accounted for in total patient fluid load (+/-60ml/kg/day).** Please see section on specific diseases for selection of diet type

Basics of a convalescent diet

High quality and quantity amino acid profile. Some amino acids become conditionally essential during disease these include various branch chain amino acids, glutamine, and arginine Error! Reference source not found., Error! Reference source not found.. Unless contraindicated high fat content, low carbohydrate (CHO) content. This reduces the incidence of insulin resistance, hyperglycaemia, respiratory dependence on carbon removal and refeeding syndrome Error! Reference source not found.. High Ω3 FA, high in essential vitamins, minerals, and electrolytes, usually low fibre. An example of such a diet is a mix of: Turkey livers, pork, chicken (10%), maize, fish oil, minerals, taurine, vitamins, and trace elements. This diet has metabolisable energy: 32% from protein, 58% from fat and 10% from CHO.

Types of enteral diets

Diets requiring digestion

Recovery type, post trauma diets-semi liquid diets, blenderised canned or dry food mixed with water or other liquid, commercial human liquid diets. Note human diets are nutritionally inadequate, may be toxic if they contain substances such as xylitol.

Diets requiring less digestion

Blenderised hydrolysate diets for cases of IBD with parenteral B12 supplementation.

Recovery or post trauma diets in South Africa

- Royal Canine Recovery c/f 8Fr tube
- Hills a/d c/f > 8 Fr tube
- Iams Maximum-Calorie c/f > 10 Fr tube

PARENTERAL NUTRITION (PN)

Indications and benefits of PN

PN is indicated in patients that cannot tolerate total enteral feeding such as those with intractable vomiting and diarrhoea where jejunostomy tubes are contra-indicated, patients in a coma or under prolonged GA and patients without a gag reflex (poisonings, neurological disease). Patients must be malnourished to gain a positive benefit. PN is a short-term intervention only. PN can be admistered via a central or peripheral catheter depending on the osmolality of the solution chosen. Catheters are dedicated single lumen or multiple lumen catheters. They are placed using a sterile technique in a closed system only. Polyurethane or silicone catheters are less thrombogenic. Solutions of < 850 mOsmol/L can be administered peripherally Error! Reference source not found.. PN requires nursing facility and point of care serum chemistry analysis to avoid metabolic complications.

Complications of PN

Complications of PN include fluid overload, extravasation and thrombophlebitis, sepsis, and metabolic complications. Metabolic complications include deficiencies and excesses in amino acids, carbohydrates or fats, electrolyte, vitamin, and minerals. Dextrose should not exceed 4mg/kg/min to avoid causing hyperglycaemia. Omega 6 based lipid emulsions should be limited to 2g/kg/day (30-40% total calories as concerns exist as to fuelling inflammatory cascades Ill animals have increased essential amino acid and conditionally essential amino acid requirements Error! Reference source not found., Error! Reference source not found., human solutions do not meet these requirements or require fluid overloading to do so. Thrombophlebitis and sepsis are also of great concern and usually presents with the patient becoming febrile and exhibiting a neutrophilia on haematology.

Some of the concerns are solved by limiting the PN nutrition to the lesser of energy requirements or fluid overload. Meaning you stop at energy requirements provided you are not fluid overloading the patient. In this case, amino acid requirements are seldom met and extra protein will need to be supplemented beyond the short term.

Determining daily amounts for PN solutions

1. Calculate **RER** = $70(kg)^{0.75}$ or 30(2-30kg)+70=kcal/day

2. Determine max safe daily fluid load = 60ml x patient weight in kg.

3. Calculate energy from dextrose, lipids and protein per ml PN fluid.

b. Lipid=10 kcal/gram
c. Dextrose=3.4 kcal/gram (usually dextrose is used as the CHO source)
d. Non-protein-energy = lipid + dextrose / 100ml (this figure is supplied)

4. Divide RER by energy/ml=ml required.

5. If ml required > max safe fluid load give max safe fluid load only, if not give RER.

6. Calculate protein needs per day where:

   RER/100 x **protein needs/100kcal**=g **protein per day**

   Protein needs per 100kcal/day

- Standard = **4-5** dogs, **6** cats
- Hepatic renal = **2-3** dogs, **4-5** cats
- Sepsis = **5-6** dogs, **6-8** cats

7. To calculate total protein supplied divide % protein concentration of the solution by 100 to get grams of protein per ml. For example, a protein content of 8.5% = 0.085g protein / ml, therefore g protein needed / day / 0.085 = ml / day to meet protein requirements. This is likely to be more than the maximum safe amount. Actual protein supplied = grams protein per ml x ml infused.

8. Extra protein supplementation needed= g protein needed per day – actual protein supplied. Actual protein supplied = ml infused x 0.085 in this case.

9. Total liquid requirement-parenteral nutrition ml=remaining liquid requirement

Example Fresenius ITNPAED 106 10kg Patient TPN (2.3 g protein / 63.91 NPE (kcal) / 100 ml)

1. RER = 30 (10) + 70 = 370 kcal / day
2. Max safe fluid load = 600ml (60 x 10)
3. Energy per ml = 2.3x4/100 + 63.91/100 = 0.092 + 0.6391 = 0.7311kcal/ml
4. RER / kcal/ml = 370 / 0.7311 = 506 ml
5. Give RER, max safe fluid load not exceeded.
6. Protein needs = 5 x 370/100 = 18,5 g / day
7. Protein supplied by 506 ml = 5.06 x 2.3g= 11,638 grams
8. Extra protein supplementation needed = 18.5 – 11.638 = 6,82 grams
9. Supplement this with EN or short term use only but monitor safe maximum
10. Fluid component of feed 506 ml. Subtract this value from daily fluid requirements.

COMBINING PN AND EN

Dogs and cats that received partial PN and EN had a survival advantage in one study compared to only EN **Error! Reference source not found.**. In another study, it was shown that concurrent EN and PPN improved GIT integrity and immunity compared to EN alone **Error! Reference source not found.**.

CRITICAL CARE NUTRITION IN SMALL ANIMALS PART II

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ABSTRACT

The basics of critical care nutrition were covered in Part I. In Part II a few disease states are briefly discussed including, nutrition in the septic patient, nutrition in acute liver disease, nutrition in kidney disease, nutrition in pancreatitis and refeeding syndrome. Although the basic principles of critical care nutrition apply in all disease states, some require specific interventions and monitoring.

INTRODUCTION

The basics of critical care nutrition apply throughout all disease states. These include serial initial nutritional assessment. Prescribing an appropriate diet for that specific condition taking the patients present state in mind. Setting goals for the nutritional intervention and then assessing whether these goals are in fact being met. When a diagnosis is possible nutritional interventions and monitoring can be further and specifically tailored to enhance patient outcome.

NUTRITION IN SEPTIC PATIENTS

Correct fluid balance and electrolytes. Select appropriate feeding method, supply adequate nutrition based on resting energy requirements (RER), control muscle catabolism using higher protein recommendations, 6g / 100kcal dogs, and 8 g / 100 kcal cats. Control hyperglycaemia or hypoglycaemia, do not add parenteral nutrition (PN) unless 50% RER are unachievable with enteral nutrition. If possible monitor and respond to perturbations in glucose, albumin, transferrin, electrolytes, fluid load, and organ failure in a serial fashion [24].

NUTRITION IN ACUTE LIVER DISEASE

Correct fluid balance and electrolytes, control gastritis or gastric ulceration, control nausea or vomiting. Ensure adequate supply or supplement Vit-K, Vit-B, Vit-E, essential amino acids (EAA) for example L-carnitine. Lactulose, Zn-gluconate and phosphatidyl choline. Select appropriate feeding method [10] :

- Is the patient encephalopathic? In house NH3
  - No-feed high quality, moderate to high quantity. protein to aid recovery
    - Royal Canine Recovery c/f 8 Fr tube
    - Hills a/d c/f > 8 Fr tube
    - Iams Maximum-Calorie c/f > 10 Fr tube
    - Blenderised Moderate Protein to High Protein diet
    - In a serial fashion monitor NH_3
  - Yes-feed high quality reduced quantity, high quality, protein to reduce encephalopathy
    - Hills l/d canine 8Fr / feline 10Fr
    - Royal Canin Renal LP canine 8Fr / feline 8Fr

NUTRITION IN KIDNEY DISEASE

Correct fluid balance and electrolytes, control gastritis or gastric ulceration, control nausea. Select appropriate feeding method. Control uraemia and hyperphosphatemia. Use a reduced to low protein, high fat low sodium and low phosphorus diet proportional to IRIS-KIDNEY staging [10] .

Diet options:

- Hills K/d canine 10Fr / feline 10Fr
- Royal Canin Renal LP canine 10Fr / feline 10Fr

- Blenderised dry renal formulas c/f > 10Fr
- Use lower protein calculations for PN

NUTRITION IN ACUTE PANCREATITIS

Correct fluid balance and electrolytes, control nausea and vomiting, control blood glucose. Select appropriate feeding method. Early enteral nutrition via oesophagostomy tube had fewer complications than parenteral feeding in one study [22]. EN has greater literature support than PN in acute pancreatitis [30]. Use a moderate protein, low fat, low fibre highly digestible diet.
Examples are:

- Royal canin GI low fat canine 8Fr
- Hills canine i/d low fat canine 10Fr
- Hills feline i/d feline 10Fr
- Iams low residual feline 10Fr

REFEEDING SYNDROME

Prolonged starvation leads to a decrease in potassium, phosphate, and magnesium intake. Serum values may stay normal at the expense of intracellular levels. Insulin activity decreases, intracellular ATP production decreases and the patient's metabolically active lean muscle mass decreases. Upon re-feeding insulin activity increases and ATP production resumes. The production of ATP requires various cycles to reactivate consuming substrates. Phosphate is also consumed in the production of these energy molecules at a rate of three to one and 2.3DPG at a rate of two to one. Higher insulin levels drive already depleted serum potassium intracellular worsening the hypokalaemia.
In order of most common and most consistent refeeding syndrome abnormalities are hypophosphatemia, hypokalaemia, hypomagnesaemia and hypocalcaemia, and thiamine deficiency; causing anaemia, bradycardia, hypothermia, tachyarrhythmia, and neurological disease respectively. The treatment of refeeding syndrome is prediction of deficiencies and serial monitoring and supplementation of deficiencies, usually intravenously and orally [8].

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FLUID THERAPY

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ABSTRACT

Fluids are a double edged sword. Too much and too little are both detrimental to health. Unfortunately there is no perfect tool to assess fluid therapy and careful assessment and regular reassessment is required. We have a large choice of fluids and the perfect fluid does not exist. This talk will address the controversies of fluid therapy and attempt to give rational advice and guidance on how we should use fluids. It important that fluids are given on a goal directed approach. Fluids should be given to achieve a resuscitation end point not just a particular volume. This approach along with cardiovascular support is vital for successful management of patients.

FLUID THERAPY

Veterinary medicine is filled with debate on which is the most appropriate therapy for a patient. Fluid therapy is no different within this context. Sixty percent (40l) of the body is made up of water. This water is divided into two main compartments namely the intracellular and extracellular compartments. The intracellular compartment is the largest and holds 60% (25l) of total body water. Intracellular fluid has potassium as the dominant cation while magnesium and sodium make up the remainder. The dominant anion is phosphate followed by smaller quantities of sulphate. The protein content of cells is 4 times higher than extracellular fluid. The extracellular fluid makes up 40% (15l) of total body fluid and is divided into two components – extravascular and intravascular components. The extravascular component consists of 66% (10l) of fluid while the intravascular component is 33% (5l). The intravascular portion is then divided again into two – red cells and plasma. The dominant cation of extravascular fluid is sodium with smaller quantities of potassium and calcium. The dominant anion is chloride followed by bicarbonate. These differences are shown in Table 1. Fluid is distributed within this system based on osmotic pressure modified by selectively permeable membranes, maintenance of elctroneutrality, maintenance of electrolyte constitutes of the various compartments and the availability of free water.

Cation/Anion (mOsmole/l)	Intracellular Fluid	Extravascular Fluid	Plasma
Na	14	142	146
K	140	4.0	4.2
Ca	0	2.4	2.5
Mg	31	1.4	1.5
Cl	4	108	105
HCO_3	10	28	27
HPO_4	11	2	2
SO_4	1	0.5	0.5
Protein	4	0.2	1.2
Total Oncotic Pressure	302.2	301.8	302.9
Total Ionic Osmotic	281.3	281.3	282.6

Table 1: Composition of Body Fluids

All fluid therapy is generally administered into the intravascular compartment and from here it distributed and managed. A complete understanding of the makeup of the fluid we administer is important to understand how these fluids will distribute within the body. Some important concepts are that water usually follows a concentration gradient and is not actively moved between compartments. Water moves to maintain osmotic neutrality between compartments. This means that if sodium is pumped from one compartment to another an equal volume of water will follow.

Water is known to distribute freely between all body water compartments. Pure water itself is not generally administered but 5% dextrose water (similar osmolarity to plasma) can be given. Once the glucose has been removed free water is then available for redistribution. This fluid readily

redistributes between all compartments. Most crystalloid solutions redistribute between the compartments with the net effect that between 3 to 5 l of crystalloids are required to increase plasma volume by 1 l.

At this point in time it is important to revisit an important pathophysiological concept of extravascular water. The accumulation of extravascular water is also known as oedema. When crystalloids are given, only 20 – 30% remains in the vascular space after an hour. This means that the remaining 70 – 80% has accumulated in the extravascular space. If the extravascular fluid accumulated in the lungs it would be known as lung oedema. The concept that the rapid administration of crystalloid fluids can result in lung oedema has been known since the Vietnam War where this was called Denang Lung. As lung oedema impairs oxygenation of blood so does oedema in other organ systems impair oxygen delivery to tissues. The amount of crystalloids infused in trauma patients is directly correlated to the development of abdominal compartment syndrome. And so we can now enter the debate are crystalloids and/or colloids better for our patients?

It is important to be reminded that no study is yet to show that more patients will survive if colloids are used compared to crystalloids and vice versa. What has been shown is that hypovolaemia is detrimental to our patients and specifically if hypovolaemia is coupled to anaemia. In the setting of acute blood loss the restoration of circulating volume is more important than necessarily which fluids are used to do it.

Saline has been recommend as a resuscitation fluid. It has been recommended as the fluid of choice in patients with hyperkalaemia as it contains no potassium. This has been debated as the acidosis (hyperchloraemic acidosis) it produces may further exacerbate the hyperkalaemia. In the treatment of hyperkalaemia, bicarbonate is given to cause alkalosis. This results in hydrogen ions being transported out of cells in exchange for potassium being transported into cells to correct the alkalosis. As saline causes an acidosis, hydrogen is now taken up by cells and potassium released. The acidosis may further exacerbate the acidosis of trauma when used during the resuscitation of patients. For these reasons, balanced resuscitation fluids are preferred – Ringers lactate, Balsol, etc. Saline has an important role to play in patients with hyponatraemia.

To correct dehydration – fluid is required to move from the vascular compartment through all the compartments and into cells. In order to achieve this effectively free water along with the required missing electrolytes are required. The cause of dehydration will determine what electrolyte imbalances are present. Fluid can be moved into cells more effectively with glucose and potassium than with higher sodium containing fluid. Maintenance fluids are high in potassium and usually contain glucose which makes them suitable except that potassium cannot be infused rapidly into the vascular compartment. A half strength Darrow's solution may be a suitable alternative with moderate potassium and glucose concentrations.

The kidneys continuously filter plasma and produce urine. They excrete electrolytes and waste products. The kidneys require a continuous supply of water. Colloids do not supply much free water and patients require free water for normal renal function. Resuscitation fluids are generally high in sodium and these fluids are often used to maintain patients for days in the clinical setting. The high sodium load results in diuresis as the sodium is dumped through the kidneys with water. Secondly, resuscitation fluid either contains no potassium or very little potassium. Hypokalaemia is commonly seen in critically ill patients who require supplementation. Maintenance fluids are high in potassium, low in sodium and contain free water once the glucose is utilised metabolically. In critically ill patients it does make sense to use a combination of resuscitation and maintenance fluids as part of standard therapy. The maintenance fluids are run at maintenance rates while the resuscitation fluids are used to restore intravascular volume and maintain circulation. They may be continuously or intermittently administered. Maintenance solutions contain magnesium and calcium. Magnesium is an important ion and should be monitored and supplemented in critically ill patients. Maintenance solution due to their high osmolarity should not be administered through a peripheral line as tissue necrosis may follow.

Hypertonic saline has been advocated as a resuscitation fluid in trauma patients. Recent evidence supports its use as a resuscitation fluid in cranial trauma patients and may be associated with better outcomes than conventional fluid therapy. The hypertonic saline causes dehydration of the brain or rather reduces or prevents brain oedema due to the hydroscopic effect. Small volumes are required of

hypertonic saline for resuscitation. The consequence is a massive sodium load that needs to be eliminated afterwards and the diuresis that follows. A hypercloraemic acidosis is also seen.

In cranial trauma patients and patients with pulmonary contusions high volume administration of resuscitation fluids can result in brain oedema and pulmonary oedema. Fluids should be used cautiously in these patients and a low volume resuscitation should be considered. Starches are also tremendously useful in these patients as they should remain in the vascular compartment and not leak out. Another scenario is the patient with a haemorrhagic gastro-enteritis. They usually present a haemoconcentrated with a normal albumin and total protein. The natural inclination is to give them lots of crystalloids for resuscitation with an invariable result of pulmonary oedema. A resuscitation plan based more on colloids and not ignoring the requirement for crystalloids can be used successfully. A similar plan for haemoconcentrated babesia patients may be relevant.

Albumin is vital for life. In critically ill patients albumin usually decreases rapidly as the endothelium becomes dysfunctional. Albumin usually sits in balance between central circulation and extra-vascular compartments. Normally it takes 24 hours to complete the cycle. With the rapid exodus in sepsis, albumin decreases. Administering frozen plasma to increase albumin is never very successful as large volumes are required and the exodus continues. Human concentrated albumin solutions have been used to increase albumin levels but again these do not result in long term increases. When the patient gets better and endothelial function returns, albumin levels usually rise rapidly. Starches are useful to use in these patients as they are larger than albumin and are less likely to leak out. This results in a better maintenance of circulating volume. If plasma osmolarity is maintained at normal values, the liver will not synthesise albumin. Colloid administration should be decreased or “weaned off” over a day or two to allow the liver to start production of albumin.

Liver failure is often associated with coagulation disorders. Many rodenticides contain warfarin like products that result in coagulation disorders. The administration of fresh frozen plasma in these patients can be life-saving.

A number of artificial colloids are available on the market. They are sold as hetastarch, pentastarch, tetrastarches and dextrans. The dextrans are older colloids and are considerably cheaper than the modern generation of starches. Alergic reactions are known to occur with the gelatins. The hydroxyethyl starches are a polymer of amylopectins that differ by molecular weight and branching of the chains. This determines their clinical properties in the patients. Amylopectin consists of a branching chains of α-1,4-linked glucose and α-1,6-linked glucose. This is easily hydrolysed by plasma amylases. The types of starches are determined by its degree of substitution. The hydroxyl group of the glucose subunits are substituted for hydroxyethyl groups. The substitution improves the stability of the compounds. The substitution is expressed as a ratio. Hetastarches have a high degree of substitution 0.6 – 0.7, pentastarches moderate substitution 0.5 and tetrastarches low substitution 0.4. The molecular weight for the starches is also described. The starches can have a broad range of weight from a few thousand to several million Daltons in weight. The starches are then described as hetastarch 450/0.7, pentastarch 200/0.5 (Haesterile) and tetrastarch 130/0.4 (Voluven). The lower substitution results in less accumulation in tissues and more rapid elimination form the body. This also reduces its effect on coagulation. Manufacturing processes have improved and the variation in molecular size of modern generation of starches is small. This makes the lower molecular weight and low substituted molecules more suitable. Renal failure and an increase in mortality has been associated with the use of hydroxyethyl starches.

The assessment of adequate intravascular volume in critically ill patients and patients undergoing anaesthesia is vital in ensuring an adequate circulation. We are all fully aware of the consequences of hypovolaemia and hypervolaemia both of which are associated with adverse outcomes. Hypovolaemia is present in 50% of human intensive care patients and is often occult and difficult to detect. The haemodynamic measurements of filling pressures, urine output and biochemical indicators are misleading and poor indicators of central blood volume. Some studies have found no correlation between mean arterial blood pressure and heart rate, systemic vascular resistance and cardiac index while others have found a weak correlation. Central venous pressure has been traditionally used as an indicator of fluid load and is a better indicator of fluid load than blood pressure. Central venous pressure should always be assessed in relation to a fluid challenge as a single reading is misleading. Central venous pressure is a poor predictor of fluid responsiveness. Radio-labelled markers (red cells and albumin) and dyes (indocyanine green) indicate the fluid

volume in circulation but they give no idea as to adequacy of fluid volume in relation to intravascular space.

Fluid responsiveness is defined as a positive increase in stroke volume in response to a fluid bolus. A human meta-analysis showed that ventricular pre-load indicators were poor indicators while dynamic parameters were better indicators of fluid responsiveness. Not all patients will respond to a bolus of fluids and increase in blood pressure and cardiac output will not be seen. Fluid responsive patients are on the preload dependant portion of the Frank-Starling curve while non-responsive patients are on the preload load independent portion of the curve. Non-responsive patients potentially benefit more from ionotropic circulatory support.

Ideally the rate of fluid administration should be calculated taking the following into account.

Maintenance Requirements: 80 – 100 ml/kg/day

The maintenance amount includes normal urine production of 1 - 2 ml/kg/hr. If the patient is oliguric this needs to be adjusted accordingly. In anuric patients the normal urine production must be removed. In essence maintenance fluid requirements without including urine production is approximately 20 ml/kg/day.

Fluid Deficits: % dehydration x BM

Dehydrated patients require additional fluid to correct this imbalance. A 10% dehydrated patient would require approximately 10% of its mass to correct the deficit. This may be given over a short period of time (± 2hours) or over 6 - 24 hours. Prior to anaesthesia it is advisable to correct as much of this imbalance as possible.

Ongoing Losses:

Ongoing losses to the gastro-intestinal tract through diarrhoea or vomiting may amount to several litres of fluid in a large dog per day. The volume of stool or vomitus produced can be used as guide to increase the fluids given.

Third Space Losses:

Third space losses are difficult to quantify as no fluid leaves the body. But, the fluid in the intravascular space is redistributed to other organs in the body. It generally results in oedema of these organ systems. Some people have considered diarrhoea as a third space loss.

Haemorrhage:

Intra operative blood loss can be measured through assessing blood on the swabs and the amount of blood accumulated in the suction system. This loss needs to be added on, on an ad hoc basis, as it cannot be done on a pre-emptive basis. Some patients may lose several litres during a procedure while others will only lose a negligible amount. It is recommended that if a patient losses more than 20% of circulating volume they should be transfused.

Loss to the Environment:

Evaporation from exposed surface areas can be high in hot dry theatres. Most theatres are air-conditioned resulting in low relative humidity and theatre lights are hot increasing evaporation. This loss is again difficult to calculate but an additional 2 – 6 ml/kg/hr can be allowed for this loss.

Increased Intravascular Space:

An increase in intravascular space may occur due to vasodilatation as a result of anaesthetic agents used. This “loss” needs to be replaced urgently. Ideally these fluids should be titrated to clinical parameters. If fluid volumes become large then vasoconstrictors should be used.

The ultimate quantity of fluid given is determined by adding the above losses together. This may result in a continually changing fluid rate. Fluid rates may initially be high to correct for a deficit but haemorrhage can occur at any stage and result in a sudden increase in fluid administration.

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HEATSTROKE

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ABSTRACT

Heatstroke occurs when body heat generated by endogenous or exogenous mechanisms exceeds the body's cooling capacity. Heatstroke leads to multi-organ involvement with cardiovascular, gastrointestinal, renal, coagulation and neurologic systems predominating. Treatment includes rapid cooling of the body temperature in conjunction with fluid resuscitation and supportive and symptomatic care. Prognosis is improved for patients brought to normal body temperature as soon as possible. Poorer prognosis has been associated with a delayed treatment, advanced neurologic signs or neurologic deterioration, hypothermia on presentation, persistent hypotension, persistent oliguria or azotemia despite fluid loading, elevated bilirubin, persistent hypoglycaemia, hypoproteinemia, increased numbers of nucleated red blood cells, and disseminated intravascular coagulation.

WHAT IS HEATSTROKE?

Heat-induced illness occurs when heat production exceeds the rate by which heat can be lost from the body. Elevated body temperature further contributes to heat production by stimulating an increase in metabolic rate and endogenous heat production. Eventually, the elevated core body temperature inhibits hypothalamic function and cooling mechanisms are diminished, contributing to an even more elevated body temperature; and cellular and organ dysfunction ensues.

MECHANISMS OF HEAT LOSS

When the core body temperature exceeds the hypothalamic set point, the body attempts to cool itself by several different methods. Radiation is transfer of heat from the body to the environment. Convection is transfer of heat to air as it moves over the skin, such as by a fan. Conduction is heat loss to a cool surface, such as a metal table. Heat can also be lost via evaporation of moisture, primarily from the respiratory tract. Radiation and convection account for the majority of heat loss in small animals. Peripheral vasodilation aids in heat loss by bringing more blood to the surface to be cooled. When environmental temperatures are high, particularly as they approach body temperature, radiation, convection and conduction cooling methods become ineffective and evaporative cooling is the most important heat loss method. High humidity hampers cooling by evaporation.

TYPES OF HEATSTROKE

Heatstroke can be either exertional or non-exertional. Exertional heatstroke occurs when the animal works or plays in a hot environment. This type of heatstroke tends to happen when animals have not acclimatized to higher ambient temperatures, such as may occur in the early spring and summer or in animals abruptly moving to a warmer climate. Animals that have acclimatized properly, such as racing dogs and military working dogs, can exercise in extremely hot conditions without ill effect if kept well hydrated and not overexerted. Non-exertional heatstroke occurs when animals are in a warm or hot environment without access to shade or water. Exertional and non-exertional heatstroke can and often do concurrently occur.

PREDISPOSING FACTORS

Both environmental and endogenous factors can predispose an animal to heat stroke. Environmental predispositions include: lack of acclimatization, high humidity, lack of shade, water or adequate ventilation (such as in a closed car), and exposure to exogenous heat sources, such as dryers. Endogenous predisposing factors contribute to heatstroke by inhibiting normal heat dissipation mechanisms. Such factors include brachycephalic conformation, laryngeal paralysis, tracheal collapse, obesity, significant cardiac disease, thick hair coat, large body size, and neurologic diseases that impair breathing/ventilation or alter the thermoregulatory centre. History of a previous heatstroke episode also puts animals at greater risk due to damage to the thermoregulatory centre.

EFFECTS ON ORGAN SYSTEMS

Heatstroke has deleterious effects on all organ systems. Thermal injury can cause denaturation of proteins, inactivation of enzyme systems and mitochondrial function and destruction of cell membranes. Additionally, hypovolemic and metabolic shock can contribute to decreased oxygen delivery to cells and subsequent cell dysfunction and cell death. Systemic inflammatory response and multiple organ failure can both develop subsequent to heatstroke.

Cardiovascular system

The initial cardiovascular response to elevated body temperature is tachycardia, cutaneous vasodilation and renal and splanchnic vasoconstriction all leading to increased cardiac output and improved heat loss through the skin. With extreme hyperthermia, vasoconstriction is inhibited by signals from the hypothalamus, thus preventing the vasoconstriction normally associated with shock, which serves to shunt blood to vital organs and maintain normal blood pressure. With time and rising body temperature, splanchnic vasodilation occurs and blood pools in the vasculature. Dehydration contributes to low plasma volume and hypovolemic shock, which further impairs heat dissipation by reducing delivery of blood to the periphery. Metabolic shock ensues as the body's metabolic rate exceeds the delivery of substrates to the cells. The cardiovascular system is further compromised by ischemia and direct thermal injury to cardiac myocytes, which can lead to ventricular arrhythmias.

Respiratory system

Pulmonary function may be compromised due to poor perfusion to the lungs, non-cardiogenic pulmonary oedema, acute respiratory distress syndrome (ARDS), haemorrhage from disseminated intravascular coagulation, and increased pulmonary vascular resistance. Lung injury reduces oxygenation of any blood that does make it through the lungs, further compromising oxygen delivery to tissues.

Central Nervous system

Neuronal tissue is very sensitive to increases in body temperature. Direct thermal injury leads to neuronal cell injury and death. Cerebral oedema, haemorrhage, infarction, poor cellular function may result in clinical signs of disorientation, stupor, coma, seizure activity, or death. Furthermore, damage to the thermoregulatory centre in the hypothalamus can impair heat loss during the current heatstroke episode as well as putting the animal at risk for future episodes.

Renal

Acute renal failure develops secondary to direct thermal damage to the tubules, decreased oxygen delivery to the kidneys, DIC-induced microthrombi, and pigmentary nephropathy from haemoglobin or myoglobin-induced renal damage. Pre-renal azotaemia from severe dehydration also contributes to renal dysfunction. Renal damage may be all or partially reversible. Some animals are left with permanent renal dysfunction after recovery from heatstroke.

Gastrointestinal and Hepatic systems

The gastrointestinal tract and especially the tips of the intestinal villi are sensitive to reductions in blood flow or oxygen delivery. Hypovolaemia, microthrombi, and pooling of blood in the splanchnic vessels reduce blood flow causing ischemia, rapid loss of GI integrity, and subsequent ulceration and GI haemorrhage. Translocation of bacteria and endotoxin ensues, which can lead to sepsis and septic shock. Hyperthermia and splanchnic hypoperfusion induces hepatocellular necrosis and cholestasis and hepatic dysfunction contributes to hypoglycaemia. Decreased hepatic macrophage function may contribute to development of sepsis since bacteria translocating across the GI tract aren't properly cleared in the liver. Hepatic dysfunction also slows production of coagulation factors and contributes to coagulation problems.

Hematologic and Coagulation systems

Patients may present with an elevated red cell count due to dehydration and subsequent hemoconcentration. Anaemia may result from haemorrhage into the GI tract, DIC, haemolysis, and RBC extravasation through leaky vessels. Endothelial damage exposes collagen and releases tissue factor, initiating the coagulation and cascade and widespread coagulation, which can result in DIC. Hyperthermia also activates platelets and damages megakaryocytes, which can result in thrombocytopenia that progressively worsens over several days. The complement cascade is also initiated, contributing to widespread inflammation.

Musculoskeletal system
Rhabdomyolysis can result, most often from exertional forms of heatstroke. Myoglobinuria can contribute to the development of acute renal failure.

CLINICAL FINDINGS

History may include a precipitating event, such as being closed in a car, tethered in the sun without water, or collapsing after exercise. Some may have a history of respiratory problems, heart disease, etc., or previous heatstroke, which may have predisposed to the current event.

A full physical examination should be performed on every patient with suspected heatstroke with special attention paid to the cardiovascular and neurologic systems. Findings depend on the duration of the event prior to presentation. While a diagnosis of heatstroke is obvious when an animal presents with an extremely high body temperature, some animals present with a normal or low body temperature if cooling measures were initiated or if the animal is in late decompensatory shock. Common findings include panting, tachycardia, dark or hyperaemic mucous membranes with brisk or prolonged capillary refill time, bounding to thready pulses and petechiae or ecchymoses. There may be pulse deficits if arrhythmias are present. Mentation and other neurologic signs vary depending on severity. Animals may present with depression, stupor, coma, ataxia, seizures, cortical blindness, or other neurologic signs. Other physical exam findings may reflect a predisposition for heatstroke, such as stridor or stertor which indicate respiratory tract abnormalities.

Common clinicopathologic findings include hemoconcentration, anaemia, hypoproteinemia, hypoglycaemia, elevated liver enzyme activities and bilirubin, azotaemia, hyperphosphatemia, electrolyte abnormalities, metabolic acidosis from hyperlactatemia, respiratory alkalosis, prolonged coagulation times, thrombocytopenia, increased urine specific gravity, proteinuria, casts, and myoglobinuria.

TREATMENT

Early external cooling is the single treatment that markedly reduces heatstroke mortality and so should be the primary goal of initial therapy. If possible, owners should completely cool the animal or at least thoroughly soak the animal with cool water prior to transporting to the veterinary clinic. Driving with the air conditioning on with a vent blowing on the animal or with windows open will facilitate evaporative and convective cooling. Once at the veterinary clinic, cooling should continue.

Much research has been done in humans with both exertional and non-exertional heatstroke. The most effective method for rapid cooling is ice water immersion. Historically there has been some concern that use of ice water may cause peripheral vasoconstriction, lock heat within the core, and produce a shivering response that could increase metabolic rate and generate more endogenous heat. However, studies have shown that ice water immersion decreases body temperature most rapidly.[4] Cold or ice water shower/soaking coupled with evaporative loss via fans is also an effective combination. Putting ice packs in the inguinal and axillary regions (over major vessels) or utilizing fans as a single method to reduce body temperature are not efficient ways to reduce body temperature in patients with heatstroke. When using water for cooling, it is imperative that the animal is soaked to the skin, otherwise, the wet outer layer of the fur/hair will trap a layer of hot air close to the skin and reduce heat loss. A similar effect also occurs when wrapping the animal in wet towels. Ice water enema, gastric lavage, peritoneal lavage and intravesicular lavage have been used as cooling methods, but these are somewhat cumbersome, inhibit the ability to monitor the body temperature (particularly with enemas) and are no more effective then external cooling. Cooling measures should be withdrawn when the body temperature is approximately 103°F, otherwise, the animal may become hypothermic if cooling continues. The author typically removes from cold/ice water bath at 105°F and then dries the patient at 103°F.

Animals with an airway obstruction should be intubated immediately; this will also facilitate heat loss since the respiratory system is a major contributor to thermoregulation in animals. Oxygen demand is increased in heatstroke (increased body temperature increases metabolic rate) so oxygen therapy may be beneficial for all heatstroke patients. An intravenous catheter should be placed and IV fluid therapy should be initiated as soon as possible after the onset of external cooling. IV Fluids will assist

with cooling as well as correction of vascular volume and acid-base abnormalities. Replacement crystalloids are the fluids of choice to replace vascular volume as well as dehydration deficits. A bolus of 20 (cat) to 30 (dog) mL/kg (1/4-1/3 of a total shock dose of 60 mL/kg in cats, 90 mL/kg dogs) is a reasonable starting point. After fluid resuscitation is completed, dehydration, ongoing losses, and maintenance fluid needs must be calculated and administered. Dextrose can be added to fluids to treat hypoglycaemia. Colloids may benefit patients with hypoproteinemia. Plasma is indicated if coagulation times are prolonged. Packed red cells or whole blood should be used if the patient has significant anaemia. Antibiotics should be considered, especially for animals that are sloughing the gastrointestinal tract. An H_2 blocker or proton pump inhibitor and sucralfate will help heal gastric ulceration. Mannitol or hypertonic saline should be used if neurologic signs are significant and do not improve or worsen after vascular volume resuscitation. Anti-seizure medications should be used to treat seizures. Corticosteroids have no clear benefit; in an experimental canine heatstroke model, dogs actually had high circulating endogenous plasma cortisol levels that rose for several hours after heatstroke. NSAIDS are contraindicated; they may cause or exacerbate gastric ulceration, renal failure, and platelet dysfunction.

Attentive monitoring is an essential part of caring for a heatstroke patient. Basic vital parameters should be serially monitored. Urine output should be quantified either by catching and measuring or weighing urine or placing a urinary catheter, especially in patients with acute renal failure. Blood pressure should also be followed closely and hypotension should be avoided. If hypotension is documented, additional fluid boluses, colloid therapy, or pressors and inotropes may be indicated.

PROGNOSIS

Mortality rate decreases from 49% to 19% for animals that are cooled prior to traveling to the veterinarian. A worse prognosis has been shown for animals with advanced neurologic signs or neurologic deterioration, hypothermia on presentation, persistent hypoglycaemia or hypoproteinemia, persistent hypotension, persistent oliguria or azotaemia despite fluid loading, elevated bilirubin, pulmonary oedema, or DIC. Most animals that die from heatstroke do so within the first 24 hours. Animals alive after 72 hours generally will survive.

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MONITORING ICU PATIENTS

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ABSTRACT

Monitoring comes from a Latin term “monere” meaning “to warn”. The aim of monitoring patients in the intensive care unit is to identify problems and correct them before they lead to irreversible changes that lead to the patient’s death. It is all about assessing oxygen delivery and ensuring that oxygen is available to every cell in the body. Oxygen is delivered by the cardiovascular system and taken by the lungs. This makes monitoring the cardiovascular and respiratory systems vital. Intensive care is usually an expensive undertaking and knowing the prognosis can assist us in our discussion with owners. It will also give us realistic expectations. This talk will discuss the principles behind monitoring patients requiring intensive care.

MONITORING PATIENTS

Monitoring comes from a Latin term “monere” meaning “to warn”. The aim of monitoring is to supply the veterinarian or veterinary nurse with enough information as to give a warning before a change in physiological values leads to an adverse outcome. An incidence is any change in physiological values that occurs that could be potentially harmful or have a negative outcome to the patient. An incident may happen acutely or result from a slower decompensation of a vital organ system over time. An accident results when an incident has occurred and it has not been rectified in time. As a result the patient suffers.

The term vital sign refers to those parameters that indicate the response of a patient’s homeostatic mechanisms. This includes the basics of heart rate, respiratory rate, blood pressure and capillary refill time for example but includes data obtained through clinical pathology and monitoring devices. The patient’s vital signs should indicate how well the patient is maintaining basic circulatory and respiratory function during anaesthesia.

The body has compensatory mechanism to ensure survival during times of stress. These compensatory mechanisms mean that normal parameters can be recorded due to compensation. Blood pressure for example is maintained by vasoconstriction and an increase in heart rate. Blood pressure will be kept in a normal range until maximal vasoconstriction and the maximum increase in heart rate is achieved. This means that once blood pressure falls all the compensatory mechanism have been exhausted. A drop in blood pressure is a late sign. Blood pressure should always be assessed taking into account heart rate and sign of peripheral vasoconstriction (pale mucous membranes, changes in capillary refill time).

The delivery of oxygen to each cell requires that sufficient haemoglobin is available, blood is fully saturated in the lungs with oxygen and that sufficient blood passes each cell. Essentially two major systems (cardiovascular and respiratory) are involved and these are generally monitored separately, but one cannot ignore the fact that they are integrated. Oxygen delivery is essentially a function of three major components: haemoglobin, saturation and cardiac output.

Shock is simply when the demand for energy exceeds the supply of energy. Haemorrhagic shock is determined by how much blood was lost, how quickly the blood was lost, the concurrent injuries, age and physical condition of the animal. If more than 35% of circulating volume is lost, hypotension and irreversible tissue ischaemia can result. Neurogenic shock is the result of loss of sympathetic tone and is usually due to central nervous system or spinal cord damage. Compressive shock is usually the failure of blood to return to the heart because of pericardial tamponade, pneumothorax, haemothorax, etc. Cardiogenic shock results due to myocardial damage. Cardiogenic shock can develop as result of hypoxia, inflammatory response and other myocardial depressants.

Shock is also classified on the basis of compensated (reversible) or decompensated (irreversible) shock. Reversible shock may rapidly become irreversible if appropriate therapy is not rapidly applied – the golden hour. Rapidly progressing decompensation is characterised by sudden cardiovascular collapse, hypotension and pooling of blood in capillary and venous compartments. Subsequent loss of oxygen, glucose and other vital nutrients leads to abnormal tissue metabolism and organ dysfunction. This stage of shock is characterised by rapid deterioration in vital organ function - brain and heart. This results in coma, stupor and cardiac arrhythmias. Laboratory investigations show severe metabolic acidosis and electrolyte abnormalities.

Vasoconstriction impairs microcirculation and results in tissue hypoxia. Nitric oxide, a potent vasodilator, release is inhibited. During the compensatory phase of shock, ATP is depleted from cells. The decrease in energy within the cell slows the sodium-potassium pump. Sodium accumulated within the cell and cellular swelling occurs eventually leading to cell death. Insufficient oxygen in metabolically active cells leads to anaerobic metabolism and the development of lactate and acidosis. Acidosis results in cellular dysfunction.

Hypothermia is common due to environmental conditions, inadequate circulation, decreased metabolic rate and infusion of cold fluids. Hypothermia causes bradycardia, ventricular arrhythmias, decreases enzyme reactions, increases membrane permeability, decreases ion pump activity and effects coagulation.

Second insult to the body readily occurs after the primary trauma has primed the body. The neurohumoral response initiated by the trauma predisposes the body to sepsis because of reperfusion injuries, immunocompromisation, decreased splanchnic perfusion and loss of integrity of the gastro-intestinal tract. Increased GIT permeability is most probably the leading cause of mortality and multiple organ failure. This is due to the translocation of bacteria and endotoxins to systemic circulation via blood and lymphatics.

To identify patients in trouble, attention during the basic clinical examination should be placed on the following:

- Mentation (Cardiorespiratory compromised patients are usually mentally dull)
- Circulation (Heart rate – usually a tachycardia, Rhythm, Pulse Pressure, Capillary refill time - usually prolonged but in hyperdynamic shock is very short)
- Respiration (Rate – fast shallow breathing is usually present in shock, Rhythm – an irregular pattern is usually present is severely compromised patients and Signs of Cyanosis. Rapid deep breathing cannot be maintained and will lead to respiratory failure. Patients with dyspnoea can be considered to be in shock)
- Temperature (Measure core temperature, palpate periphery – ears, paws)
- Blood Smear (Look at all cell types – inflammatory response, platelets)
- Micro-organism (Babesia, Bacteria, Protozoa, Fungal)

Sepsis (a condition requiring early aggressive therapy and intensive care) can simply be defined as the host's response to infection involving the release of numerous cytokines and mediator. The table below has been suggested as a possible way to identify patients with SIRS and Sepsis. At least two of the following needs to present for SIRS and micro-organism for Sepsis.

Parameters	Abnormal	Normal	Abnormal
Temperature	< 37.8	37.8 – 39.2	>39.2
Heart Rate	< 80	80 – 120	> 120
Respiratory Rate	< 12	12 – 20	> 40
WBC/mm^3	< 5	5 – 18	> 18
Micro-organism			

Table 1: Identification of SIRS and Sepsis based on the 1992 consensus conference.

These criteria for SIRS or sepsis are not perfect and a number of cases may be missed. This definition is based on minor changes in 2 of the 4 parameters. When the above definition is applied, most sick patients and many healthy people during the course of the day fulfil the criteria for SIRS. A good example of this is babesiosis. Most patients with babesia do very well on relatively conservative therapy. This makes the above criteria unhelpful in identifying patients in trouble.

An expanded list of criteria for the identification of sepsis has been proposed due to the inadequacies of the original definition. It is important to realise that a single parameter is not pathognomonic for SIRS or sepsis. The presence of these signs indicates the possibility that it is present and the final diagnosis becomes a clinical judgement.

Infection, documented or suspected, and some of the following:
General variables
Fever (core temperature > 39.2°C) (human - > 38.3°C)
Hypothermia (core temperature < 37.8°C) (human < 36°C)
Heart rate > 120 or > 2 standard deviation above normal (human > 90)
Tachypnea
Altered mental status
Significant oedema or positive fluid balance (> 20 ml/kg over 24 hours)
Hyperglycemia (plasma glucose > 120 mg/dl or 7.7 mmol/l) in the absence of diabetes
Inflammatory variables
Leukocytosis (WBC > 18 000 µl) (human > 12 000 µl)
Leukopenia (WBC < 5 000 µl) (human < 4 000 µl)
Normal WBC count with > 10 % immature forms
Plasma C-reactive protein > 2 standard deviations above normal
Plasma procalcitonin > 2 standard deviations above normal
Haemodynamic variables
Arterial hypotension (SBP < 90 mmHg, MAP < 70 mmHg, or a SBP decrease > 40 mmHg or < 2 standard deviations below normal
SvO_2 > 70%
Cardiac index > 3.5 l/min/m^2
Organ Dysfunction Variables
Arterial hypoxaemia (PaO_2/FiO_2 < 300)
Acute oliguria (urine output < 0.5 ml/kg/hr)
Creatinine increase > 0.5 mg/dl
Coagulation abnormalities (INR > 1.5 or aPTT > 60 sec)
Ileus (absent bowel sounds)
Thrombocytopaenia (platelets < 100 000 µl)
Hyperbilirubinaemia (plasma total bilirubin > 4 mg/dl or 70 mmol/l)
Tissue perfusion variables
Hyperlactaemia (> 2.1 mmol/l) (human > 1 mmol/l)
Decreased capillary refill time or mottling

Table 2: Diagnostic criteria for Sepsis. Human values are given in brackets at the end. The veterinary values have been taken from published literature used in the definitions of SIRS and sepsis.

Sepsis is a clinical syndrome defined by deleterious host response to an infectious process. Severe sepsis is defined as filling the criteria for sepsis plus the addition of one or more organ failures or organ dysfunctions. Septic shock is defined as sepsis with presence of hypotension (systolic blood pressure below 90 mmHg) despite adequate fluid resuscitation and the presence of hypoperfusion (raised blood lactate).

An approach to the characterisation and staging of sepsis has been proposed – Predisposition, Insult, Response and Organ dysfunction (PIRO). This approach is based the stratification of cancer developed in 1946 by Pierre Denoix – Tumour Node Metastasis (TNM).

- Predisposition – Genetic predisposition, chronic Illness
- Insult – Infection, endotoxin, microbes, injury, ischaemia
- Response – Physiological shock, mediators and markers of severity of sepsis
- Organ Dysfunction

The occurrence of organ failures is predictable based on serial measurements of physiological, biochemical, haematological parameters and changes in systemic inflammatory response mediators (interleukins, granulocyte colony-stimulating factor). It has been stated that multiple organ

failure/dysfunction can be recognised by having one abnormal serum chemistry result per organ system. This statement may not always be true. A good example would be a raised liver enzyme. Under these circumstances, liver damage is present but the liver may well be able to continue its functions (produce albumin, clotting factors and remove harmful and waste products (ammonia, bilirubin, etc). Chronic hepatic failure is associated with normal to low enzyme levels. In such organs, an assessment of function goes beyond just an elevation in serum enzymes.

Acute renal failure has an incidence of 5 – 15% with an associated mortality as an isolated organ failure of 10 – 15%. When renal failure is associated with multiple organ failure in man the mortality is between 50 – 90%. A mortality of 62% has been reported for hospital acquired renal failure in dogs. Initial urine output (oliguria) was associated with an increase in mortality. Renal failure has been estimated to occur 2.2% of babesia cases. Pancreatitis is estimated to occur at rate of at least 0.04% in babesiosis[1]. DIC has been shown to occur at rate of 27% in dogs admitted to an ICU with a disease known to cause DIC. An incidence of DIC of 12.2% has been reported in dogs with cancer. The highest incidence occurred in patients with hemangiosarcomas, mammary carcinomas, and pulmonary adenocarcinomas. Acute liver failure can have a mortality rate as high as 90%. Common complications following on liver failure include hepatic encephalopathy, hepatorenal syndrome, cardiomyopathy and coagulation disorders.

The Acute Patient Physiologic and Laboratory Evaluation Score are available for dogs and cats to predict mortality. The trauma score was developed to predict mortality in trauma patients. Knowledge of the expected outcome can be used to give a realistic prognosis. Scoring systems using basic physiological data have been developed to identify patients in trouble. These early warning systems may improve outcomes.

Capillary refill time gives us an indication of peripheral blood pressure and circulation. A normal capillary refill time is 1 to 2 seconds. It is important to realise that a dead patient also has a normal capillary refill time. Instead of arterial blood filling the capillary bed venous blood fills the bed. A prolonged capillary refill time is usually an indication of hypotension or shock. Mucous membrane colour is also observed when testing the capillary refill time. Normally the mucous membranes are pink. Pink mucous membranes do not indicate that all is well. After euthanasia an animal's mucous membranes will remain pink for several minutes. Blue mucous membranes (cyanosis) and very bright pink mucous membranes with very fast capillary refill times (hyperdynamic shock) indicate impending doom if not corrected. Certain breeds of dogs (Chows, Dalmatians, German shepherds) may have normally pigmented mucous membranes. The tongue, buccal, conjunctiva, prepuce and vulva may be used to assess mucous membrane colour.

Blood pressure is an easily measured variable of perfusion along with heart rate. Blood pressure changes dynamically all the time and can change with each reading. Not only is a mean blood pressure below <60 – 70 mmHg is associated with a poor outcome but also the amount of time the blood pressure is below 60 mmHg. Blood pressure should be monitored continuously and low blood pressure should be treated. In order to identify shock, blood pressure should be interpreted with heart rate. The shock index is defined as the heart rate divided by the systolic blood pressure. A normal ratio is < 0.8. Values greater than 1 are associated with shock and should prompt immediate treatment.

Urine output is a useful tool for monitoring blood pressure and perfusion. Normal urine production is 1 – 2 mls/kg/min but in ICU patients a urine production of 0.5 ml/kg may be all that is achievable. If renal perfusion (blood pressure, blood flow) is not adequate, urine production ceases. Urine analysis should also be undertaken.

Visual inspection of the mucous membranes for cyanosis as a method for determining hypoxia is a very unreliable indicator. Desaturation is often accompanied by an increase in heart rate and electrocardiographic evidence of hypoxia. Pulse oximetry improves the detection of hypoxaemia 20 fold and that of hypoventilation 3 fold. Pulse oximetry can be monitored continuously and used to determine the requirement and effect of oxygen supplementation. Although oxygen is essential for survival is highly toxic. Oxygen should be supplemented to achieve a saturation of between 88 – 96%. Hyperoxia is associated with poorer outcomes and at saturation of 100% hyperoxia can be present.

Capnography is a useful tool to monitor respiratory and cardiovascular function. The device measures inspired and expired carbon dioxide. In order for the carbon dioxide to appear in the expired gas

adequate alveolar ventilation and pulmonary circulation are required. With a drop in alveolar ventilation expired carbon dioxide rises and with a drop in perfusion, expired carbon dioxide drops. The end tidal concentration of carbon dioxide is a measure of ventilatory function (normal ventilation is controlled by blood levels of carbon dioxide). A rise in end tidal carbon dioxide, providing perfusion remains constant, indicates hypoventilation while a drop indicates hyperventilation. The waveform generated by a capnograph may be indicative of a number of respiratory and cardiovascular disorders.

Blood gases are used to assess arterial and mixed venous oxygen content, pH and base deficits. A rise in partial pressure of carbon dioxide and a drop in partial pressure of oxygen indicate ventilatory failure. Usually a respiratory acidosis prevails. Circulatory problems are usually more evident in a mixed venous sample. pH, base deficits and lactate are parameters that correlate to perfusion. Hypoxia is defined as a partial pressure of oxygen less than 60 mmHg. All hypoxic patients should receive oxygen and should definitely be considered for ventilation. Hypoxia is again readily diagnosed if a blood gas analysis is done. Pulse oximetry can be used to diagnose hypoxia if saturation is less than 90%. Ideally all patients with a saturation of less than 94% should receive supplemental oxygenation. The most common cause of hypoxia is the result of pulmonary injury through acute respiratory distress syndrome, intra-pulmonary shunting and extra-vascular lung water.

Normal healthy dogs have blood lactate of 1.80 +/- 0.84 mmol/l (1.48 – 2.11 mmol/l). Puppies have higher blood lactate that decreases during the first 28 days of life. Reference ranges for puppies are 4 days 1.07 – 6.59 mmol/l and 10 – 28 days 0.80 – 4.60. Lactate values between 3 – 5 mmol is considered a mild elevation, 5 – 7 mmol/l moderate and > 7 mmol/l is severe. Single values of lactate have been correlated to outcome but more importantly is the rate at which lactate is cleared. Lactate is also not a perfect indicator of hypoperfusion but elevation should always raise concern.

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RECOGNIZING RESPIRATORY PATTERNS IN THE DYSPNOEIC PATIENT: KEYS TO NARROWING THE DIFFERENTIAL LIST

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ABSTRACT

Respiratory distress is a common presenting problem for emergency patients and can be an acquired problem in hospitalized patients. These patients present a diagnostic and therapeutic challenge as they are fragile and may not tolerate extensive or invasive diagnostics. As such, familiarity with respiratory patterns and physical examination findings will help facilitate diagnosis. Prompt identification of the problem and appropriate therapy are essential to prevent decompensation and death. Respiratory patterns, coupled with specific historical and physical findings, will help narrow the differential list and lead to a more focused diagnostic and treatment plan. Stertor or stridor and an obstructive respiratory pattern, marked by prolonged inspiratory or expiratory phases with full lung expansion, are indicative of anatomic or functional airway obstruction. Treatment involves oxygen, sedation, cooling as needed and intubation if severe. A restrictive pattern, or shallow, rapid thoracic excursions, is indicative of pleural space or pulmonary parenchymal disease and these are further differentiated via auscultation. Oxygen and thoracocentesis will help most patients with pleural space disease. Treatment for pulmonary parenchymal disease involves oxygen, sedation as needed, and disease specific therapies.

PATTERNS OF RESPIRATION

Normal respiration is barely noticeable at rest. Chest movement is minimal and effortless; the ribs move cranially and slightly outward with each breath. The abdomen moves passively slightly outward as the diaphragm moves caudally. The thorax and abdomen move together with each breath.

Signs of Respiratory Distress

Dyspnoeic patients will have an increased respiratory rate and effort, may breathe with an open mouth, flare nostrils, seem anxious or restless, be orthopnoeic, may be cyanotic, and may have an abnormal respiratory pattern. Orthopnoea is manifest as elbows out and head and neck extended upwards in a dog, and generally as sternal recumbency with head and neck extended up in a cat.

Cyanosis is an insensitive indicator of hypoxia, however it indicates SEVERE oxygen deficiency if present. Cyanosis corresponds to a PaO2 of approximately 40 mm Hg. Cyanosis will only be apparent to our eyes once 5 g/dl of deoxygenated haemoglobin is present in the blood – moderate to severely anaemic animals may never be cyanotic because they do not have enough haemoglobin!

Noise may also be heard in dyspnoeic patients that have airway obstructions. Stridor, a high-pitched sound similar to wind whistling through a crack in a window, is heard when air is moving through a very small opening. Stertor, a snoring noise, is associated with vibration of soft tissues as air moves over them; much like a flag makes noise as it is whipped about by a wind.

Additionally, with onset of dyspnoea and increased work of breathing, animals may develop paradoxical respiration which can include any of the following: 1) chest and abdomen move in opposite directions 2) inward collapse of ribs/intercostal spaces during inspiration due to increased negative pressure or weakness of the intercostal muscles (fatigue) 3) inward collapse of the abdomen during inspiration. Abdominal effort can be quite pronounced.

The patient should be continuously monitored for ***signs of fatigue and impending arrest*** as intervention prior to respiratory arrest typically results in a better outcome. Decreased mentation, vocalization (dog and cat), restlessness, frantic movements, changing body position, lateral recumbency (especially the cat), and mydriasis (cat) are signs of hypoxaemia and impending arrest. Some (mostly dogs) will also develop nystagmus when severely hypoxic.

Recognizing abnormal respiratory patterns
Dyspnoea can originate from dysfunction of one of five major areas in the body: upper airway, lower airway, pulmonary parenchyma, pleural space, and thoracic wall/diaphragm. In combination with history and physical examination, observations of the patient's respiratory pattern may help localize the source of dyspnoea and focus the differential list.

Abnormal respiratory patterns fit into three major categories: restrictive, obstructive, and hypoventilation.

Obstructive respiratory pattern
Characterized by long respiratory cycles. This occurs primarily due to narrowing of airways. In dynamic extrathoracic upper airway problems, there is increased inspiratory effort. In dynamic intrathoracic airway problems, the effort is increased on expiration. This difference occurs due to cycling of airway diameters during the normal respiratory cycle. On inspiration, the intrathoracic trachea is normally pulled open and on exhalation, the diameter is passively decreased. The opposite occurs for the extrathoracic airway. If the obstruction is fixed, there may be increased effort on both inspiration and expiration.

Restrictive respiratory pattern
Characterized by short, shallow and rapid breaths because the lungs are either stiff or there is restriction to lung expansion - something compressing on and preventing the lungs from expanding fully. This type of pattern is seen with pulmonary parenchymal disease and pleural space disease.

Hypoventilation pattern
Characterized by reduced chest wall excursions and sometimes "fish mouth" breathing. This abnormal pattern is sometimes easily overlooked because the animals typically don't "look" *dyspnoeic* since they are unable to breathe harder, like we usually expect with respiratory problems. Hypoventilation is seen with thoracic wall trauma (flail chest, open thoracic wound) and weakness such as might occur with neuromuscular diseases.

FINDINGS RELATED TO LOCATION OF DYSPNOEA

Airway
Airway problems are generally obstructive in nature. Patients demonstrate an *obstructive respiratory pattern*. They may have stridor or stertor and also often cough. A honking noise is a classic finding with collapsing trachea. Choking, gagging, ptyalism, and foaming from the mouth all suggest an upper airway issue. Animals may also paw at face/ neck, especially if they have a foreign body stuck proximally. They may also appear to "vomit" or gag – this is often marked coughing with expectoration of phlegm/discharge. The discharge may be swallowed rather than expelled to the outside. Finally, animals with severe airway obstruction may present for collapse. Auscultation of the lungs may reveal referred upper airway noises – to differentiate true lung sounds from airway noise, auscultate the lungs as well as along the length of the trachea. Referred upper airway noises are louder over the trachea/larynx than can be heard over the lungs. Sometimes, the obstruction can be so severe that the patient makes no noise and passes no air despite marked effort.

Upper airway obstructions include diseases of the nasal cavity, pharynx/larynx, and trachea. Specific problems associated with the nasal cavity include stenotic nares, choanal atresia, and obstruction from discharge, masses, or foreign material. Most animals with disease confined strictly to the nose may have open mouth breathing but are not truly dyspnoeic or hypoxic. In the pharynx/ larynx, laryngeal polyps, elongated soft palate, oedema, laryngeal collapse, inflammation, foreign body, trauma, paralysis, neoplasia, or everted saccules can occur. Tracheal diseases include collapse, stenosis, extraluminal compression, trauma, foreign body inhalation, neoplasia and parasites (*Oslerus osleri*, cuterebra).

The main aspects of managing obstruction are Oxygen/Airway, Sedation, and Temperature control. Treatment for dyspnoea of any type starts with oxygen but for obstructed patients, ensuring a patent airway is also paramount. If the animal is mildly affected, conservative management with sedation or an anxiolytic is usually effective. Sedation helps because the excess respiratory effort brought on by anxiety exacerbates abnormal airflow and makes the obstruction worse. By sedating the animal, it will be less stressed and breath with less force, thus allowing air to flow more easily through the narrowed passages. You must watch the animal after administering sedatives to be sure it does not relax too much and become unable to ventilate. Be prepared to intubate if necessary…

For a severely dyspnoeic animal that is not able to move air, is panicking, showing signs of impending arrest, or is cyanotic, (or if it occludes completely after sedation) the airway must be secured immediately!! Animals that are severely hypoxic and near exhaustion sometimes can be intubated with little sedation. If sedation is necessary, most dyspneic animals require lower doses of drugs for intubation than healthy counterparts. Choose an endotracheal tube that is a few sizes smaller than you might normally use for the same animal – this will increase your chance of successful intubation on the first try. If there is significant obstruction, an even smaller tube may be more appropriate. If an endotracheal tube cannot be successfully passed, an emergent tracheostomy or a tracheal insufflation catheter may be needed for stabilization.

Animals lose a lot of heat via the respiratory tract, especially when panting. If the airway is obstructed, the animal loses the ability to effectively thermoregulate and they can get extremely hyperthermic. Always check the body temperature of an animal with airway obstruction! Cooling measures including cold-water bath, circulating cold water blankets, fans, or low environmental temperature in an oxygen cage. Securing an airway will also rapidly facilitate temperature regulation. Finally, an **anti-inflammatory** dose of corticosteroids may also help by reducing inflammation in the airway that can contribute to airway occlusion.

Lower airway diseases involve areas distal to the first few branches of bronchi. Feline asthma, inflammatory (allergic, environmental irritants) or infectious (eg: Mycoplasma) bronchitis, parasites, and chronic obstructive pulmonary disease (COPD) are examples. Initial treatment for suspected bronchial disease includes oxygen therapy and usually a bronchodilator, either terbutaline (a beta-agonist) or aminophylline/theophylline (both phosphodiesterase inhibitors). Even though terbutaline is primarily a β_2 agonist, it can have cardiostimulatory effects; therefore, many avoid the β-agonist in animals with known significant heart disease or if it is unclear whether the animal is dyspneic from heart failure. Anti-inflammatory doses of corticosteroids are also often indicated and helpful for treating animals with bronchial disease.

Pulmonary parenchyma

Pulmonary parenchymal disease manifests with a *restrictive respiratory pattern* – short, shallow respirations, sometimes accompanied by profound abdominal effort. Examination findings consistent with parenchymal disease include normal to loud bronchovesicular sounds or crackles. Lung sounds may actually be diminished in animals with such severe disease that the most of the alveoli in an area are collapsed or full of fluid such that no air moves through it. Additional clinical signs more specific to the underlying cause may also occur in animals with parenchymal disease. Cough (in dogs), haemoptysis (from haemorrhage, contusions), fever mucopurulent nasal discharge or halitosis (with pneumonia) or a murmur, arrhythmia, or gallop rhythm (for heart failure) may also be identified in some patients. Some of the more common differentials for parenchymal disease include pneumonia (aspiration, bacterial, fungal, parasitic, viral), neoplasia, contusion or haemorrhage, oedema (cardiogenic, non-cardiogenic), inflammation (such as pulmonary infiltrates of eosinophils), heartworms, pulmonary thromboembolism, smoke/toxin inhalation, and acute respiratory distress syndrome (ARDS).

Initial treatment for animals with parenchymal disease, as for all other causes of dyspnoea, is oxygen therapy. Because of the variety of parenchymal diseases, treatment for each disease is considerably different. Other than congestive heart failure, most parenchymal diseases do not respond quickly to appropriate therapy. So if history and physical examination do not identify or strongly suggest the etiology, additional diagnostics are often necessary to tailor therapy.

Pleural space

Pleural space disease manifests with a *restrictive respiratory pattern*, usually with an exaggerated inspiration and excessive abdominal effort. Differentials for pleural space disease include pneumothorax, haemothorax, pyothorax, chylothorax, pure or modified transudates (hydrothorax) or neoplastic effusions. Soft tissue differentials include neoplastic masses, abscesses or granulomas, or diaphragmatic hernia.

Lung and heart sounds are muffled. Auscultation may reveal a fluid- or air-lung interface, a distinct line that separates normal lung sounds from muffled lung sounds. Sometimes, borborygmi may be heard with a diaphragmatic hernia. Cats that have severe pleural effusion sometimes gag or retch. Animals with right-sided congestive heart failure may have a murmur or arrhythmia.

Initial treatment for pleural space disease is oxygen therapy followed quickly by therapeutic thoracocentesis. Any fluid obtained should be submitted for fluid analysis/cytology, +/- culture, and other biochemical tests as indicated by the cytology. If diaphragmatic hernia is strongly suspected (such as by presence of borborygmi in the thorax), centesis should be done with extra care if performed to remove air from a distended stomach in the thorax. Thoracostomy tube may be indicated for persistent or recurrent pneumothorax.

Thoracic wall and diaphragm

Thoracic wall disease can sometimes be easy to identify because of obvious disruption of the thoracic cage, usually from trauma. Other times, physical findings may be subtler and an animal may hypoventilate from failure of the bellows apparatus, have decreased chest wall movement, increased abdominal movement on inspiration, or lack of intercostal movement.

Differentials for hypoventilation primarily include trauma and neuromuscular causes. Thoracic wall trauma causing hypoventilation can include rib fractures, flail chest, or open/penetrating chest wounds. These may also be accompanied by pulmonary parenchymal injury, such as contusion. Non-traumatic differentials for hypoventilation include peripheral neurologic causes such as myasthenia gravis, botulism, tick paralysis, polyradiculoneuritis (coonhound paralysis), or tetanus, and central neurologic causes such diseases of the diseases of the brain, cervical spine or phrenic nerve. Electrolyte abnormalities (hypokalemia), musculoskeletal disease, and severe abdominal distension can also interfere with normal thoracic wall function.

Initial treatment for thoracic wall disease is oxygen supplementation. For thoracic traumatic cases, work to protect the integrity of the pleural space - if there are any obvious holes or a flail chest, cover the holes and put the (covered) damaged side ***down*** on the table. This will help stabilize the chest wall and hopefully allows aeration of the good lungs, thus making it easier for the animal to ventilate. If the damage is extensive and the animal cannot be stabilized with bandaging and positioning manoeuvres, it must be rapidly intubated and ventilated until the damage can be treated. Neuromuscular causes for hypoventilation generally require intubation and ventilation if severe. If ascites or gastric dilatation (such as from GDV) is interfering with respiration, gas or fluid should be evacuated until the animal can ventilate comfortably.

During diagnosis and treatment of the underlying cause of hypoventilation, monitoring for adequacy of ventilation using end tidal CO_2 or (venous) blood gas, if available, can be useful. Monitoring for oxygenation (pulse oximetry) is also important.

Masqueraders

There are other problems that may look like dyspnoea on first glance. Fear/excitement, hyperthermia, pain, rib fractures, metabolic acidosis (diabetic ketoacidosis, lactic acidosis, uraemia), anaemia, haemoglobinopathy, and hypoglycaemia (especially in neonates) - all can look remarkably like dyspnoea. A thorough history, physical examination and other diagnostics such as pulse oximetry, radiography, and blood work may be needed to identify these causes.

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SEPSIS: THE SURVIVING SEPSIS CAMPAIGN AND RELEVANCE TO VETERINARY PATIENTS

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ABSTRACT

Sepsis is a systemic inflammatory response to infection. Sepsis can occur subsequent to severe infection with any type of organism at sites throughout the body. Early sepsis is characterized clinically by the hyperdynamic phase in dogs: fever, tachycardia, vasodilation, bounding pulses, and red mucous membranes. Cats do not display a hyperdynamic phase, instead manifesting pallor, hypotension, abdominal pain and often a relative bradycardia. The Human Surviving Sepsis Campaign Early and aggressive resuscitation is accomplished with fluids and pressors/inotropes, as needed, and should be completed as soon as possible after sepsis identification. Early antibiotic administration has been associated with better patient outcomes for people with septic shock. Use of corticosteroids is indicated only for treating septic patients that are still hypotensive despite full volume resuscitation and pressor/inotrope therapy. Additional therapy includes source control, and diligent nursing care and monitoring.

INTRODUCTION

Sepsis is a common and potentially deadly consequence of severe infection. Fundamentally, sepsis is a systemic response resulting from unchecked disseminated inflammation in response to an infectious organism. Septic shock is a subset of sepsis in which the ensuing circulatory and metabolic abnormalities lead to hypotension and an increased mortality.

Sepsis can result from infection in any number of locations in the body. Common sources include septic peritonitis from foreign body-induced or ulcer-induced perforation, enteritis or colitis, pyometra, pyelonephritis, hepatic abscess, prostatic abscess, pyothorax, severe pneumonia, endocarditis, meningitis, deep pyoderma or infection of burn or other skin lesions, bite wounds, septic arthritis and infections secondary to immunosuppression such as by chemotherapy, immunosuppressive drugs or primary neutropenias.

Bacteria are classically considered as causes of sepsis. The trigger for bacterial sepsis is a microbial toxin such as endotoxin from gram-negative organisms or the release of exotoxins or peptidoglycans from gram-positive organisms. Fungal, viral and protozoal organisms can also induce sepsis.

White blood cells and platelets are recruited to sites of infection and inflammation by cytokines, such as tumor necrosis factor and IL-1, and contribute to inflammation and local activation of the coagulation cascade. Normally, a balance of pro and anti-inflammatory cytokines keeps the inflammation and coagulation at a local level. Dysregulation of this local response leads to systemic inflammation and microthrombosis, which can progress to organ dysfunction and organ failure. Overproduction of nitric oxide contributes to vasodilation and vasodilatory shock. Endothelial dysfunction, increased vascular permeability, and mitochondrial dysfunction can also contribute to cellular oxygen deficits.

RECOGNIZING SEPSIS IN DOGS AND CATS

Early identification of the septic patient, which can be difficult, especially in the cat, is paramount to successful treatment of sepsis. Sepsis occurs as a continuum of clinical signs. Early stages are marked by a hyperdynamic response in the dog (not in the cat) while later stages are marked by progressive Systemic Inflammatory Response (SIRS), Multiple Organ Dysfunction Syndrome (MODS) and shock.

In the dog[3,4], early signs of sepsis include tachycardia, bounding pulses, rapid capillary refill time, red or bright pink (injected) mucous membranes, and fever.[3,4] Tachycardia and an increase in cardiac contractility occur in response to tissue hypoxia. Bounding pulses result from the increase in cardiac output coupled with the systemic vasodilation. Vasodilation is also the cause of the injected mucous membranes that characterize this early “hyperdynamic” phase of septic shock in the dog. Fever is induced by the effects of inflammatory mediators on the thermoregulatory centres in the brain. Glucose

can be increased, decreased or normal in this stage due to dysregulation of glucose production and consumption within the body. Signs of late sepsis include hypoglycaemia, thready pulses, prolonged capillary refill time, pale mucous membranes, cool extremities, stupor, hypothermia and multiple organ failure. Distributive shock is caused by massive inflammatory mediator-induced systemic vasodilation and myocardial dysfunction.

As for many clinical conditions, the cat has a unique presentation for sepsis that is different from the dog and most other species.[1] The hyperdynamic phase of septic shock is not appreciated in cats; they do not develop injected mucous membranes, bounding pulses, or sometimes even tachycardia. Sepsis in cats is generally marked by lethargy, pale mucous membranes, tachypnoea, weak pulses, hypotension, hypothermia, icterus, and diffuse abdominal pain (even in the absence of a primary abdominal problem). Cats may present with tachycardia or a relative bradycardia, a heart rate, which is inappropriately low given the state of shock (eg 120-140 bpm).

TREATMENT OF SEPSIS

Sepsis has an extremely high mortality rate, estimated to be approximately 30% in people with severe sepsis and up to 50% of those in septic shock. Mortality in dogs is also approximately 50%, with increasing mortality rates for dogs with MODS.

In 2001, a landmark study by Rivers, et al,[6] showed a marked reduction in mortality rate for patients treated with “Early Goal-Directed septic patients in which CVP, MAP, central venous oxygen saturation, and haematocrit were optimized for the first six hours of hospitalization (compared to standard therapy) showed significant increases in mean central venous oxygen saturation, lower lactate, lower base deficit, and higher pH. Early goal-directed therapy group also showed reduced in-hospital (38% versus 59%), 28-day and 60-day mortality compared to patients receiving standard therapy.

The Surviving Sepsis Campaign[5]

Also in 2001, a group of international experts in sepsis and critical care came together and issued the “Barcelona Declaration” in an attempt to improve recognition and outcome for patients with sepsis. Their ultimate goal was to reduce worldwide sepsis mortality by 25 % within five years via a 6 point action plan which included Awareness, Diagnosis, Treatment, Education, Counseling, and Referral of septic patients. Furthermore, the group began a critical evaluation of evidence to form a set of recommendations known as the “Surviving Sepsis Campaign (SSC)” which uses a the Grades of Recommendation, Assessment, Development and Evaluation (GRADE) system to help qualify the quality of evidence from high to very low and to clarify the strength of recommendations form weak to strong. The first evidence-based recommendations were published in 2004 and these were revised and updated with new data in 2008 and 2012 and 2016. Published SSC updates, its revisions, new recommendations, tools for implementation, and patient/care-giver information are available online at the Surviving Sepsis Campaign website. (www.survivingsepsis.org)

Because the volume of data presented in the Surviving Sepsis Campaign was immense, the recommendations were simplified by grouping them into “severe sepsis bundles.” The bundles are defined as groups of therapies that may be more efficacious when implemented together, rather than individually. Subsequent to the 2016 SSC revision, the updated bundle recommendations are pending. To date, the SSC recommends a “3-hour bundle” and a “6-hour bundle.”

The 3-hour bundle should be completed within the first 3 hours of identification of sepsis/sepsis shock. It includes 1) measuring lactate, 2) obtaining cultures prior to antibiotic administration, 3) administering broad spectrum antibiotics as soon as possible after identification of sepsis, 4) and fluid resuscitation if hypotensive or hyperlactataemic. The 6-hour bundle includes 5) treating hypotension with pressors as needed to maintain a mean blood pressure of 65 mmHg, 6) reassessing vascular volume if hypotension is persistent, and 7) re-measuring lactate if it was initially elevated.

Lactate is a product of anaerobic metabolism. Decreased oxygen delivery (oxygen content, blood flow, blood pressure) and decreased lactate metabolism (hepatic dysfunction) can contribute to lactate accumulation. Lactate levels are prognostic in human septic shock, particularly if hyperlactatemia is persistent despite therapy. Lactate is also a better prognosticator than oxygen variables and may be better at identifying patients with hypoperfusion that are not yet hypotensive. Studies in dogs have also shown an association between higher lactate levels and persistent hyperlactatemia with poorer

prognosis. Several point-of -are devices can be reliably used for lactate measurement in small animals. Given its frequent association with prognosis, it makes sense to measure lactate in critically ill patients.

Cultures and antimicrobial therapy

Obtaining cultures is recommended as long as it does not interfere with timely starting of antibiotic therapy. The primary site(s) of infection should be cultured as soon as possible. The SSC also recommends obtaining two or more blood cultures from septic patients; one or more should be percutaneous and one should be obtained from every vascular access device that has been in place for >48 hours. Source control (eg, surgery, drainage, debridement, as indicated) at the site of infection should be achieved as soon as possible after successful resuscitation.

Intravenous antibiotics should ideally be started within 1 hour of recognizing severe sepsis or septic shock. Pending culture and sensitivity results, initial empirical antibiotic choice should be broad spectrum and cover gram negative, gram positive and anaerobic organisms or cover the presumed organisms common at the presumed source of infection. Antibiotic choice must also take into account penetration at the site of infection, potential side effects, and underlying organ dysfunction in the patient. Once culture and sensitivity results have been obtained, antibiotic choices may need to be changed. If combination therapy is employed, the regimen should be reduced once susceptibilities are available or decreased once a non-infectious cause is identified. Total duration of therapy is limited to 7-10 days unless the source cannot be drained or immune deficiencies are present.

Multiple human sepsis studies looking at timing of antibiotic therapy have been published since the last SSC recommendations. Most support early administration of appropriate antibiotics. Worse prognosis has been reported for patients that start antibiotics after achieving septic shock. Patients at low risk of death receiving combination therapy had higher mortality than those receiving monotherapy in one study. One study showed mortality was no worse for patients started on the wrong antibiotic (organisms ultimately found not to be susceptible) as long as it was started early after sepsis identification. There are no veterinary studies critically evaluating antibiotic timing in sepsis outcome.

Fluid therapy and management of hypotension

Resuscitation should begin immediately upon identification of hypotension or hyperlactatemia. Combinations of isotonic and hypertonic crystalloids, hydroxyethylstarches, and biologic colloids (plasma, albumin) can be use for fluid resuscitation. In human medicine, sepsis is a contraindication for using synthetic colloids; the effects of hydroxyethylstarches on veterinary patients are less clearly defined. Human albumin is recommended for people requiring colloids.

In veterinary medicine, species-specific albumins are either unavailable or limited in availability (canine albumin), and human albumin is highly antigenic and likely to induce a complication. Many studies exist looking at fluid types and responses in septic animals, with each fluid seeming to have pros and cons in different situations. Fluid boluses should be used to treat hypotension as long as there is a response to the boluses (an improvement in pulse pressure, stroke volume, blood pressure, heart rate); and the patients should be reassessed regularly to help guide need for additional fluid therapy. Fluid overload has recently become a hot topic in human critical care, as it has been associated with increased mortality in many studies. After volume resuscitation, ongoing fluid therapy with crystalloids and colloids is continued to address hypovolemia, dehydration, ongoing fluid losses, electrolyte imbalances, low oncotic pressure and coagulation abnormalities (DIC).

Constant rate infusions of *pressors or inotropes* may be needed for hypotensive patients unresponsive to fluid resuscitation. In humans, the two SSC drugs of choice are 1) norepinephrine (NE) and 2) epinephrine with a goal of titrating to a mean arterial pressure of $\geq$ 65 mmHg. Dopamine is considered an alternate to NE in people with absolute or relative bradycardia and low risk of tachyarrhythmias. In humans, dopamine is more often associated with tachycardia and tachyarrhythmias, which has caused dopamine to be less favoured except in cases of relative bradycardia. Use of dopamine fits with most of the septic cats where a higher heart rate may be a beneficial effect of the dopamine, as many feline patients are relatively bradycardic. Endogenous vasopressin (ADH) levels have been documented to decrease in septic shock patients. Vasopressin is considered as an addition to NE. Dobutamine is recommended for human patients with ongoing hypotension after vasopressor use.

Steroids are essential for maintenance of vasomotor tone and vascular response to catecholamines; thus, physiologic doses of steroids may be indicated in patients with absolute or relative adrenal

insufficiency. Relative adrenal insufficiency (or Critical-illness related corticosteroid Insufficiency, CIRCI) has been documented in humans and animals[2] with sepsis. Patients are suspected of having sepsis-induced adrenal insufficiency based on persistent hypotension (septic shock) despite adequate fluid loading and lack of response to pressors and inotropes; these patients may benefit from a supraphysiologic dose of corticosteroids. SSC does not recommend ACTH testing in those thought to have adrenal insufficiency in sepsis. The drug of choice in human medicine is hydrocortisone given as a constant infusion that is tapered once vasopressors are no longer required. The optimal steroid regimen in veterinary patients is unknown.

MONITORING

The septic patient is often unstable and is at risk for rapid decompensation. As such, intensive and frequent monitoring is imperative. Early identification of a downward trend will allow prompt management that can be life saving. Identifying problems late in the stage of decompensation may result in irreparable organ damage or death.

Monitoring of the septic patient should always include frequent assessment of physiologic parameters including temperature, heart rate, respiratory rate, respiratory effort, pulse rate and quality, mucous membrane color, capillary refill time, urine output and mentation. Continuous electrocardiogram (ECG) and blood pressure are also essential. Pulse oximetry and blood gases are useful monitoring tools for most septic patients. Repeat bloodwork evaluation should include at least twice daily monitoring of PCV/TS and glucose. CBC, chemistry, and coagulation profile should also be routinely monitored.

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RECOGNIZING AND UNDERSTANDING SHOCK

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ABSTRACT

Shock is a complex clinical condition that results when cellular energy production is inadequate to meet metabolic requirements. There a numerous potential causes for shock and these can be broadly classified: hypovolemic, distributive, cardiogenic, obstructive, and metabolic. Clinical signs of shock are mediated by the sympathetic nervous system and vary with the severity of the patient's condition. For all but cardiogenic shock, a fluid bolus is the first step in treating shock. Supportive and symptomatic care and monitoring are also indicated. The goal of shock therapy is to restore effective circulating volume as evidenced by normalization of patient vital signs.

WHAT IS SHOCK?

Shock is defined as inadequate cellular energy production resulting from insufficient oxygen or nutrient delivery to meet cellular energy requirements. This energy deprivation leads to cellular dysfunction and ultimately, to cell death. At the tissue and organ level, shock can lead to the systemic inflammatory response (SIRS) and to organ dysfunction or failure (MODS).

CATEGORIES OF SHOCK

Causes of shock are divided into several categories including hypovolemic, distributive (including septic), cardiogenic, obstructive and metabolic. Some patients can have more than one type of shock.

Hypovolemic shock

Low effective circulating blood volume. Essentially, there is not enough blood to fill the blood vessels and circulate to the tissues. Hypovolaemia can be caused by haemorrhage, loss of fluid via vomiting, diarrhoea, polyuria, or third space pooling of fluids into the chest or abdomen.

Distributive shock

State of relative hypovolaemia due either to maldistribution of blood flow, as occurs with trauma, or due to vasodilation (*vasodilatory shock*) as occurs with anaphylaxis, vasodilatory drugs, or neurologic causes. Septic shock is a subcategory of distributive shock. Septic shock begins with an infection, which leads to release of inflammatory mediators that subsequently cause dilation of vessels and maldistribution of blood flow.

Cardiogenic shock

Low forward flow of blood due to heart related problems. It may be caused by heart failure due to dilated cardiomyopathy (poor contractility), hypertrophic cardiomyopathy (poor relaxation), valvular regurgitation (leaky valves), or stenotic (narrow) lesions preventing forward flow. Cardiac arrhythmias, myocardial depression from anaesthetic drugs or sepsis, and cardiac damage from trauma or toxins can also cause cardiogenic shock.

Obstructive shock

Caused by a physical obstruction impeding blood flow. Pericardial effusion is a cause of obstructive shock because it impairs cardiac filling, especially of the right side. Thromboembolic disease (vascular blood clots), masses impeding blood flow, and kinked or compressed vessels as occurs with GDV are other examples of obstructive shock.

Metabolic shock

Caused by inadequate delivery of substrates needed to make energy. One cause is reduced blood oxygen content due to either decreased PaO_2 (dissolved O_2) such as from lung disease, reduced oxygen carrying due to anaemia, or from haemoglobinopathies (haemoglobin is defective and there is reduced ability to carry oxygen on red cells). Sepsis, heat stroke (elevated metabolic rate exceeds body's

ability to deliver substrate), cyanide poisoning (interferes with mitochondrial energy production) and hypoglycaemia are other causes of metabolic shock. This may also be called dysoxic shock, which identifies those scenarios where cellular utilization of oxygen is impaired.

CLINICAL SIGNS OF SHOCK

Patients with shock can present in several different ways, depending on how severe or advanced the condition is. Stages of shock include compensatory (or hyperdynamic), early decompensatory, or late decompensatory.

Clinical signs of patients with *compensatory shock* may include an increased respiratory rate, increased heart rate, rapid capillary refill time, normal to dark pink mucous membranes (dog), normal mentation, and normal blood pressure. These clinical signs result when epinephrine and other catecholamines are released in response to a decrease in pressure detected by aortic baroreceptors leaving the heart, thereby increasing the strength of cardiac contraction, increasing heart rate, and increasing blood pressure by increased systemic vascular resistance (via vasoconstriction). This category is also called the hyperdynamic phase of shock.

Clinical signs in patients with *early decompensatory shock* include tachycardia (cat or dog) or bradycardia (cat), prolonged capillary refill time, pale mucous membranes, decreased pulse quality, hypotension, decreased body temperature, and mental depression. These clinical signs are the result of redistribution of blood flow to the vital organs (heart and brain) with subsequent decreased blood flow to other organs. The reduced blood flow to non-vital organs leads to onset of anaerobic metabolism and lactic acidosis (a build up of lactate in the blood causing a low pH).

Clinical signs in patients with *late decompensatory shock* include bradycardia (slow heart rate) despite severe hypotension and low cardiac output, pale or cyanotic mucous membranes, absent capillary refill time, weak or nonpalpable pulses, decreased cardiac sounds, hypothermia, anuria, and stupor or coma. Cardiopulmonary arrest is imminent. These clinical signs are the result of failure of normal compensatory mechanisms due to ongoing lack of oxygen and nutrient delivery. This form of shock is often not responsive to even the most intensive therapy.

Septic shock is unique in clinical signs of shock in that it is marked by hyperaemic (red) mucous membranes and hypotension. Any animal in shock presenting with red mucous membranes should have sepsis as a top differential. Other types of vasodilatory shock, such as anaphylaxis, mast cell tumour degranulation, hypoadrenocorticism, or toxins (cyanide, carbon monoxide), should also be considered.

Additional clinical signs may be seen during or after shock and are related to decreased oxygen delivery to specific tissues. For instance, bloody diarrhoea and/or vomiting can result from poor perfusion of the intestinal tract. Acute renal failure can result from decreased renal perfusion, arrhythmias from poor perfusion of the coronary arteries, and leaky alveoli that fill with fluid (acute respiratory distress syndrome) may develop from damage to the lung.

TREATMENT FOR SHOCK

Restoration of effective circulating volume and oxygen delivery are the main goals of treating shock. Effective circulating volume is determined by blood pressure and cardiac output. Blood pressure is determined by cardiac output and systemic vascular resistance (vascular tone) which are related by the equation: BP = CO x SVR. Cardiac output is determined by heart rate and stroke volume: CO = HR x SV. Stroke volume is determined by strength of cardiac contraction and blood volume. In treating shock, we can manipulate many of these parameters to try to improve effective circulating volume.

Fluid therapy

For most cases other than cardiogenic shock, fluid therapy is the first step in treatment of shock. Shock fluid therapy involves quickly administering large quantities of intravenous crystalloid fluids or smaller quantities of hypertonic solutions or colloids. Fluid therapy improves stroke volume, which subsequently improves cardiac output and, therefore, blood pressure.

The type and amount of fluids used depends on the underlying problem and the type and severity of shock present. Isotonic crystalloids (water based solutions containing small osmotically active particles

that are permeable to cell membranes), hypertonic saline, and colloids (water based solutions containing large molecules that are restricted to the plasma compartment) can be used for fluid resuscitation. Packed red cells or whole blood can be used to resuscitate patients with catastrophic haemorrhage or severe anaemia. Plasma is not often used for resuscitation, as the product is usually frozen and unavailable for immediate use.

When administering fluids, calculate the shock dose, administer a portion of the full dose and reassess the patient. Not all animals need the entire shock dose. When using crystalloids in conjunction with colloids, the crystalloid dose is initially reduced by half to reduce the risk of volume overload.

Fluid Type	Dose	Comments
Isotonic Replacement Crystalloids (LRS, Plasmalyte, Norm-R, Hartmanns 0.9% NaCl)	Dog: 90 mL/kg Cat: 50-60 mL/kg	This dose is based on a full blood volume for the animal. Calculate the shock dose, administer ¼-1/3 of the dose over 15-20 minutes, and reassess.
Hypertonic saline	3-5 mL/kg	Administer slowly (over ~15 minutes), repeat once if needed
Colloid (hetastarch, tetrastarch)	Dog: up to 20 mL/kg Cat: up to 15 mL/kg	Administer 5 mL/kg bolus over 15 minutes, then reassess.

Treatment of compensatory shock can often be accomplished with crystalloids alone. A combination of fluid types should be used for early and late decompensatory shock as well as for disease states complicated by brain or pulmonary disease where excess fluid leakage into these organs could profoundly deteriorate organ function. Resuscitation for catastrophic hemorrhage involves stopping hemorrhage and replacing intravascular volume and oxygen carrying capacity with a combination of blood products, colloids and crystalloids. Care should be taken to prevent rapid or extreme increases in hydrostatic pressure after hemorrhage, as blood clots may be dislodged and bleeding may begin again.

Sympathomimetics: Inotropes and pressors

Patients that remain hypotensive despite aggressive fluid therapy and adequate vascular volume may need sympathomimetic support. Sympathomimetics '*mimic'* the sympathetic nervous system, the hormonal system responsible for the "fight or flight" response. Sympathomimetics provide positive inotropic or pressor support and are only administered if the patient has adequate circulating volume. Positive inotropic drugs (β-agonists) increase strength of cardiac contraction; thereby improving cardiac output and, subsequently, blood pressure. Heart rate is also often increased. Examples of drugs used for their positive inotropic effects include dobutamine, mid dose dopamine, and epinephrine. Vasopressors, including α- agonists (eg, phenylephrine, high dose dopamine, epinephrine, norepinephrine) or vasopressin, increase systemic vascular resistance by causing vasoconstriction, which increases blood pressure. Inotropes and pressors may be part of therapy in some cases of cardiogenic shock, especially those cases of myocardial failure (eg: dilated cardiomyopathy).

Metabolic shock or dysoxic shock treatment is aimed at the specific underlying cause. For instance, animals in shock from anaemia need red cell transfusions, animals with inability to carry oxygen on haemoglobin because of Tylenol toxicity need antioxidant therapy, hypoglycaemic animals need glucose, etc.

Other therapy

Fluids and sympathomimetics are the two main treatments to restore effective circulating volume and eliminate the state of shock. Other therapies are needed to combat the secondary effects that reduced oxygen delivery has on tissues.

Warming

Animals in shock are often hypothermic. This is especially true for cats. Additionally, the feline cholinergic receptors are not as responsive to catecholamines during hypothermia, which may contribute to the development and maintenance of hypotension in the cat. Compared to dogs, smaller aliquots of fluids should be administered more slowly and the cat should be actively rewarmed to a temperature of 99-100°F. If the cat is still hypotensive after full rewarming, additional fluids may be administered. Aggressive fluid resuscitation prior to rewarming can lead to volume overload once the cat is normothermic.

Antibiotics
Sometimes indicated as part of treating shock or its aftermath. Antibiotics are always indicated in states of septic shock or if the animal has a documented infection. Evidence or suspicion of breach of normal body defenses, such as an animal that is sloughing its gut, is another clear indication for antibiotic therapy to prevent translocation of bacteria across the body surface and into the blood stream, causing a secondary sepsis.

Corticosteroids
Controversial therapy. Steroids are hypothesized to be good for shock therapy because they reduce inflammation and stabilize lysosomal and cell membranes and improve some aspects of metabolism (eg, endogenous glucose production). Steroids can also have deleterious effects including immunosuppression and gastrointestinal bleeding. Studies on the efficacy of steroids have had mixed results. Most have demonstrated that steroids are effective if given BEFORE the insult, not after. Some studies have even shown higher mortality rates in patients treated with steroids. There are “shock doses” of steroids published in some drug formularies – the doses are usually extremely high immunosuppressive doses which are more likely to have negative side effects on the GI tract than standard anti-inflammatory doses. Steroids are generally not indicated for shock.

There are two instances when steroids are indicated for treating shock. The first is for patients with relative or absolute adrenal insufficiency. Relative adrenal insufficiency has been documented in septic patients. The second potential indication is in anaphylactic shock for which steroids are sometimes given in conjunction with antihistamines and epinephrine.

ENDPOINTS OF SHOCK RESUSCITATION

The goal of shock therapy is to titrate fluids and medications to administer the minimum that is needed to reach resuscitation endpoints. Endpoints include restoration of effective circulating fluid volume as evidenced by pink mucous membranes with normal CRT, normal temperature and heart rate, strong pulse quality and normal blood pressure, improved mentation, and adequate urine output. Once endpoints have been achieved, additional fluid therapy should be instituted to maintain intravascular volume and perfusion and to address hydration and ongoing sensible and insensible losses. Type and rate of fluid administered depends on the underlying problem.

If endpoints are not achieved with fluid therapy, causes of nonresponsive shock should be investigated and treated. The most common causes of nonresponsive shock include inadequate volume replacement, ongoing losses (such as via haemorrhage or third spacing/extravasation) excessive vasodilation or vasoconstriction, hypoglycaemia, electrolyte imbalances, underlying cardiac dysfunction or arrhythmias, ischemia/hypoxia, hypoadrenocorticism, hypothyroidism, or hypothermia.

MONITORING

Animals that are treated for shock and stabilized must be monitored to be sure they do not become critical again. Animals may respond initially to fluid boluses but then slip back into a state of shock as fluid leaks from vessels or is lost as haemorrhage or GI, urinary or third space losses. Intensive monitoring and treatment for other organ dysfunction is also important. Patients suffering from shock are at risk for a systemic inflammatory response syndrome (SIRS), which can progress to multiple organ dysfunction syndrome (MODS) and ultimately multiorgan failure (MOFS).

Monitoring can help identify subtle changes in the patient that can be treated early. Monitoring vital parameters, (temperature, pulse respiratory rate and effort), pulse quality, mucous membranes and CRT, mentation), blood pressure, and urine output give indications of adequacy of perfusion. Monitoring of glucose, oxygenation parameters, ECG, respiratory or gastrointestinal signs, clotting parameters, and neurologic status gives indication of other organ function.

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TOXICITIES: TREATMENT AND PRINCIPLES

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ABSTRACT

The initial management of the poisoned patients should include stabilisation of the patient after which a good history should be obtained from the owner. Once stable decontamination of the patient can take place. Decontamination inhibits or minimises further absorption of the toxin and promote its excretion or elimination from the body. It can include bathing, dilution, induction of emesis, gastric lavage and the use of absorbents and cathartics. Intravenous lipid emulsion therapy is being used more and more in veterinary medicine for the treatment of patients that have been poisoned with lipophilic compounds. ILET is a relatively cheap and easy to administer, and is considered comparatively safe, but should only be used in cases where the standard of care or antidote therapy is cost prohibitive or the toxicosis is severe enough to require non standard interventions, and the owner has given consent.

INTRODUCTION

Veterinarians encounter accidental, and even malicious poisoning of small animal patients relatively frequently due to the availability of various toxic substances in and around the household. Not only does each of the potential poisons have various toxicities, but in veterinary medicine there is also species differences in toxicity that needs to be taken into account. The initial management of the poisoned patients should include stabilisation (if showing clinical signs), and then obtaining a good history from the owner. The clinician should try to gather as much information as possible about the toxic substance involved, as this will aid in the risk assessment that is to follow once the patient is stable. This risk assessment will help the clinician determine the most appropriate approach to decontamination and treatment of this specific poisoned patient to ensure the best outcome.

INITIAL STABILISATION

A veterinarian should examine patients with suspected intoxication immediately. The patients should be triaged by assessing the airways for patency and determine their ability to protect their airways, breathing, circulation and any dysfunction of the central nervous system (CNS) [1,2]. Any life threatening disorders detected must be treated and managed appropriately as with any other emergency patient[3] (e.g. flow by oxygen if in respiratory distress, seizure management if seizing etc). Once the patient is stable, or if it presented in a stable condition, a thorough history should be obtained from the owner and a full physical examination performed on the patient[1].

HISTORY

It is important to try to establish what the patient has been exposed to (active ingredient) and the formulation of the product (extended release or a sustained release etc.). Asking the owner to bring in the container or product may help with this. Other details that should be confirmed are what dose the animal was exposed to, the time of exposure, did the owner see any clinical signs before the animal was brought in, and did they give any home treatment[1,4]. Once this information has been established, the clinician can then administer an antidote (if available) or decide on the appropriate method of decontamination and supportive care for that patient[4]. In veterinary medicine there are only a few toxicants that have specific antidotes or reversal agents[5] so decontamination and supportive care remains the basis of the management and treatment in these patient.

DECONTAMINATION OF THE INTOXICATED PATIENT

Decontamination inhibits or minimises further absorption of the toxin and promote its excretion or elimination from the body. The use of decontamination will depend on the time of exposure, as it can only be performed within a narrow window of time (usually < 1 to 2 hours after exposure), the type of toxin involved and the potential for adverse effects to occur from the decontamination process[1,6]. Decontamination can include

dilution, induction of emesis, gastric lavage and the use of absorbents and cathartics[7]. Dermal decontamination should only be performed once the patient is stable. Dishwashing soaps are usually recommended over shampoos because of their ability to disperse greasy substances. Insecticidal shampoos should never be used for dermal decontamination. In some cases, bathing may need to be repeated to completely remove the substance. After bathing, the patient should be thoroughly rinsed to remove any soap from the skin and coat and dried well[8].

Dilution

Is usually used if a patient has ingested an irritant or corrosive substance that causes irritation or ulceration to the gastrointestinal tract. Small amounts (2-6ml/kg) of water or milk can be given to the patient to decrease the irritating effect of the ingested substance[7,8]. Do not exceed the recommended volume of 2-6ml/kg, as larger amounts could lead to vomiting, which will cause the oesophagus to be re-exposed to the corrosive substance and will increase the risk of aspiration[7]. For ocular exposure, irrigation with copious quantity of body temperature saline or water for at least 20 – 30 minutes should be performed to dilute and wash away the toxin in question thereby prevent further exposure[8].

Induction of emesis

One of the primary ways of decontaminating veterinary patients is via induction of emesis. This is usually only effective if done as soon as possible (within 2–3 hours of ingestion) as efficacy declines the longer the time between ingestion and emesis. Insufficient mass in the stomach may result in the retrieval of only small amounts of the ingested toxin when inducing emesis so feeding a small moist meal before inducing emesis may increase the likelihood of the emesis being productive[7,8]. Not all cases are suitable for induction of emesis as there are a number of contraindications to the use of emetics, also, some substances (e.g. cannabis, antihistamines) have an anti-emetic effect and so emetics would be ineffective in cases where these products have been ingested[9]. Emesis should never be induced if the animal is showing signs of a reduced cough reflex, somnolence or has seizures[1]. Hydrogen peroxide (H_2O_2) and apomorphine are frequently used to induce emesis in canine patients. Pet owners are also often advised telephonically by veterinarians to administer hydrogen peroxide for immediate emesis at home for intoxicated pets. The published dose recommended is 1–2 mL/kg of 3% H_2O_2 administered orally[1,7,10]. In a recent study, it was suggests that the use of 3% H_2O_2 to induce emesis in dogs should not be considered entirely innocuous as they found it caused substantial gastric mucosal degeneration, necrosis, inflammation, haemorrhage, and oedema that was sustained for 1 week following the administration of the H_2O_2. These effects generally resolved by 2 weeks post administration. From this reason, apomorphine should be the emetic of choice in dogs, and vomiting typically occurs within 1–20 minutes of administration (depending on route of administration). If apomorphine is not readily available for use within 1–2 hours of toxin ingestion, a veterinarian may consider advising at-home use of H_2O_2, if the benefits of decontamination outweigh the risks of its use[10]. Induction of emesis in cats is challenging. Alpha-2 adrenergic agonists (e.g. xylazine, medetomidine) have been used off-label as emetics in cats, as vomiting is an adverse side effect of these drugs. A study found that both xylazine and medetomidine are equally effective in inducing emesis in cats when administered IM[11]. There are no safe, effective emetic agents for pet owners to use at home in cats[1].

Gastric lavage

Not commonly performed as it requires general anaesthesia, but when it is done, a cuffed endotracheal tube should always be in place to prevent aspiration of stomach contents. Gastric lavage involves the passage of a tube via the mouth into the stomach, followed by repeated administration and removal of small volumes (5-10ml/kg) of warm liquid to flush out the stomach contents. The end of the tube should be occluded before its removal[8]. Activated charcoal can be placed into the stomach through the tube before it is removed. The indications to perform a gastric lavage are in symptomatic patients that are showing clinical signs predisposing them toward aspiration pneumonia, but still need controlled decontamination or when the toxicant or ingested material can potentially cause a bezoar or concretion or foreign body[1,4,8]. *Absorbents* - These products are used to prevent the systemic absorption of the toxicant from the gastrointestinal tract by absorbing to the toxin and facilitate its excretion. The most common absorbent used is activated charcoal (AC)[7]. It has a large surface area enabling it to absorb many drugs and toxins and is not absorbed in the gastrointestinal tract (GIT) allowing all the ingested charcoal to be excreted in the faeces.[12] The use of AC in human medicine is declining in favour of other treatment modalities such as haemodialysis and plasmapheresis, but due to limited availability of theses treatment modalities in veterinary medicine along with financial limitations of pet owners, activated charcoal is still used as an absorbent in animals[13]. To maximize adsorption of the toxicant, AC should be administered as soon as possible after the exposure. In veterinary medicine, administration of AC as long as six hours out may still be beneficial with certain toxicoses, particularly if the toxicant has delayed release (e.g. extended or sustained release) or undergoes

enterohepatic recirculation[13]. The recommended dose of AC is 1-5g/kg once off along with a cathartic to encourage transit through the GIT[12]. Multidoses of AC can be used at 1-2g/kg, without a cathartic every 4 -6 hours for 24 hours if the ingested toxicant undergoes enterohepatic recirculation, or is a long acting or slow release formulation. AC alone, and in combined with a cathartic can lead to hypernatremia due to fluid shifts in the intestines, therefore it is very important not to administer it to dehydrated patients, and if multidosing is used, to monitor the sodium levels regularly[7,12]. Other contraindications for using AC is if the animal cannot protect its airway, has ileus, or is in hypovolaemic shock, and in the cases that have ingested toxins that are not bound by AC effectively e.g. alcohol, heavy metals, petroleum products etc.[7,12]. Giving AC with food can reduce the total absorptive capacity of the AC but the amount was found not to be clinically significant, therefore it can be given with food if needed[14].

Cathartics

These increase the speed and transit time of material in the GI tract, enhancing elimination of the toxin and decrease the time available for systemic toxin absorption[13]. For this reason, they are often used with AC. Cathartics are contraindicated in patients that are dehydrated, have ileus or an intestinal obstruction[7].

INTRAVENOUS LIPID EMULSION THERAPY (ILET)

Lipid emulsions are used as a component of parenteral nutrition and as a vehicle for drug delivery. In human medicine, the use of intravenous lipid emulsions (ILE) in the resuscitation of patients that have received an accidental overdose of local anesthetic has become common practice[15]. It has also been studied in poisonings caused by other lipophilic cardiotoxic and neurotoxic drugs with favorable results[15]. However, its use is still generally reserved for life threatening conditions caused by severe toxicoses, and those patients that don't respond to conventional therapies. The use of ILET in veterinary medicine to treat toxicities from lipophilic compounds is a relatively new treatment modality that is becoming more popular. The suitability of a compound for treatment with IV lipid therapy is determined by two factors: its lipophilicity and half-life. The list of compound that have been treated with ILET is continually growing (table 1). The use of ILE for animals that have been poisoned is off-label and considered experimental, so the owner must approve its use before it can be administered. The mechanism of action of lipid infusion is not completely understood, but there are two main theories: a lipid sink mechanism and a metabolic effect. The "lipid sink" mechanism allows lipophilic agents to partition out of the plasma and accelerate elimination. The metabolic effect is thought to be due to the lipids providing a source of energy to the myocardial cells, thereby reducing the toxic effects on the heart[15,16]. The use of ILE should only be considered in cases with severe toxicoses with a product that is lipid soluble and has a high mortality rate and/or when the traditional therapies are cost prohibited or have failed[17]. It is important that the patient's tissue perfusion and oxygenation is corrected before the ILET is started[17], so standard symptomatic and supportive care should still be continued along with the ILET[16]. The most commonly used formulation for treating lipophilic dug toxicoses is a 20% intravenous lipid emulsion (Intralipid® 20%, Fresenius Kabi). The recommended dosage guidelines have been extrapolated from human data and are as follows: administer a bolus of 1.5ml/kg IV over 1 minute, followed by a constant rate infusion (CRI) of 0.25ml/kg/min over 30-60 min. If some improvement is noted but some clinical signs persist intermittent boluses of 1.5ml/kg may be repeated at 4 – 6 hourly intervals for 24 hours and to a maximum of 8ml/kg/day. Alternatively, a constant rate infusion of 0.5ml/kg/hour for up to 24 hours is presumed reasonably safe. If no improvement is noted after the first bolus and CRI, ILET is unlikely to work and must be discontinued[17]. The patient's serum should be monitored every 2 hours and the ILET should not be repeated if the serum is orange or yellow[16]. All patients should be monitored in hospital until all their clinical parameters are normal, the signs of the toxicity have resolved and their serum is no longer lipaemic[16].

Potential adverse effects that can occur with ILET are pancreatitis due to persistent lipaemia, therefore it should be used with caution in cases with a history of pancreatitis. Some patients can develop hypersensitivity due to the ILE constituents. Extravasation may cause mild swelling and pain around the site. Side effects secondary to microbial contamination can lead to fatal septicaemia or destabilisation of the emulsion. Most commercial lipid emulsion preparations are stabile at room temperature for up to 2 years but once opened need to be used within 24 hours or discarded and replaced every 24 hours. Appropriate product storage and strict aseptic technique during the ILET is imperative to prevent contamination[15,17]. ILE can limit the therapeutic effect of regular lipophilic drugs that are given simultaneously and the hyperlipaemia can interfere with common laboratory tests. "Fat overload syndrome" has been described in human patients, however it has not been described in veterinary patients with the use of the current recommended doses[16-18].

CONCLUSION

Initial management of a poisoned patient will involve stabilisation, obtaining a history, followed by a risk assessment to determine the most appropriate approach to decontamination and further treatment. Every patient will be different and each should be managed accordingly. Due to the large number of possible toxic substances that the patients can be exposed to a poisons information center should be contacted as soon as possible if the clinician is not familiar with the toxic substance so that they can provide specific advice on treatment and management for that specific toxin.

Table 1: List of toxicants in which the use of intravenous lipid emulsion therapy has been used in small animals:

Toxicant	Reference
Baclofen	19,20
Bromethaline	21
Diltiazem	22
Ibuprofen	23
Ivermectin	20,24
Lidocaine	25
Moxidectin	20
Naproxen	26
Permethrin	27

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SURGICAL TREATMENT OF CERVICAL DISC DISEASE: VENTRAL SLOT VS. DISTRACTION FUSION STABILISATION

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ABSTRACT

Cervical myelopathy occurs in dogs with almost the same frequency as spinal conditions in the thoracolumbar and lumbosacral regions. The range of neurologic dysfunction is broad as the nerve interruption occurs far cranial in the central nervous system. The most common clinical complaint seen is severe neck pain, especially in acute cases of disc extrusion. Loss of motor function does occur with more severe spinal cord compression, but it is significantly less common than in the thoracolumbar region and tends to be more apparent in large breed dogs with chronic progressive lesions. Chronic progressive stenosis of the cervical spinal cord in large breed dogs creates its own set of clinical signs that are collectively known as Wobbler Syndrome. This condition is easy to recognise, but greater diagnostic skill is required to pin point the actual cause of the stenosis and decide whether medical verses surgical management is warranted. The cervical spinal cord is more amenable to medical management than other segments of the spinal cord. This is due to the larger canal to cord ratio and because pain is the most common presenting sign. Surgery is still the treatment of choice in most cases of acute disc disease, as it results in a rapid resolution of clinical signs and a very favourable prognosis for return to normal function. The two most common approaches for surgery of the cervical region are the ventral slot procedure and the dorsal laminectomy. This article will concentrate on the ventral approach and discuss the two main techniques for this, being ventral slot verses distraction fusion stabilisation techniques.

INTRODUCTION

Intervertebral Disc Prolapse (IVDP) is still the most common cause of compression and spinal cord stenosis in the cervical spinal cord. Here the rule that Hansen type 1 is more common in small breeds of dogs and Hansen Type II discs are seen in the larger breeds of dogs, definitely applies. Large breed dogs also suffer from a variety of other causes of cervical spinal cord compression like chronic instability, spondylolithesis and facet joint hypertrophy (also known as bulbous facet joints). The clinical signs from these stenotic lesions are collectively known as Wobbler Syndrome.

SURGICAL ANATOMY

The surgical anatomy of the cervical vertebral column consists of 7 vertebrae. No intervertebral disc is found between C1 and C2. Distinctive features of the first, second and sixth vertebra provides important landmarks for surgeons. There is a large venous sinus on the floor of the cervical vertebral canal and the cervical arteries travel lateral to the vertebrae. Luckily these are not often encountered during standard surgical techniques, as they can be responsible for severe peri-operative haemorrhage.

Main segments of the cranial spinal cord include C1-5, C6-T2 and caudal to T2. Tetraparesis is seen in all of these segments; however it is accompanied by normal to hyperreflexic thoracic limb reflexes when segments C1-5 are affected. Hyporeflexia of the thoracic limb with a decreased withdrawel reflex, usually but not always, indicates a pathologic process involving C6-T2 segments. The presence of concurrent Horner's Syndrome, may indicate a C6-T2 lesion, but more specifically refers to T1-T3 lesions.

HISTORY AND CLINICAL EXAM

Observation of the animal's gait, posture and mentation, as well as postural reactions, reflexes and cranial nerve function are all-important steps in the diagnosis of cervical spinal cord compression. We need to know how long the signs have been present, at what age they started, how fast the clinical signs have progressed and what previous treatments were given.

Large breed dogs are often easier to examine outside the consulting room on a “non-slip” surface, where they can be trotted out and turned in circles. Carefully note any muscle atrophy, asymmetry, ataxia, high stepping gait abnormalities or dragging of the claws. “Root signs” caused by compression of specific nerve roots will manifest as pain or lameness in a limb and provide us with further valuable information. Remember that treatment with an anti-inflammatory could mask the clinical signs and make the assessment of the hyperpathic level inaccurate. It is important to know this information before starting your neurological assessment.

The clinical signs of Wobbler Syndrome are characterised by compression of the cervical spinal cord or cervical nerve roots or both. It causes various degrees of neurologic dysfunction that are usually slowly progressive. The condition is typically non painful, unless specific nerve root entrapment is present. By far the highest reason for presentation of these dogs is gait abnormalities.

Nerve compression manifests differently in the forelimbs, which tend to show a choppy, spastic gait, as opposed to the pelvic limbs, which, show obvious ataxia. Central cord syndrome refers to the condition where very ventrally situated compression in the caudal cervical region cause lower motor neuron deficits in the thoracic limbs, whilst pelvic limbs are minimally affected.

DIAGNOSTIC IMAGING

Advanced diagnostic imaging modalities have changed our lives as far as explaining the pathophysiology involved in cervical spinal cord lesions. They provide specific guidelines of the origin of the compression and we can visualise the actual exiting nerve roots to evaluate the degree of impingement that they are subjected to. These techniques, however, are not a substitute for a thorough clinical work-up and good quality survey radiographs. Magnetic Resonance Imaging (MRI) and Computer Tomography (CT) examinations are much more likely to be diagnostic if the clinical examination has narrowed down the area of interest to a small segment of the spinal cord. Narrower image slices can then be utilised to identify, lateralise and quantify the lesion. Understanding the scope of MRI/CT verses conventional myelography is important. In the author’s opinion, it is beneficial to have both a myelogram and MRI study prior to making a decision on decompressive surgery. These are complicated cases with a variable prognosis and the more information a clinician has at their disposal, the better informed the client can be about the associated benefits verses risks of the procedure.

TREATMENT OPTIONS FOR CERVICAL COMPRESSIVE LESIONS

There is considerable controversy regarding the management of disc-associated cervical spondylomyelopathy. This controversy arises from the fact that the pathophysiology in many cases of Wobbler Syndrome, stems from a primary instability between cervical vertebrae. This instability creates secondary hyperplastic changes in the longitudinal ligament, facet joints and joint capsules of the vertebral column. Many surgeons feel a need to address this primary instability, however it is important to note that this “instability” has never been documented and is unlikely to be present at the time of presentation, due to the severe hyperplastic and osteoarthritic changes in these dogs. In their opinions, the compression has a dynamic component that is causing a significant compression and which is exacerbated by the dog’s normal range of motion. A vast number of distraction stabilisation or distraction fusion techniques have been described in the literature.

As mentioned earlier, cervical spinal cord lesions are much more amenable to medical management as the signs are slowly progressive. Pain is often the only clinical sign in acute cases and is typically responsive to corticosteroids or NSAIDS. A recent study compared outcomes of dogs treated medically verses surgically and found that 54% treated medically improved and 27% were unchanged at long term follow up. In the same study 81% of dogs were improved by surgical treatment in the owner’s opinions. The conclusions being that the results are very similar when owner’s long-term perceptions are considered.

The problem arises when the long-term damage to the spinal cord is assessed. We need to carefully weigh up the risk verses benefit ratio. Surgery may make these cases initially worse, but it aims to halt the progression of clinical signs and minimise the damage to the spinal cord that ongoing, low grade trauma causes.

Veterinary neurosurgeons are equally split between those who feel that distraction fusion is necessary and those, like the author, that prefer a more conservative surgical approach. The aims of ventral slot or preferably “slanted ventral slot” techniques are to remove the compression creating the spinal cord stenosis and thus relieve the clinical signs. They significantly reduce the compression affecting the cord and by leaving the ventral annulus fibrosis unharmed, barely affect the dogs post surgical vertebral stability.

Distraction stabilisation techniques should aim to provide optimal distraction over the long-term, should be able to be used in multiple adjacent disc spaces and should have minimal complications. They should also decrease the likelihood of adjacent disc disease or “domino effect” occurring. Published results suggest that no current technique fulfils all of these criteria. The most consistent problems encountered are adjacent disc disease and implant complications, many of which are catastrophic and may results in untimely euthanasia. One study of a large number of Doberman Pinschers showed that neural function immediately post surgery was normal or improved in 83% of these dogs, however in the long term only 65% were considered neurologically normal and 56 % of dogs were dead by the end of the study period, which was not an unreasonable length of time.

POST OPERATIVE CARE

Neurosurgical patients are often significantly debilitated following decompression. This is particularly true in large breed dogs, where surgery is more invasive and dogs are heavier and more uncoordinated during recovery. Until their normal range of function returns, they require a huge amount of nursing care to allow them to recover optimally. It is important that hospital nursing staff has a high level of training for this and are properly informed of which parameters need to be monitored. The post-operative care of spinal patients can basically be divided into pain control, the nutritional management, bladder management, physical rehabilitation and the management of pressure sores and other complications.

CONCLUSION

Cervical myelopathy is a particularly challenging dilemma in neurosurgery. Acute cases presenting with severe cervical pain, are easily managed with ventral slot decompressive surgery. More chronic cases in large breed dogs with inherent primary instability are much more challenging. The surgeon has to distinguish between those patients that will respond well to medical management with analgesics and anti-inflammatory verses those that require surgical decompression or surgical distraction fusion.

In the author’s opinion, distraction fusion techniques open up a whole realm of complications that far outweigh their potential benefits. The author prefers a more conservative surgical approach where an accurate diagnosis of the primary problem is made using advanced imaging techniques. The stenosis is then removed via the surgical approach that is most likely to provide accurate visualization, without negatively affecting stability. In most cases, ventral slot decompression is the author’s technique of choice and even in large ventral or ventrolateral compressions; the stenosis can be addressed sufficiently to provide immediate and long-term relief to the patient.

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CLOSURE OF LARGE SKIN WOUNDS IN COMPANION ANIMALS

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ABSTRACT

Skin represents the largest organ in the body. It is one of the most common organs that the veterinarian has to deal with on a daily basis. It is one of the most versatile organs, especially in the canine and feline patient. Skin surgery is one of the most exciting areas of surgery that enables the surgeon the chance to make a massive difference to the patient with little specialised equipment. This gives most general practitioners the chance to return the patient to normal function as quickly as possible without having to refer these cases. Techniques for closure depend on the wound, the patient and the trauma causing the wound. This will dictate how and when the wound is closed. From a simple laceration that can be closed by primary closure to the traumatic mechanical avulsion injury that needs to be treated as an open wound and then closed with multiple axial pattern flaps. These are all within reach of the general practitioner's skill set.

INTRODUCTION

The main aim of reconstructive skin surgery is to return the patient to as normal a function as soon as possible without compromising the patient further. The decision on how to close a wound depends on the condition of the patient and the wound itself.

When dealing with a patient with a massive wound caused by severe trauma it is easy to overlook the whole patient and concentrate on the wound. The rest of the body should be assessed for life threatening wounds and appropriate action taken to support the patient. Appropriate shock treatment should be administered and the patient monitored closely for deterioration. Depending on the cause of the trauma, radiographs should be taken to assess for any fractures. Open fractures and wounds present an orthopaedic emergency that should be stabilised as soon as possible depending on the condition of the patient. Effective pain control should be instituted as this will aid in recovery of the patient. Opioids form the cornerstone of pain control in severely traumatised patients. Ideally the pure agonists are mostly used even though they have potential side effects. Dissociative agents and lignocaine can be used together with opioids in intravenous anaesthesia. In anxious, uncompromised patients, medetomodine can be added to the infusion. NSAID's are useful but should not be used in hypovolemic patients. Local and regional anaesthesia can be useful in severely compromised patients. Further information on pain control can be found in other texts.

Once the patient has been assessed and stabilised, the wound can now be addressed. When deciding on how to close the wound it is important to consider.

- The cause of the wound
- The area affected by the wound
- The amount of tissue loss to the wound
- Fractures associated with the wound
- The blood supply to the wounded area and viability of the surrounding skin
- The degree of contamination of foreign material
- The degree of bacterial contamination
- The time from wound to presentation

As a general rule if there is any doubt to a wound being contaminated, it should not be closed

Contamination of a wound is an important assessment. However, local blood supply to the wound and surrounding tissue is vital in the healing of the wound and in the method of closure of the wound. The type of trauma causing the wound will affect the degree of vascular compromise to the wound. The extent of vascular damage in high velocity trauma, cutaneous avulsion and degloving wounds is usually not visible at initial presentation and these wounds should always be managed as open wounds. Open fractures are often associated with severe soft tissue and vascular damage. These

present a challenge in that the wound closure should be done as soon as possible to prevent osteomyelitis but this may not be possible. The instability caused by fractures further contributes to slow wound healing and infection of the soft tissue and bone. Ideally stabilisation of the fracture should be undertaken as soon as possible given the status of the patient. Reconstruction of the skin should be performed over the stabilised fracture if possible. External fixators are helpful in dealing with open fractures when the skin cannot be reconstructed. These enable the wound to be managed and the fracture stabilised.

Intra-venous antibiotics have a role to play. However, in areas of vascular compromise the active concentration at the wound site is questionable. The type of antibiotics initially chosen should be effective against beta lactamase producing bacteria. Once an infection becomes stabilised, the choice should be based on a culture taken from infected tissue. Wound lavage and manual debridement are the most important modalities in management of a contaminated wound with vascular compromise.

Lavage of the wound is the single most important treatment in managing an open, contaminated wound. Lavage provides the following advantages

- Rehydrate necrotic tissue
- Reduce bacterial contamination and remove foreign material
- Removes cytokines and toxins associated with infected wounds

Lavage is useful to help remove adherent dressings from the wound during bandage changes. Large volumes of flushing should be used as this removes and dilutes bacteria and contaminants in the wound.

The best lavage solution should be Isotonic and it has been suggested that solutions with a buffer are best such as ringers lactate. No evidence has shown that the use of anti-septic solutions added to ringers lactate is better. If anything any anti-septic's are cytotoxic and can inhibit wound healing. As mentioned, volume is the key in wound lavage. The larger the wound, the larger the volume of fluid used. The pressure at which this is delivered is controversial - high pressures will dislodge visible contaminants but could drive bacteria deeper into the wound. The recommended pressure is 6-8 psi. A guide line is the use of a 1L ringers bag connected to an admin set with a 3 way stop cock connected to a 20-30ml syringe used for lavage through an 18 gauge needle. Lavage should be carried out as often as needed. This can be twice a day in large exudative wounds, to every 3-5 days in wounds that are starting to granulate.

All wounds should be bandaged. This prevents contamination of the wound with hospital bacteria. Bandages create a warm moist environment that stimulates wound healing. They will act as the first stage of mechanical debridement. Adherent dressing will remove debris and bacterial load when the primary contact layer is removed. Once again bandages need to be changed as often as required by the wound. Large exudative wounds may require bandage changes twice a day, to every 3-5 days in wounds that have granulated. Bandage changes should always be performed under aseptic conditions. This will prevent hospital bacteria contaminating the wound.

Regular bandage changes in patients with severe wounds are very painful in the initial stages. Adequate analgesia should be provided during bandage changes and in-between in patients with severe injuries. Feeding is very important as large wounds will exude protein rich exudate. These patients will lose albumin and important co-factors. Daily serum plasma measurements should be taken to monitor this and fresh frozen plasma should be given as needed. I always consider placing an oesophagostomy tube at initial presentation or when stable enough for initial debridement. This will enable ease of feeding as soon as possible to help with protein levels.

Surgical debridement should be started once the patient is stable enough to undergo sedation. Surgical debridement entails staged removal of visible necrotic and non-viable tissue. This should be done with sharp sterile surgical instruments under aseptic conditions. Debridement should be performed daily until all necrotic tissue has been removed and granulation tissue is covering the wound.

Once a wound has granulated, then non-adherent dressings should be applied. These will stimulate epithelisation and contraction of the wound. A decision on closure of the wound can now be made.

WOUND CLOSURE

Primary wound closure defines a clean wound or a clean contaminated wound that has just occurred. These wounds can be closed by simple apposition of the wound edges, which allows first intention healing.

Delayed primary closure is closure of a wound by apposition of the wound edges before formation of granulation tissue. These are wounds that require some debridement prior to closure.

Secondary closure defines closure of a wound after the formation of granulation tissue.

Second intention healing describes the process when a wound is left to heal by contraction and epithelialisation.

TECHNIQUES FOR CLOSURE OF LARGE WOUNDS

The main goal of wound closure is to obtain rapid wound closure and return to function using the simplest technique. Primary closure with undermining of the surrounding skin should always be the first choice if possible. Simple tension relieving techniques can help in some cases. These techniques include simply placing more sutures in the wound to spread the tension, Far-Near suture techniques, multiple tension relieving incisions, Z-plasty and walking sutures.

In large wounds, these techniques will not result in closure of the defect. Larger wounds require local subdermal plexus flaps, axial pattern flaps, free skin grafts and even microvascular free tissue transfer flaps.

Local subdermal plexus flaps

These generally are adjacent to the wound to be closed and involve essentially moving skin from an area with ample skin to close the defect. They contain vessels of the subdermal plexus, tracking on from the base of the flap. This blood supply is less reliable than a known direct cutaneous vessel. The larger the base of the flap the better the blood supply. A rough estimate for harvesting these flaps is a ratio of length to base of 2:1. It is best to only create flaps just large enough to cover the defect.

Upon elevation of the flap, incise the skin sharply with a scalpel blade. The skin is then bluntly undermined with a pair of metenzbaum scissors. The skin should always be undermined deep to the subdermal plexus or platysma muscle where present. The flap should then be handled with extreme care, especially the tip of the flap as this has the least reliable blood supply.

There are many different shapes of local subdermal plexus flaps, with the simplest being a single pedicle advancement flap, bipedicle advancement flap, rotational flap, transposition flap, axillary and inguinal fold flaps and direct distant flaps. These flaps can be used to close a vast multitude of wounds over the body. However the size of the flap is determined by the width of the base and the number of subdermal vessels incorporated into the flap. To ensure the best chance of survival, the widest base possible and minimal tension should be used to close the wound with a local subdermal plexus flap.

Axial pattern flaps

Axial pattern flaps are the gold standard in closure of large wounds over the body, hind leg and proximal fore leg. These flaps provide durable full thickness skin and the flap brings its own direct cutaneous artery with it. The incorporation of a direct cutaneous artery is the defining characteristic over local subdermal plexus flaps. Axial pattern flaps have a 50% increased survival area than subdermal plexus flaps. These flaps can be used in areas with poor vascular supply and are more resistant to high motion.

All the direct cutaneous vessels have been described in dogs and cats and have well known landmarks, which makes harvesting the flap simple and very effective. There is no need for direct visualisation of the vessels in the base of the flap given these landmarks. Attempts to dissect out the vessels are not encouraged as it may lead to damage of the vessel and flap necrosis.

Most wounds in most areas of the body can be closed using an axial pattern flap with some manipulation of the skin. These flaps are excellent in the closure of chronic non-healing wounds, radiated wounds or clean surgical wounds post large resection as they bring a blood supply provided by the direct cutaneous artery in each flap.

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CRANIAL CRUCIATE LIGAMENT DISEASE

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ABSTRACT

Cranial cruciate disease is the most commonly encountered cause of lameness seen in small animal practice. Numerous surgical techniques have been described for the treatment of cranial cruciate deficient stifles in the dog. Broadly speaking two types of surgical techniques may be used, either an extracapsular stabilization technique or one of the tibial osteotomy techniques. Extracapsular stabilization techniques include lateral sutures, fascia lata grafts and more recently the TightRope technique by Arthrex. Tibial osteotomy techniques started with the cranial closing wedge osteotomy as described by Slocum. The Tibial Levelling Osteotomy (TUPELO) was the next procedure developed by Slocum. Other tibial osteotomy techniques include the Tibial Tuberosity Advancement (TTA), the Triple Tibial Osteotomy (TTO), the Tibial Tuberosity Advancement Rapide (TTAr), Modified Maquet Procedure (MMP) and the CORA Based Levelling Osteotomy (CBLO). All these tibial osteotomy procedure may be grouped together as Mechanical Modifying Osteotomies (MMO) or Geometric Modifying Osteotomies (GMO) and appear to give the best long-term results.

INTRODUCTION

Cranial cruciate ligament disease is the most common cause of lameness in canines. Partial and complete cranial cruciate ligament rupture leads to stifle instability i.e. craniocaudal motion of the tibia relative to the femur and internal rotation of the tibia. This stifle instability results in the progressive development of osteoarthrosis of the affected joint.

Tibio-femoral shear forces are generated during weight bearing (stance phase) and are transmitted across the stifle joint. Cranial tibial thrust is that component of the tibio-femoral shear force which results in cranial movement of the tibia relative to the femur. The cranial cruciate ligament provides a passive restraint against this cranial tibial thrust. The caudal horn of the medial meniscus is also a passive restraint against cranial tibial thrust. Active forces acting on the stifle joint include the stifle extensors (quadriceps femoris muscles), stifle flexors (semimembranous, semitendinous and biceps femoris muscles) and the extensors of the hock (gastrocnemius muscles). Cranial tibial thrust is a force created by weight bearing and muscular contraction (stifle and hock extensors) resulting in compression of the tibial plateau against the femoral condyles. In the active model of the stifle this force is counteracted by the passive restraints (CCL and caudal horn of the medial meniscus) and the active restraints (flexor muscles of the stifle).

Slocum proposed that the tibio-femoral shear force was parallel to the functional axis of the tibia and that cranial tibial thrust would be neutralized if the tibial plateau were perpendicular to the functional axis of the tibia (a TPA=0˚). The TPLO technique involves a radial osteotomy of the proximal tibia and rotation of the proximal fragment (tibial plateau) along this osteotomy to reduce the tibial plateau angle to zero. It was then determined that a TPA=0˚ resulted in a caudal tibial thrust during weight bearing and potential overload of the caudal cruciate ligament. Current clinical practice is to reduce the TPA to 5˚-7˚.

Further biomechanical investigations proposed that the tibio-femoral shear force was in fact parallel to the patella ligament. This resulted in the development of the Tibial Tuberosity Advancement technique (TTA). The TTA procedure involves a single osteotomy of the tibial tuberosity, which is then advanced a predetermined distance and stabilized with a specially designed implant. By advancing the tibial tuberosity (and insertion of the patella ligament) the tibial plateau is made perpendicular to the patella ligament.

The Triple Tibial Osteotomy was developed and combines features of the TTA and the Cranial Closing Wedge Osteotomy. The TTA allows for reduction of the tibial plateau slope to a point where it is perpendicular to the patella ligament. The TTA achieves this by some rotation of the tibial slope as well as some cranial advancement of the tibial tuberosity. This is achieved by a series of three tibial

osteotomies. There is limited specialized equipment required to perform the procedure (wedgie, saw guide and measuring device). All the tibial plateau levelling techniques appear to have similar results, complication rates and outcomes.

MEDIAL MENISCUS

Undiagnosed and untreated medial meniscus tears are a major reason for continued post-operative lameness and poor results. The medial meniscus is prone to injury due to the fact it is a passive stabilizer of cranial tibial thrust and is attached to the medial collateral ligament. This attachment to the medial collateral ligament prevents the caudal horn of the meniscus from being mobile. In a CCL deficient stifle joint the tibia moves cranially relative to the femur. The caudal pole of the medial meniscus acts as a “wedge” and counteracts cranial tibial thrust. Due to its attachment the caudal horn of the medial meniscus may become trapped between the tibial plateau and the femoral condyles. The menisci are important structures of the stifle joint in that they improve congruency between the femoral condyles and the tibial plateau, facilitate load transmission of forces across the joint and act as a passive restraint to cranial tibial thrust. Partial menisectomy and medial meniscal release reduce the meniscus’s functionality therefore resulting in overload of the articular cartilage and subchondral bone. The result being the development of osteoarthrosis. Medial meniscus release (MNR) remains a controversial topic. All menisci must be carefully examined at the time of surgery; this should include palpation with a small right-angled probe. Arthroscopy may allow more accurate examination of the caudal pole of the medial meniscus.

PRE-OPERATIVE ASSESSMENT

Accurate radiographs of the affected stifle are essential in order to accurately plan the surgery fro both TTO and TTAr. An extended mediolateral view (stifle at 135°) is required with the femoral condyles superimposed.

Measurements for the TTO procedure include the length of the patella ligament and the angle between the tibial plateau and the patella ligament. The length of the patella ligament equals the length of the tibial crest osteotomy while the angle between the tibial plateau and the patella ligament is known as the correction angle. The angle is inserted into a formula to obtain the wedge angle.
For the TTAr procedure one needs to calculate the tibial tuberosity advancement required. This is done using either the common tangent method or the “Ness” method. One also requires the measurement for the position of the Maquet hole and the thickness of the cortex at this level.

TRIPLE TIBIAL OSTEOTOMY (TTO)

TTO surgery involves a straight incomplete osteotomy of the tibial tuberosity. A wedge shaped ostectomy is then performed caudal to the tibial tuberosity osteotomy i.e. a wedge shaped piece of bone is removed form the tibial shaft. The ostectomy is then reduced and stabilized with a clover shaped bone plate with three screws in the proximal fragment and three to four screws distally. This results in the tibial plateau being reduced and the tibial tuberosity being advanced.

Indications for the TTO include partial and complete CCL rupture, especially in large breed dogs, individuals with pathological tibial plateau angles and revision of failed extracapsular stabilizations. TTO allows for correction of other abnormalities simultaneously i.e. patella luxations, genu varum/valgum and tibial torsional deformities.

TIBIAL TUBEROSITY ADVANCEMENT RAPIDE (TTAR)

TTAr involves a single straight incomplete osteotomy of the tibial tuberosity. The distal end of the osteotomy is the Maquet hole. The osteotomy is then progressively levered open until the required advancement is achieved and then the appropriate sized cage is inserted and stabilized with screws to the tibial tuberosity and the tibial diaphysis.

Indications for the TTAr are similar to those for the TTO. Correction of genu valgum/varum is not possible with the TTAr. Dogs with very steep tibial plateau angles may also be better treated with a TTO as the advancement of the tibial tuberosity with the TTAr may be too large. The largest cage available for the TTAr is a 15mm cage.

COMPLICATIONS

The overall complication rate of the TTO procedure is comparable to those of the TPLO and TTA surgeries. Most of the complications seen are relatively minor and easily corrected. The most common complications intra-operatively include fracture through the distal end of the tibial crest osteotomy, Fracture through the caudal tibial hinge/cortex, haemorrhage from the popliteal artery and uncommonly intra-articular screw placement. Post –operative complications include tibial crest fracture, infection, late meniscal tears and undiagnosed meniscal tears. Tibial crest fractures are best stabilized with a single K-wire and a figure of eight tension band. The incidence of implant failure (plate and screws) is very low.

The complication rate with TTAr is similar to those of the TTO. Most complications seen are minor complications not requiring further surgery. Complications seen included fissure formation distally from the Maquet hole and fracture of the tibial tuberosity distally. Management for these complications is not necessary unless there is displacement of the tibial tuberosity.

TTAr surgery is lees invasive than TTO surgery as no dissection is required around the caudal aspect of the tibial. This also reduces the risk of damage to the popliteal artery.

CONCLUSION

Tibial plateau levelling provides dynamic stabilization of the stifle joint during the stance phase of weight bearing. There are numerous techniques available to achieve tibial plateau levelling. Evidence based surgery does not indicate any one technique to be superior to another at this point in time. The advantages of the TTO technique are that it is a relatively simple surgery to perform, requires limited specialized equipment, has a low complication rate and has comparable results / outcomes and complication rates to the TPLO and TTA. TTAr surgery is a simple and effective method of treating cranial cruciate ligament disease. The advantage of TTAr surgery is the reduced surgical time compared to a TTO surgery. Further objective research studies are required (force plate analysis, etc.). The overall complication rate with both surgeries is acceptable and the outcomes are good clinically.

DECISION MAKING IN FRACTURE REPAIR

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ABSTRACT

Correct evaluation and assessment of fracture patients in their entirety is essential to applying the correct treatment protocols. Fractures are seldom if every life threatening injuries. It is essential that life-threatening injuries (thorax, abdomen etc.) are evaluated and treated first. Once stabilised treatment for the fracture may be performed. The fracture must be assessed, classified and an appropriate fixation method chosen for that particular fracture in that specific patient. Surgeon experience and preference is another important factor in decision making with respect to fracture management.

INTRODUCTION

Most fractures seen in small animal practice are as a result of significant trauma. It is important therefore to assess and evaluate the entire patient rather than concentrating on the fractures initially. Fractures are not life threatening injuries. Initial assessment and stabilization should be done prior to fracture evaluation and assessment. Life threatening conditions should be assessed and treated first.

Once the patient has been stabilized full evaluation of the fracture and surrounding soft tissue should be carried out.

Pain management should be instituted. Fractures distal to the elbow and stifle should be immobilized with external coaptation prior to surgical treatment.

FRACTURE CLASSIFICATION

Fractures should all be classified according to location (bone and location in the bone) as well as fracture configuration (transverse, oblique, comminuted etc.). One also needs to classify the fracture as closed or open. Open fractures may be further classified as Type I, II, IIIa, IIIb or IIIc. The distinction between different types of open fractures is based on degree of soft tissue trauma / loss and whether or not there is bone loss. Growth plate fractures in immature animal should be identified and classified according the Salter-Harris system. This allows for appropriate treatment and prognostication.

Accurate fracture classification allows one to assess the biomechanical forces that will need to be neutralized in a particular fracture and therefore which fracture fixation method would be best for that particular fracture.

FRACTURE TREATMENT PLANNING

Accurate planning for the treatment of a fracture should take into account all variables that may influence fracture healing and allow for the optimal treatment of a specific fracture. Factors to be considered include mechanical, biological and clinical factors as well as the fracture configuration. Mechanical factors include patient size, number of limbs affected, whether load sharing is possible etc. Biological factors include open vs. closed fracture, extent of soft tissue trauma, patient age, concurrent disease and type of surgical approach. Clinical factors include patient and client compliance. Mechanical factors influence the stress placed on the implant and the bone – implant interface. Biological factors influence the expected time to clinical union. Fracture healing may be thought of as a race between fracture healing and implant failure. Successful outcome depends on fracture healing occurring before implant failure. Prior to surgery one should have more than one treatment plan, i.e. a plan A, B and C.

A good knowledge of the various fracture fixation systems and their correct application techniques is essential in fracture management. Another factor to consider is surgeon experience and preference. Many times there may be more than one acceptable method of stabilization for a particular fracture. It is advisable that the surgeon use a technique that he / she is familiar with and has experience using.

PATIENT PREPARATION AND FRACTURE REDUCTION

Basic surgical principles are very important for orthopaedic surgery and the importance of soft tissue in successful fracture heal must not be underestimated. Atraumatic surgical technique is very important. Recently there has been a big move towards minimally invasive fracture repair, so called biological osteosynthesis. This technique minimizes the amount of soft tissue disruption and maintains a favorable environment for fracture healing. In this situation fracture reduction may be achieved by closed reduction, minimally invasive reduction or by the “open but do not touch” approach.

POSTOPERATIVE ASSESSMENT

Postoperative radiographs are necessary to assess reduction, alignment and implant placement. They also serve a comparison / reference to compare future radiographs to in order to assess fracture healing. Postoperative management is highly variable between different patient and fractures. Lower limb fracture generally benefit from a support bandage for 48 – 72hrs to minimize postoperative swelling. Specific fixation techniques may require more regular check ups e.g. external skeletal fixators vs. plate & screws. Follow up radiographs are generally advised every 4 – 6 weeks until clinical union is achieved. Radiographs may be taken more frequently under certain circumstances (immature patients, potential delayed or non unions).

APPROACH TO THE DYSPNOEIC BRACHYCEPHALIC PATIENT

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ABSTRACT

It is not uncommon in veterinary science that we are presented with a cyanotic dog and a frantic owner. The cause of this sudden respiratory distress can be due to Laryngeal Paralysis, Laryngeal Collapse, Tracheal collapse or a combination. These conditions are loosely grouped into the category of be Brachycephalic Syndrome (BS) or Upper Airway Obstruction Syndrome (UAOS). Our task is to recognise the condition that we see in brachycephalic dogs and to proceed swiftly to being able to stabilise them. Many of these dogs are overweight and may have concurrent heart disease or tracheal collapse. We use a combination of oxygen supplementation, corticosteroids, sedatives and cooling techniques. At a later stage, we can perform a more thorough physical exam and diagnostic techniques in order to make a diagnosis of the underlying cause. The veterinarian must decide whether medical or surgical intervention is more likely to prevent similar recurrence. Surgical intervention includes techniques to reduce the obstruction to the lumen of the upper airway. Wedge naroplasty, staphylectomy (soft palate resection), ventriculoectomy (resection of everted laryngeal ventricles) and cricoarytenoid laryngoplasty are some of these techniques that will be discussed. Postoperative care involves careful monitoring and symptomatic treatment. Dogs should be in a cool environment, have oxygen supplementation when needed and supportive medical treatment. Weight restriction and exercise moderation, is vital to the Long term success of surgery.

INTRODUCTION

Brachycephalic syndrome refers to a combination of problems causing partial or complete obstruction to the upper airway. Breeds affected include: English Bulldogs, Boxers, Boston Terriers, Pug, Shih Tzu, Pekingese, Shar Pei, French Bulldog and Cavalier King Charles Spaniel. These breeds all have a typically compressed face. They have an inherited developmental defect where the bones grow to normal width, but reduced length. The soft tissues of the head are not proportionately reduced and are often even redundant. The components of UAOS include stenotic nares, elongated soft palate, everted laryngeal saccules and tracheal hypoplasia. In more severe cases the disease condition progresses to laryngeal or tracheal collapse. Bulldogs, in particular, are very susceptible to this sudden onset of respiratory distress. Affected animals are often young (2-4 years) and otherwise relatively healthy. The condition is much more prevalent in dogs than in cats.

STABILISING THE PATIENT IN RESPIRATORY DISTRESS

The classic signs of acute respiratory distress include open mouth, laboured breathing, cyanosis, abducted forelimbs, restlessness and even collapse. These animals are often overweight on presentation and may have concurrent heart disease or tracheal collapse. Animals exhibiting these signs should be handled with care so as not to compromise them any further. Removing them from their distressed owners and taking them to a cool, quiet environment where oxygen supplementation can be administered is the first important step. Supplementary oxygen can be started immediately and can be administered via a facemask, nasal insufflation or by placing the animal in an oxygen cage (which is usually the least distressing for them). Hyperthermia develops rapidly and so cooling techniques including using icepacks directly on the animal; in the axilla, inguinal area or on the extremities or even on the intravenous fluid line should be employed. Wetting the animal and directing an electric fan on them, may also help to bring the body temperature back under control. Before physical examination is possible, the dog may require a sedative like Temgesic and possibly even treatment with an intravenous corticosteroid. They should be allowed to remain unrestrained and in the position that they are most comfortable in.

In more severe cases, the veterinarian may have to consider endotracheal intubation or even an emergency tracheostomy. Intravenous fluid administration is not usually necessary in the immediate

stabilisation and care should be taken if it is administered, not to overhydrate the already compromised lungs.

ESTABLISHING THE CAUSE OF THE RESPIRATORY DISTRESS

The clinical examination of these dogs aims to determine whether this is an upper or lower airway problem. A full history and physical examination are always needed. Determine vaccination history and any possible exposure to infectious diseases. Infectious diseases are more common in younger patients whilst bronchitis, tracheal collapse and heart conditions are more likely in older dogs.

Questions to ask include:

- How long have signs of exercise intolerance or breathing difficulty been present?
- What activity or environment initiated the attack?
- Does dog ever cough?
- Has there been any change in bark quality or intensity?
- Has there been any vomiting or regurgitation?
- Has there been a change in attitude or appetite?
- Do you have a video of the episode?

By assessing the abnormal respiratory sounds and where they originate from, we can gain some clues as to the underlying cause of the obstruction. It is important to note at his point that animals should never make a noise when breathing. Young animals, which present with noisy breathing, have to have some form of obstruction. This should be recognised by veterinarians and corrected as early as possible. We will primarily handle the causes of respiratory distress, which occur in brachycephalic dogs. These can be divided into nasal problems (stenotic nares), laryngeal obstructions (soft palate and everted laryngeal ventricles) and tracheal abnormalities (Tracheal hypoplasia).

Stenotic nares are visually apparent and easy to diagnose. Observing the external openings during inspiration, we can clearly see whether there is paradoxical closure as animal tries to breathe in. Stenotic nares may also present with signs of sneezing, inspiratory stridors or mucopurulent discharges. Other causes of nasal obstruction include traumatic, neoplastic or infectious nasal disease.

Gentle pressure over the laryngeal area can often exacerbate a respiratory problem at the level of the larynx. Stethoscopic auscultation is useful for localizing the obstruction to this region. A characteristic inspiratory stertor or snoring is a common finding in laryngeal obstruction cases. Once the problem has been isolated to the larynx, we need to decide whether we are dealing with laryngeal paralysis, obstruction by the soft palate or ventricles or laryngeal collapse. Survey radiographs should be taken of the laryngeal region as well as the thoracic cavity (3 views). This should be followed by a full visual examination of the laryngeal region with the animal lightly anaesthetised.

Tracheal collapse is a condition that occurs as a result of weakening and dorso-ventral flattening of the C shaped cartilage rings which leads to tracheal obstruction. It is not classically associated with BS, but may occur following chronic obstructive airway disease. It usually occurs in toy breeds and may well have a congenital aetiology. The clinical signs involve gagging, dyspnoea and exercise intolerance. A classic “goose-honk” cough may also be present. The diagnosis relies on good quality survey radiographs of the cervical and thoracic regions. In some instances it is important to obtain inspiratory as well as expiratory thoracic radiographs to check whether collapse occurs during specific stages of respiration and which sections of the trachea and main stem bronchi are affected. If a definitive diagnosis is still not possible, then tracheal endoscopy can be done.

VISUAL EXAMINATION OF THE UPPER AIRWAY

Patients with respiratory distress caused by BS are considered anaesthetic risks and every precaution should be taken to make sure that they survive these diagnostic procedures. Certain drugs can also inhibit laryngeal function and these should be avoided if an accurate assessment of laryngeal function is to be made. Pre-oxygenation of affected dogs for 3-5 minutes with 100% oxygen prior to induction can significantly decrease risks. Propofol is generally used as the induction agent of choice as it has the advantage of having a very short half-life. The animal should only be anaesthetised to the level

where the tongue can be retracted, but the swallowing mechanism is not affected. Oxygen supplementation should be on hand and used immediately if oxygen saturation cannot be maintained. Using a good light source and a long tongue depressor or laryngoscope we start by assessing the length of the soft palate in relation to the tip of the epiglottis. The normal guideline is that a 1-3 mm overlap between the two is acceptable. In the case of an elongated soft palate, there may be as much as a 12-20mm overlap. The palate may even be sucked caudally between the corniculate processes during respiration and this causes a severe disruption to the normal laminar airflow.

Be careful not to retract the tongue too forcefully, as this can give a false under-estimation of the length of the soft palate

The soft palate should then be lifted dorsally and the epiglottis moved ventrally so that the entrance to the glottis can be examined. This allows the relationship between the corniculate and cuneiform processes of the arytenoid cartilages to be assessed. Any contact between them or overlap may indicate a degree of laryngeal collapse. Laryngeal collapse is graded from 1-4 depending on the degree of collapse. In Grade 1 collapse, laryngeal ventricle eversion is the only abnormality detected. Grade 2 is when we see mediation deviation of the cuneiform cartilage and sometimes the presence of aryepiglottic folds. In Grade 3 collapse, the corniculate process of the arytenoid cartilage shows medial diversion and in the most severe form, Grade 4, we see complete overlap of these cartilages.
We need to establish whether the vocal cords are abducting appropriately during inspiration. In the case of an animal suffering from laryngeal paralysis, the vocal folds cannot be adequately abducted and so they obstruct the laryngeal glottis. This exacerbates the negative pressure within the airway and leads to a vicious cycle of respiratory obstruction and eventually upper airway collapse. A useful aid in confirming whether paralysis is present or not, is to inject the animal with Doxapram (Dopram) at a dose of 0.5-2 mg/kg IV whilst observing the vocal folds for movement. The increased respiratory effort that occurs when Dopram is given makes it easier to see whether there is adequate abduction.
If all of the above structures appear within normal limits and no obvious cause for the respiratory obstruction can be identified, then endoscopy of the trachea and bronchi is indicated. This is usually done with the animal fully anaesthetised, but not intubated. Care must be taken that the scope is of small enough diameter so as not to obstruct oxygen flow to the lungs. Oxygen can be administered via the biopsy portal of the scope if insufficient oxygen saturation is being achieved.

MEDICAL MANAGEMENT OF BRACHYCEPHALIC SYNDROME

The detailed medical management of this condition is outside the scope of this article, however it is an integral part of treatment. Affected dogs frequently require lifelong treatment with proton pump inhibitors and mucolytics. Corticosteroids, bronchodilators and antitussives are often used in the short term, emergency treatment to decrease airway inflammation and mucous production.

Gastro-oesophageal reflux is a common concurrent finding in patients with respiratory obstruction. Stenosis of the airway causes severe inspiratory pressures, which predispose the patient to reflux. During visual examination of the upper airway, inflammation and ulceration of the arytenoids cartilages is often seen and concurrent treatment of these patients with a proton pump inhibitor like Omeprazole (1mg/kg once daily) is often indicated lifelong.

Further to medications, owners need to be well coached on the benefits of weight control, exercise moderation, and preventing affected dogs from over excitement and hyperthermia.

SURGICAL REPAIR OF BRACHYCEPHALIC SYNDROME

Stenotic nares are found frequently in brachycephalic breeds. Although a minor component of the syndrome, having insufficient external passage for air causes animals to open mouth breathe and increase respiratory effort through rest of airway. The surgical repair is not technically demanding, it consists of performing bilateral wedge naroplasties. It is important that these wedges are sufficiently deep to open the nares at least 1cm into the nasal passages. Fine absorbable suture material is used to close the defects and haemorrhage is usually easily controlled.

The most common and invariably significant obstruction to airflow stems from an elongated (and often substantially thickened) soft palate. Surgical resection of the elongated soft palate (staphylectomy) is performed via an oral approach and aims to remove excessive tissue so that the obstruction to the

dorsal glottis is removed. The excessive tissue is clamped and sharply resected. A monofilament absorbable suture material in a continuous pattern is used to close the defect. Intraoperative haemorrhage and oedema, as well as the post operative risks of dog developing obstruction from the surgery, are good reasons that only surgeons experienced with the technique should attempt it. More advanced techniques for resection using laser excision has been performed successfully, however the equipment needed is expensive and not readily available.

Further upper airway obstruction can be caused by eversion of the laryngeal ventricles into the opening of the glottis. These ventricles are mucosal diverticulae that are situated rostal and lateral to the vocal folds. When excessive negative pressure is experienced within the upper airway, these ventricles tend to evert medially and become extremely oedematous. They then block the lower half of the airway at the laryngeal glottis. Many animals have a combination of the above conditions. When an elongated soft palate obstructs the dorsal half of the glottis and the everted laryngeal ventricles, the lower half, it can lead to as much as 80 to 90% obstruction of the glottis. Surgical correction is straightforward resection of the saccules. The only difficultly is that visualisation is obstructed due to the caudal position of the saccules and endotracheal tube being in the way.

Laryngeal paralysis is not considered a component of Brachycephalic Syndrome, however it can present within the same breeds. It can either be an idiopathic or congenital condition or can be caused by damage to the recurrent laryngeal or caudal laryngeal nerve. The recurrent laryngeal nerve runs rostrally along the outside of the lateral trachea and innervates the intrinsic muscles of the larynx. Its function is to cause abduction of the vocal cords and arytenoid cartilages during inspiration. Damage to the nerve is generally an irreversible condition and requires surgery to permanently abduct the arytenoids cartilages on one or both sides. Various techniques are available for this procedure, but the most commonly used technique is the cricoarytenoid laryngoplasty or lateralisation.

Tracheal hypoplasia is common in English Bulldogs, alone or in combination with the other components of BS. The trachea is considered hypoplastic when the ratio of tracheal diameter to thoracic inlet diameter is less than 0.2. It can cause severe clinical signs of very young patients and treatment is largely symptomatic. Repair of the other obstructive issues is performed in the hope that the dog will then cope with their narrow tracheal lumen.

POSTOPERATIVE CARE OF UPPER AIRWAY PATIENTS

Continuous observation of patients that have undergone upper airway surgery is vital for the first 48-72 hours. Extubation should be delayed for as long as possible and animals should be kept in a cool, quiet environment. At all times, the surgeon involved should be prepared to perform an emergency tracheostomy, if acute swelling and respiratory distress develops. Intravenous fluid administration should be continued at maintenance doses and Metoclopramide can be added to these fluids to help prevent vomition during the recovery period. Corticosteroids can be used at anti-inflammatory doses (0.5-2mg/kg), but are usually administered at least 2-4 hours prior to surgery for beneficial effects to be evident. It advisable to have these patients on a proton pump inhibitor like omeprazole at 1mg/kg OID for at least 2 weeks post surgery. In many cases this drug is continued lifelong to decrease the chances of acid reflux. Acid reflux is a known cause of cartilage degeneration and collapse.

Water can be offered to these patients once they have fully regained swallowing function, but food is often withheld for 24 hours as aspiration pneumonia and irritation of the surgical site causing vomition are valid concerns.

PROGNOSIS FOR PATIENTS WITH UAOS

The prognosis in these cases is obviously dependant on the initial cause, but most animals do poorly without surgical intervention. Respiratory distress tends to result in a progressive cycle of mucosal swelling, cartilage fatigue and increasing collapse of the airway. Younger animals tend to do better with surgery than older animals and this is mainly due to the absence of cartilage collapse of the larynx and trachea that occurs with time. In cases of severe laryngeal or tracheal collapse, the prognosis is generally guarded. Owners that follow the advised weight reduction programs and ensure optimal body condition score are likely to have a much higher degree of success.

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HOW TO PERFORM AN EMERGENCY TRACHEOSTOMY

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INTRODUCTION

A tracheostomy is usually performed to allow air to enter the trachea distal to an upper airway obstruction, such as trauma, anaphylaxis, neoplasia and conformational defects.[1,2] In addition, this techniques has proved useful for planned airway management during surgical manipulation of the oral cavity. A tracheostomy is either temporary, when a tube is inserted, or permanent, when a stoma is created. A temporary tracheostomy is most commonly performed to provide an alternate airflow route. It may be performed either during surgery for relief of an upper airway obstruction or as an emergency procedure in a severely dyspnoeic patient requiring medical treatment.[3] Temporary tracheostomies are usually maintained only for a short period of time.

OPERATIVE TECHNIQUE

Patient positioning and preparation

The patient should be positioned in dorsal recumbency, with some support under the neck, and the forelimbs secured on either side of the thorax. The ventral neck should be clipped from the manubrium sterni to the caudal angle of the jaw and aseptically prepared for surgery.

Additional instruments

Because tracheostomy tube placement may need to be performed in an emergency, supplies for the procedure should be readily available. Ideally a surgical pack dedicated to the procedure should be kept in the emergency treatment area. Supplies should include tracheostomy tubes of the appropriate sizes (no larger than 75% of the diameter of the trachea). Two or three different sizes should be readily available. For routine airway maintenance, a non-cuffed tube, with an inner cannula should be used. For maintenance of anaesthesia or prolonged mechanical ventilation, a tube with an inner cannula and a high volume, low-pressure cuff is more appropriate. These tubes should only have the cuff gently inflated. The cuff should be deflated and repositioned every 2 hours to minimize pressure necrosis of the tracheal mucousa.[1,4] Other supplies should include sterile surgical instruments, large monofilament suture material for stay sutures, and umbilical tape to secure the tube around the neck following its placement.

Surgical technique

The larynx and the trachea should be palpated as the anatomical landmarks. An incision of approximately 7cm should be made over the trachea running caudal from the larynx. The sternohyoideus muscles are separated at the midline and pulled laterally. Gelpi or self-retaining retractors help with the exposure of the ventral trachea. Stay sutures are placed around the tracheal rings just cranial and caudal to the proposed annular ligament incision. They should be clearly labelled and will later allow for stabilization and opening of the trachea when changing the tracheostomy tube. An incision is made in one of the annular ligaments between the third and fifth tracheal rings, in a horizontal or transverse fashion. The incision of the annular ligament should not exceed 50% of the diameter of the trachea. The tracheostomy tube is then placed and secured by tying umbilical tape around each side of the tube flange and then tying the two ends around the animal's neck. The tube size should be large enough to adequate airflow, but small enough to provide room for airflow around the tube.[3] This helps to prevent silent death from occlusion of the tube lumen.[2] The skin and subcutaneous tissue should only be partially closed on each side of the incision.

POST OPERATIVE AND TRACHEOSTOMY TUBE CARE

After a tracheostomy has been performed, intensive management and 24-hour observation of the patient are essential. A ready assortment of supplies for tube management should be kept near the

patient at all times.[5] If possible, a nurse should be dedicated to the patient to ensure that subtle changes in respiration are noted because deterioration can occur rapidly.[2,3]

The tracheostomy tube bypasses the normal warming and humidification mechanisms and creates airway inflammation in addition to the inflammation associated with its placement. Dedicated care is essential to prevent potentially fatal occlusion of the tube by exudates and airway mucous.[1]

The inner cannula cleaning should be removed for cleaning whenever an increased noise or effort associated with breathing is noticed, or every 2 hours initially. The cannula should be cleaned thoroughly using warm water, dried and replaced.[1] If the inner cannula is repeatedly full of mucous and exudate, nebulized air should be provided for the animals to breathe. Alternatively 0.1ml/kg sterile saline can be installed into the tube every 2 hours. This may, however, induce a transient cough.[3]

Suction is another valuable technique for the management of temporary tracheostomy tubes. It is however not a benign procedure and should be done sparingly and with great care. Repeated suctioning of the airway can exacerbate airway inflammation or may occlude the airways during the procedure. Suction is most commonly needed in smaller cats and dogs. The patient should be pre-oxygenated for at least 10 breaths prior to suctioning. The catheter should be introduced aseptically into the tube and suction applied for no longer than 10-12 seconds while gently rotating the suction tube.[5] Uninterrupted suctioning can lead to atelectasis and hypoxia.[3] Suction should ideally be performed at least four times per day. Some coughing, retching or gagging can be expected during the procedure. Ideally a sterile catheter should be used each time.

Coupage and multiple controlled periods of exercise on a harness can be used to mobilize secretions. Antibiotics are typically not continued beyond the operative period. The tracheostomy wound should be inspected daily and cleaned as necessary.

If the above measures do not relieve breathing difficulty, the whole tube should be replaced. Some literature suggests replacing tubes at a minimum of twice daily. This should not be done in the absence of a clinician. The patient is pre-oxygenated and the trachea stabilized using the stay sutures, applying gentle traction away from the wound. The old tube is removed and a new one rapidly inserted. Tubes should be cleaned and soaked in 2% chlorhexidine and rinsed with sterile saline before replacement.[1]

TUBE REMOVAL

The decision to remove the tube coincides with resolution of the inciting cause. Direct observation following tube removal is essential to ensure that respiratory distress does not occur. Tracheostomy wounds heal by second intention within 7-10 days.[6,7] During this time, owners are instructed to clean the stoma at least twice daily.

COMPLICATIONS

Immediate complications occur in approximately 50% of patients and include plugging of the tube with mucous and debris, inadvertent tube removal, gagging, coughing, vomiting, subcutaneous emphysema, pneumomediastinum, pneumothorax, infection and respiratory distress.[3,8] The most common complication is plugging of the tube. Cats appear to have a higher complication rate and this is thought to be due to more mucous secretions in this species and an increased risk of tube occlusion.[3]

The most significant long-term complication of tracheostomy is stenosis, which may occur at the tracheostomy site itself or in the area where the cuff of the tube was inflated.[2]

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BASICS OF EXTERNAL SKELETAL FIXATION

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ABSTRACT

External skeletal fixation is one of the most versatile tools available to the veterinary surgeon when dealing with orthopaedic problems. Mostly fractures are treated but these systems are very useful when treating abnormalities of skeletal development and ligamentous injuries. They are often used to augment surgical repairs of difficult fractures or when treating complications of some standard repair methods. This presentation will give an overview of the basic principles used when applying these techniques and also some practical guidelines that can be used in everyday practice. If one ignores the basic rules then failure can be expected therefore very important to understand the application guidelines.

INTRODUCTION

The aim of this presentation is a basic overview of the principles of external skeletal fixation and then also the practical application of a versatile easy to use system in your practice.

Three types of external fixation systems exist. Linear fixation uses straight connecting bars when connecting the bone pins. Circular systems use full rings or half rings to connect a series of pins or wires that are seated in the bone. Free flow systems use moulded connecting bars which allows a lot of freedom when connecting pins.

Three different pin types can be used namely smooth, positive threaded or negative threaded pins. Threaded pins can be end-threaded or centre-threaded depending on their intended use. Negative profile pins have the same diameter thread as the smooth shank of the pin. Positive profile pins have a wider diameter at the threaded portion than at the shank of the pin.

Circular systems will not be discussed further as they are mainly used for angular limb corrections and limb lengthening procedures.

Linear systems use metal or graphite connecting rods which are secured to the bone pins using a clamp. Various different systems also exist in this category with its own advantages and disadvantages.

Free flow systems use silicon or plastic tubing placed over the bone pins and then injected with a thermoplastic agent like poly methyl methacrylate or another type of epoxy cement. Epoxy putty can also be moulded without using tubing material.

FRAME CONFIGURATIONS

Different frame configurations can be used depending on the type of fracture being treated. In order of increasing strength we can use Unilateral, Unilateral biplanar, Bilateral and Bilateral biplanar frames.

Pin fixation clamps should be placed on the inside of the frame to shorten the pin length which will increase the pin stiffness. Three to four pins should be placed per segment of bone with a total of 6-8 pins usually. Smooth pins should be angled (35 degrees) to decrease pin loosening which makes it more difficult to place more pins. Threaded pins can be placed perpendicular to the bone. Pins should span the whole length of the bone but not closer than 3 times the diameter of the pin or half the diameter of the bone to the fracture ends or the joints. Larger pins are also stiffer but should not be larger than 20-30 % the diameter of the bone. Placing a second connecting bar on unilateral configurations will double the strength and stiffness of the frame.

PRINCIPLES OF PLACING AN EXTERNAL SKELETAL FIXATOR

The pin-bone interface is the most important part of any fixator. Pin loosening should not occur before bony union. Threaded pins have better bone holding properties. Positive threaded pins are stronger than negative because the thread diameter is wider than the shank. Negative profile pins are weak at the thread-shank junction. The drilling of pins into the bone creates heat and structural damage. Drill speed should be 300 rpm or less. Power operated drills give better control as it increases hand stability. Pre-drilling holes with a slightly smaller diameter drill bit will decrease damage to the pin-bone interface. Pins should not be “backed-out” but inserted to the correct depth the first time. Crossing the widest diameter of the bone will also increase strength of the frame. Aseptic surgical principles should be used when repairing the fracture. When using the free flow system, the surgical site should be closed before working with the tubing and epoxy cement. A useful tip is to use trans-cutaneous “skewer” pins to hold the fracture reduction, then to close the wound and after application of the connecting bars, the skewer pins can be removed. Frames should not interfere with joint movement or the body wall. Safe corridors should be used when placing pins to avoid large muscle bellies and neuro-vascular bundles. The surgical approach should be as limited as possible to preserve blood supply and enhance bone healing. Pins should not be inserted through the same incision as the surgical approach but through separate small stab incisions. Blunt tunnels are made through the soft tissue and a drill sleeve is used if possible.

Pins should be bent at the level of the skin to facilitate the placement of the tubing. The tube is impaled over the pin ends and then lowered to approximately 1 cm from the skin. If a large amount of swelling is anticipated post operatively then this distance can be increased. After injecting the epoxy cement, the sharp pin ends are cut at the level of the connecting bar and the bar is bandaged to avoid laceration of other body parts from sharp cut pin ends.

The connecting bar determines the strength and stiffness of the fixator. A 19 mm diameter epoxy column is stronger than a 4.8 mm stainless steel connecting bar.

POST-OPERATIVE CARE

Owners are told to clean the pin-skin interface daily with damp cotton wool and to observe for any seepage or changes of the frame or pins. If they notice any foul smelling discharge or non-clear seepage then they must seek advice from their vet. Strict confinement in nothing more than an average bedroom sized area with no running or jumping and only a few short lead walks daily is prescribed for 4-8 weeks. Acrylic bars can crack and break so the owner must inspect the integrity of the bar daily. Radiographic follow up at 4 weeks and then every 2-3 weeks. Most fractures should heal before 12 weeks. The fixator is removed when evidence of bony union is found. From 5-6 weeks some of the pins or bars can be removed to “dynamize” the fracture and speed up healing. No benefit is achieved after 12 weeks.

POSSIBLE COMPLICATIONS

Pin tract seepage is commonly seen and usually no cause for concern.

Premature pin loosening is a serious complication and usually require pin removal and addition of other pins or methods to maintain stability of the fracture site. This can usually be seen on a radiograph as a radio-lucent “halo” around the pin.

Pin tract infection is an uncommon finding but a serious complication requiring bacterial culture and antibiotics and will often flare up after medication is finished and sometime only resolve after implant removal.

Patient interference is rarely encountered but can hamper the healing process and one should be aware of possible complications with certain breeds and individuals. Owner compliance is of paramount importance when utilizing an external skeletal fixation device.

OTHER USES

Most common uses are for long bone fractures where internal fixation is not available or indicated.

Fractures of the femur and humerus often require modification of frame type to a hybrid configuration where the distal pin or pins can be placed as full pins from lateral to medial. This can then be connected to the lateral connecting bar with a diagonal bar to increase frame strength. Fractures distal to the elbow and stifle can easily be treated by unilateral and bilateral configurations.

An external fixator is very useful when requiring additional stabilization for a comminuted fracture for instance where a plate could not utilize enough screw holes or when an intra-medullary pin is used but rotation has to be counteracted. Often these pins can also be incorporated into the frame and is then called a tie-in configuration.

External fixators are commonly used where access is needed to the skin where large wounds are present and need to be treated. These are much more difficult to monitor and treat under a bandage. If needed bandages can be placed over frames in the initial period to maintain sterility and keep wounds moist.

Septic or sterile, non-unions can also be treated by using external fixation techniques. This allows the surgeon to easily remove implants once union has been achieved.

Temporary stabilization of joints or regions where ligament injuries have been repaired like Achilles tendon ruptures or collateral ligament damage can easily be achieved by external skeletal fixation. This allows time for the repaired structures to gain in strength before being tested by the patient.

These versatile systems have also been used in metacarpal and metatarsal fractures. Dr Fitzpatrick described the “CLAW” for these injuries where intra-medullary pins are incorporated into epoxy putty with trans-carpal or trans-tarsal pins.

Mandibular fractures that are multiple or comminuted can be treated with external fixation.

Often these systems are used to stabilize limbs where previous surgery ie TPLO has had catastrophic failure or with failed tibial tuberosity transpositions. In these cases the fixator is used trans-articularly to allow healing of the tibial tuberosity.

DISADVANTAGES OF ACRYLIC FRAMES

- Fumes of the curing process are noxious and toxic.
- Heat generation during curing can add to damage to the pin-bone interface.
- If holes made in tubing are too big, the acrylic will leak out during application.
- Difficult to maintain reduction during curing process.
- Column can break if not strong enough or when air bubbles were introduced during application.
- Adjustment and removal of individual pins post operatively is difficult.

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FORELIMB JOINT DISEASE IN THE DOG

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ABSTRACT

The majority of synovial joints in the dog are subjected the significant stress during weight bearing. This requires a joint, which is highly resistant to wear and tear and one that allows almost frictionless movement. There are numerous conditions, which affect the joints of the canine forelimb. Early diagnosis and treatment is crucial to effective treatment and therefore improved prognosis. Arthroscopy allows minimally invasive surgery of joints and has significant advantages over arthrotomy. These advantages include rapid recovery and reduced morbidity, ability to treat multiple limbs simultaneously, improved visualisation of intra-articular structures. Shoulder lameness and elbow dysplasia are two conditions were arthroscopy has allowed improved early diagnosis and treatment. Many conditions causing shoulder lameness present with similar symptoms and radiographic signs, arthroscopy therefore allows accurate diagnosis via a minimally invasive modality. All joint disease has the potential to result in the development of osteoarthrosis. Osteoarthrosis is a chronic progressive degenerative joint disease. Treatment includes long-term conservative management and in some cases surgery (joint replacement, arthrodesis, arthroplasty). Common causes of shoulder lameness in dogs includes OCD of the caudal humeral head, tenosynovitis of the biceps tendon, medial shoulder instability, incomplete ossification of the caudal glenoid rim and mineralization in the tendon of insertion of the supraspinatus muscle. Diagnosis of these conditions relies on careful clinical examination, radiology, ultrasound and importantly arthroscopic examination. Elbow dysplasia is a common cause of forelimb lameness in young large breed dogs. Diagnosis can be challenging and again arthroscopy can play and important role in early diagnosis and treatment. Types of Elbow dysplasia include Fragmented Medial Coronoid (FMCP), OCD of the medial humeral condyle, Ununited anconeal process (UAP) and elbow incongruency.

INTRODUCTION

Joints provide stability to the body during weight bearing and propulsion. Joints are subjected to significant forces during normal function and this leads to wear and tear. Joints must therefore be resistant to wear and allow frictionless movement. The anatomical structure of the joint is critical to achieving this function. Any injury and damage to the joint will compromise its ability to function normally and lead to the development of osteoarthrosis. Arthroscopy allows minimally invasive examination and treatment of joint pathology.

ANATOMY

Synovial joints are major joints of the appendicular skeleton. These joints allow significant movement and are weight bearing joints. All synovial joints consist of a joint space, joint capsule, joint fluid, articular cartilage, subchondral bone and some joints may have intra-articular structures e.g. ligaments or menisci. The articular cartilage is composed of a cellular component (chondrocytes) and an extracellular matrix (glycosaminoglycan's, proteoglycans and collagen). Synovial fluid (joint fluid) is secreted into the joint by synovial cells of the joint capsule. Synovial fluid's main functions are lubrication of the joint and nutrition of the articular cartilage (chondrocytes). Articular cartilage is an avascular structure. The extracellular matrix consists of collagen fibres (type II), proteoglycans and water. The proteoglycans are made up of subunits of glycosaminoglycan's (chondroitin sulphates, keratin sulphates) linked to a central protein core. The proteoglycans are hydrophilic i.e. attract water molecules while the collagen gives the cartilage its tensile strength.

RESPONSE TO INJURY

Aging results in changes to extracellular matrix. There is a decrease in the total collagen content as well as a change in the types of collagen (↓ type II; ↑ type I and III). The ratio of glycosaminoglycan's changes (↓ chondroitin sulphate; ↑ keratin sulphate) and there is a decrease in the total amount of proteoglycans. These changes in turn results in a decreased tensile strength and a decrease in the

water content. This translates into a decreased ability to function normally and an increased susceptibility to injury.

When articular cartilage in subjected to an injury and inflammatory cascade is initiated within the joint and inflammatory mediators are released. This inflammatory process will result in increased levels of proteinases (e.g. metalloproteinases) within the joint. These proteinases accelerate degradation of the extracellular matrix. The in turn results in further decreased ability of the joint to function normally. The end result of this process is progressive irreversible osteoarthrosis

ARTHROSCOPY AND FORELIMB JOINT DISEASE

The recent development of arthroscopy in small animal veterinary surgery has improved our ability to diagnosis and treat canine joint disease. Arthroscopy offers multiple advantages over conventional arthrotomy when it comes to the diagnostic and surgical treatment of joint disease.

Advantages of arthroscopy include:

- Shortened recovery time / decreased hospitalization
- Minimally invasive surgery
- Reduced post operative pain
- Allows second look procedures
- Earlier return to function and therefore less loss of range of motion
- No /minimal post operative wound care and bandages
- Visualise joint under magnification and in its normal fluid environment
- Much lower risk of post op complications (<0.56% complication rate)
- Allows documentation of pathology (photographs & video)
- Good cosmetic results.

Disadvantages of arthroscopy:

- Expensive and fragile equipment
- Steep learning curve initially
- Specialized sterilization of some equipment.

The major complications that can be encountered with arthroscopy include extravasation of fluid into the periarticular tissues, iatrogenic damage to the articular cartilage with the scope and instruments and intra-articular haemorrhage. None of these complications are serious. Nerve injury has been described but is very rare. The risk of iatrogenic cartilage injury and nerve damage decreases significantly with improved technique.

Equipment required for arthroscopy:

- Arthroscope
- Egress cannula
- Arthroscopy sheath
- Light source
- Arthroscopy camera
- Printer / video recorder
- Hand instruments
- Radiofrequency unit
- Shaver.

Because of its minimally invasive nature and subsequent advantages arthroscopy offers an ideal modality for both diagnosis and treatment of numerous joint diseases of the canine forelimb.

SHOULDER LAMENESS

There are numerous conditions of the canine shoulder joint that may result in lameness. These include osteochondritis dissecans of the humeral head, bicipital tenosynovitis, medial shoulder instability, supraspinatus insertion strain / calcification and incomplete ossification of the caudal glenoid rim. Most of these conditions show very similar clinical symptoms and similar radiographic

findings. Arthroscopy is therefore an ideal modality for the diagnosis of these conditions as well as the treatment of most of them.

Osteochondritis dissecans of the humeral head
OCD results from osteochondrosis and is the development of a flap of cartilage from the caudal aspect of the humeral head. The flap may remain partially attached or may become completely detached. The condition typically affects young large breed dogs between the ages of 4 – 10 months. Radiography is usually diagnostic. There is typically a flattening of the caudal humeral head.

Surgical treatment involves the removal of the cartilage flap and any necrotic subchondral bone. Arthroscopy has the advantage of allowing visualization of the entire joint and the ability to treat both shoulders simultaneously if required. Post-operative management involves restricted activity for 4 – 6 weeks and NSAIDs. The long-term prognosis for shoulder OCD is good to excellent despite the fact that there will be some osteoarthrosis.

Bicipital tenosynovitis
Bicipital tenosynovitis is an inflammatory condition of the biceps tendon, which originates on the supraglenoid tubercle of the scapula. It may also involve a partial tear / rupture of the origin of the biceps tendon. Clinical signs are usually and chronic weight bearing lameness. Pain may be detected on direct palpation over the biceps tendon or on shoulder flexion/extension. The cause of the injury may be chronic repetitive trauma or an acute injury. It is most commonly seen in adult medium to large breed dogs. Diagnostic tests include radiography, ultrasound, positive contrast radiography and arthroscopy. Radiology may reveal mineralization in the proximal tendon adjacent to the supraglenoid tubercle. Arthroscopy allows accurate diagnosis and then treatment. Treatment of choice for bicipital tenosynovitis or partial rupture is to release the tendon from its attachment on the scapula. Postoperative management involves restricted activity for 4 weeks and NSAIDs as required for analgesia.

Incomplete ossification of the caudal glenoid rim
Incomplete ossification of the caudal glenoid rim occurs mainly in medium to large breed dogs and may be an incidental finding. It is however a cause of lameness in certain cases. Clinically it manifests as a chronic weight bearing lameness with pain on shoulder flexion and extension. Arthroscopy provides the ideal modality for ruling out other potential causes of lameness (bicipital tenosynovitis, medial shoulder instability etc) before diagnosing incomplete ossification as the cause of the lameness. Treatment involves removal of the osteochondral fragment. Long-term prognosis is good to excellent.

Medial shoulder instability
Medial shoulder instability is the result of injury to the medial glenohumeral ligaments, medial joint capsule and or the tendon of the subscapularis muscle. These structures may be stretched, partially torn or completely ruptured. Clinical signs are similar to the other conditions discussed – chronic weight bearing lameness. There may be atrophy of the shoulder muscles and increased abduction of the humerus may be palpable. Radiographic findings are unremarkable and confirmation of the diagnosis requires arthroscopy. This condition may be treated conservatively (rest) although this is seldom successful. Surgical treatment involves reconstruction / imbrications of the medial ligaments and joint capsule or thermal ablation with a radiofrequency probe during arthroscopy. Prognosis is dependent on the degree of laxity and whether there has been damage to the labrum of the glenoid.

Calcification of the supraspinatus insertion
Calcification within the tendon of insertion of the supraspinatus tendon may be a cause of shoulder lameness although it is often seen as an incidental finding on radiographs. Clinical symptoms are typically a chronic weight bearing lameness, worse after exercise. It is most common in middle aged large breed dogs. Because it is often an incidental finding arthroscopy is an ideal modality to rule in / out other causes of shoulder lameness (e.g. medial shoulder instability, bicipital tenosynovitis etc) Treatment options include conservative treatment with rest and NSAIDs or surgical excision of the calcification. Tenectomy of the insertion of the supraspinatus may also be performed to treat this condition.

ELBOW DYSPLASIA

Elbow dysplasia is a term to describe a number of conditions that affect the elbow joint typically in young large breed dogs. The major conditions of elbow dysplasia include OCD of the medial humeral condyle, fragmented medial coronoid process of the ulna (FMCP), ununited anconeal process (UAP) and elbow incongruency. With the exception of UAP these conditions can be challenging to diagnose in their early stages. Early diagnosis and prompt treatment gives the best long-term prognosis.

Radiography may indicate a degree of suspicion in young dogs but is seldom diagnostic for FMCP and OCD of the medial humeral condyle. Arthroscopy allows detailed examination of the medial compartment of the joint, accurate diagnosis as well as treatment in these cases. Treatment for OCD cases involves removal of the cartilage flap and necrotic bone. In cases of FMCP the lose osteochondral fragment is removed and the necrotic subchondral bone is curetted.

Arthroscopy has significant advantages in the treatment of elbow dysplasia:

- Early diagnosis and treatment prior to the onset of significant arthrosis
- Assessment of incongruency
- Bilateral treatment simultaneously
- Reduced morbidity and quicker recovery
- No bandages post-operatively

All cases of UAP should also be assessed arthroscopically for concurrent FMCP and to determine whether a fibrous union exists between the ulna and the anconeal process.

Other indications for arthroscopy of forelimb joints include septic arthritis, intra-articular fracture reduction, assessment of ligamentous injuries and carpal instability.

Arthroscopy will become more widely available as surgeons become more familiar with the technique. Indications will also continue to grow especially and scopes become smaller and therefore arthroscopy of smaller joints and smaller patients becomes a reality.

OSTEOARTHROSIS

Osteoarthrosis (OA) is a common debilitating disease seen in both canines and felines. It is a common consequence of previous joint disease or injury. Osteoarthrosis is a chronic progressive irreversible degenerative joint disease. Because of its progressive nature OA can have a significant negative impact on a patient's quality of life.

Long-term management of OA requires a multifaceted approach. Surgery may be a component of the treatment but medical management, diet, weight management, chrondroprotectants, etc are the cornerstone of the treatment of osteoarthrosis in small animals.

Conservative treatment of OA

This would include:

- Weight reduction
- Exercise modulation
- Nutrition (e.g. Hills J/D)
- Lifestyle modification (esp. cats)
- Analgesics (NSAIDs, opioids)
- Rehabilitation / Physical therapy
- Chondroprotectants

Surgery for OA

Possibilities would be:

- Stabilization of ligamentous injuries and intra-articular fractures
- Arthrodesis
- Arthroplasty (e.g. femur head and neck excision)
- Joint replacement

The major causes of OA in dogs include hip dysplasia, elbow dysplasia and cranial cruciate ligament disease. Clinical symptoms of OA include lameness, difficulty rising from a sitting or lying position, difficulty jumping and going up stairs and pain. Clinical symptoms in cats differ from those seen in dogs. The major clinical signs in cats include lifestyle and behavioural changes rather than lameness. Lameness is seldom seen as a presenting symptom in cats with OA.

APPROACH TO HINDLIMB LAMENESS

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ABSTRACT

The aim of this talk is to provide the general practitioner with a systematic guide to the approach to canine lameness in general practice. Identification of the clinically significant anatomical region eases diagnosis of the underlying disease process. Diagnostic aids and basic tests will also be discussed. Careful history taking from the owner is the first step to finding the source of the lameness. Video footage taken by owners is a very useful tool, as dogs often do not show the severity of the lameness or the lameness at all when in a clinic/hospital environment. Physical examination must be performed methodically and must always be the same to have a routine the clinician can rely on. If these first steps are done in a thorough manner then finding the source of the lameness should be a lot easier. Now one can start using diagnostic tools to our disposal like radiography, joint cytology, magnetic resonance imaging, computed tomography, ultrasonography etc. Without finding the source and cause of the lameness one will not be able to treat the condition with reasonable success.

INTRODUCTION

When confronted with a case of lameness in a dog, it is very easy to make presumptive diagnoses based on observation and prevalence of conditions, especially when breed and age considerations is overemphasized. It is therefore very important to always use the same approach and diagnostic steps in order to not misdiagnose or overlook other important clinical findings. The following subcategories help to systematically evaluate these patients:

- History
- General examination
- Orthopaedic examination
- Neurological examination
- Diagnostic imaging
- Joint cytology
- Other modalities eg: Arthroscopy, Ultrasonography, CT, MRI, Scintigraphy

HISTORY

Observe the patient from the waiting room to the consultation room. This will reveal clues as to severity of lameness, limb affected, difficulty rising etc. It is also important to observe the animal while taking down the history from the owner. Owners often confuse the affected limb or are unsure when questioned in detail. The history should start with general questions pertaining to vaccination status, general health, previous illness or intervention. Then only focus on the lameness presented. Owners usually want to start discussing the problem at hand and can omit important clinical information relating to the problem if not questioned appropriately. Always obtain information regarding the degree of pain and lameness, duration, rate of progression or improvement if any, previous trauma and also the effect of rest, exercise, weather and time of day on the lameness. Urge clients to bring video footage of especially long standing cases and also to keep a diary of severity and frequency of the problem.

GENERAL EXAMINATION

Temperature, pulse and respiration should be taken as for any clinical case. Body condition score is extremely important in all orthopaedic cases. Chest auscultation and abdominal palpation to look for concurrent or related abnormalities. This is more important in trauma cases where multiple injuries are usually present.

The patient's lifestyle must always be considered. The athletic demand on an animal can vary greatly depending on the expected mobility of the animal.

ORTHOPAEDIC EXAMINATION

The initial history and observation should form a strong link with the findings of the orthopaedic examination. The first observation should consider the conformation of the patient. Animals with very upright hocks often suffer from stifle conditions for example. Overdeveloped forequarters could focus the clinician's attention to a bilateral hindquarter problem. Observing the way the animal sit is also of importance, because animals with painful stifle joints often sit with the affected limb to the side of the body to increase the distance from the tuber ischium to the calcaneus to relieve pain. Weight bearing when standing can alert us to the affected limb or shifting continuously can also indicate multiple limb involvement. Muscle atrophy and circumference of certain muscle groups should be evaluated next. A normal tape measure can be used to identify atrophy and also to monitor progression. The most common muscle group measured is usually the thigh muscles. It is important to measure the same level on both sides.

Next we observe the animal at a walk and then at a trot. This should ideally be performed outside of the hospital away from the owner on a level non-slippery surface. The aim is to identify the lame limb and also attempt to identify the affected region by observing the specific gait characteristics. With time a clinician can develop the skill to identify upper limb lameness versus lower limb lameness.

Physical palpation of the patient is the most important step of the entire diagnostic workup. This should be performed systematically and each clinician will develop a method that works personally.

- Always palpate the suspected affected limb last
- Get a feel for the animal's response to palpation by starting with a clinically sound limb
- Always start at the digits and palpate proximally
- Each individual joint and bone should be palpated for pain, swelling or crepitus
- Joints should be evaluated for instability that could be caused by ligamentous injury. Therefore it is important to have some knowledge of normal range of motions for certain joints.
- Palpation of muscles and groups for swelling, atrophy or pain. Certain muscle groups are prone to fibrous contracture
- Placing pressure with one's fingers on individual long bones is important especially in young large breed dogs to evaluate for possible panosteitis. This usually found near the nutrient foramen of each long bone.

NEUROLOGICAL EXAMINATION

A detailed description of the neurological examination is not within the scope of this paper. The aim is to provide a basic guideline to identify neurological abnormalities and differentiate them from orthopaedic conditions. This can easily be missed clinically and should always be ruled out when dealing with an orthopaedic condition.

Observing the gait of the animal is again the most important step. This will identify most neurological cases and then a more detailed neurological investigation can be performed.
Testing the animal's proprioception and placing reflexes must always be performed. Wheelbarrow tests and hemi walking should be focused on if suspecting a neurological abnormality. Limb reflexes can often be the only abnormality found but these should be repeatable.

Neck manipulation for flexibility, muscle spasm and pain response is vital. The thoraco-lumbar spine must be evaluated by individually putting pressure on each dorsal spinous process whilst keeping the other hand on the abdomen of the animal to feel for tensing of the abdominal muscles. The lumbo-sacral junction is tested for pain by standing behind the patient, lifting the hind limbs in a flexed position off the ground and putting pressure on the pelvis dorsally with the other hand.
Cranial nerves and peripheral sensation tests complete the neurological examination.

DIAGNOSTIC IMAGING

Radiographic examination of the affected area is the most powerful tool at the disposal of the clinician. Comparative views should be taken between affected and sound limb. More than one projection is important. Good collimation and use of slow cassettes to improve detail of images can all help to reach a diagnosis. Stress radiographic views are usually used when suspecting ligamentous

damage of joint displacement. Contrast studies can be employed in certain cases. Stress radiography must be performed when suspected joint laxity or dynamic pathology.

JOINT CYTOLOGY

Synovial fluid aspiration should be attempted if this could help in finding a definitive diagnosis. Smears are made by placing a drop of joint fluid in the middle of a microscope slide and smearing it with another slide placed flat on the first slide. Air drying must then be performed before fixing and staining using Diff Quick. Always look at the smears before sending to a cytologist. The most important thing rule in or out, is the presence of a large number of neutrophils. This usually indicates either a septic joint condition or immune mediated arthropathy. If in doubt, it is best to send a sample for bacterial culture and the smears to a cytologist for interpretation. Joint fluid should ideally be placed in blood culture bottles when sent to the laboratory for culture.

ARTHROSCOPY

This is the gold standard when evaluating intra-articular structures and articular cartilage. Studies have shown that one can often have a poor correlation between clinical and radiographic findings when compared to arthroscopy. This modality helps to reach an early diagnosis; it is minimally invasive and usually results in a fast recovery from the procedure. The negative aspects are costs involved and the need for general anaesthesia.

MRI AND CT

These modalities are becoming more available and used more frequently. The can provide information not available with plain film radiography. CT very helpful for fracture and angular limb correction planning. Also used for elbow investigations and to find bony pathology as soon as possible. MRI is used more and more in human medicine for evaluating joint conditions. Veterinary literature is starting to appear on this subject.

SCINTIGRAPHY

Usually employed when we deal with an undetermined source of lameness or pain. This will identify a site of inflammation and direct the clinician to the area of interest.

LOCAL ANAESTHETIC BLOCKS

This is not used routinely but can certainly be used if indicated. Can help to differentiate a suspected spinal condition from a joint problem. The effect on a specific lameness can be difficult to predict as not a lot of data is available on the subject.

FORCE PLATE ANALYSIS

The gold standard for evaluating limb function but correlates poorly with clinical observation and not widely available.

EXAMINATION OF SPECIFIC REGIONS

Distal limb

Examine the nails for any sign of trauma and especially the nailbed for swelling or thickening. The footpads and interdigital skin are often neglected and dogs with interdigital dermatitis can have significant lameness, which can be difficult to treat. Each phalangeal joint and phalanx is palpated individually for any pain, swelling, crepitus or deformity. Sesamoid bones must be examined for signs of pain usually. Bipartite sesamoids in especially the Rottweiler can be a source of lameness but can also be an incidental finding if not associated with pain. The metatarsal bones are palpated for abnormalities. It is also important to place medio-lateral and cranio-caudal stress on these structures to evaluate for excessive laxity or decreased range of motion.

Tarsus

Palpate this joint for swelling and pain. Range of motion should be evaluated. Maximum extension is usually 175 degrees. Flexion should only be possible together with stifle flexion. This is important when specifically evaluating the achilles tendon mechanism. The plantar ligament is the primary caudal stabilizer of the tarsus. Disruption of this structure usually results in significant lameness and

instability of this region. Other conditions commonly encountered are fractures of the malleoli and ligamentous injuries of the collateral ligaments usually seen with trauma. OCD of the talus can be found in young large breed dogs and usually results in significant lameness with a poor long-term prognosis.

Stifle

Observe the patient in a sitting position. Dogs with a positive “sit test” do not sit with the hock close to the ischium to relive stifle pain in flexion. The stifle must be evaluated for range of motion and pain without affecting the hip or hock joint. The joint also be evaluated for a cranial drawer sign in the unsedated patient because some dogs will show pain when performing this manipulation. Joint effusion is most easily judged by palpating the medial and lateral aspect of the patella tendon to feel for soft fluctuant distension. A hard thickening over the medial aspect of the stifle joint often indicate cruciate ligament disease due to the fact that the medial meniscus is attached to the medial collateral ligament. Palpation or hearing a “click” sound when dog is walking can indicate a meniscal injury. Owners often notice the clicking sound. Examination of the sedated patient focuses on the cranial drawer test. It is important to evaluate for the cranial drawer sign in flexion and extension. Partial cruciate ruptures only have laxity in flexion. These cases can be severely lame and painful. The tibial compression test can also be used to evaluate cranial cruciate ligament integrity. This test is not easy to perform but can be useful in large patients where it is difficult to perform a cranial drawer test. The tibia is compressed by flexing the tibio-tarsal joint while palpating the tibial crest for cranial translation.

Hip

The normal hip joint can usually be extended to almost parallel with the lumbar spine. When evaluating the hip for pain it is very easy to cause stifle pain because stifle extension occurs when extending the hip caudally. The clinician often places his/her hand on the stifle joint when manipulating the hip, which can elicit a pain response originating from the stifle and not from the hip. Abduction of the hip must also be checked for pain and discomfort.

It is very important that one always performs a rectal examination, abdominal palpation, evaluate femoral pulses and/or perform a urinalysis if one cannot find the source of the lameness by other means. It is obvious to see how important these steps are when considering how frequently lumbar spinal problems are missed and diagnosed as abdominal pain/bloat.

CONCLUSION

Without a systematic approach to hind limb lameness, clinicians will have a low success rate in finding the source of the lameness in these cases. This leads to decreased confidence when confronted with these mysteries. Clinicians should practice these examinations in order to become proficient in them.

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SURGICAL OPTIONS FOR THE REPAIR OF CANINE HIP DYSPLASIA

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ABSTRACT

Canine hip dysplasia (CHD) is a heritable condition with abnormal development of the femoral head and the acetabulum. Initial joint laxity leads to secondary changes in the bone and soft tissue structures of the coxofemoral joint. This degeneration leads to osteo-arthritis later. Although we have many options to treat CHD and do so quite successfully, prevention of the condition through selective breeding has failed to eradicate the condition. Schemes to try and reduce the incidence of this condition evolve all the time and so does the approach to treating these cases. The majority of dogs seen in practice are treated non-surgically. In my opinion veterinarians overestimate the degree of success achieved with these protocols. Many patients presented in referral practice arrive in significant pain with owners that are under the impression that their pet's pain is well controlled. Surgery for these dogs have come a long way and success rates are fairly high with regards to pain relief and return to function. This paper will aim to give the clinician a systematic approach to these cases and provide information on the different techniques available for surgical repair of Canine hip dysplasia.

INTRODUCTION

Canine hip dysplasia (CHD) is a heritable condition of abnormal development of the coxofemoral joint. Joint laxity progresses to joint deformity with secondary bony and soft tissue changes. This ultimately leads to osteo-arthritis with bone on bone contact that typically presents as obvious hip pain on manipulation. Most cases especially in the initial phases show a very subtle lameness or no lameness at all. A lot of owners just think that their pet is lethargic and weak. They often play less as puppies and also struggle to rise or go upstairs or jump onto objects. Diagnosis of hip dysplasia is fairly straightforward with radiographs being the main modality used to confirm and evaluate the extent of changes to the hip joints. It is important to remember how critical it is to be convinced that the hips are actually the sole cause of the clinical signs and owner complaint. This can be challenging in mature dogs with multiple radiographic abnormalities especially spinal pathology. In some cases further investigation with other modalities like MRI are needed to exclude spinal pathology. Video footage taken by the owner and also evaluated in slow motion can help a lot with deciding what the cause of the clinical signs is. Gait evaluation often shows weight transfer to the fore-quarter with subsequent over development of the shoulder and neck region. These dogs often run with their heads below the horizontal plane of the top of their shoulder blades as they compensate and move weight cranially when running.

DIAGNOSIS

Once you have established that the patient is suffering from hip pain, you have to make sure that there is no concomitant conditions like cruciate disease or lumbo-sacral disease that could also play a role clinically. The next step would be the radiographic evaluation done under deep sedation. Once the patient is sedated deeply, the veterinarian must also palpate the hip and knee joints. Degree of laxity, crepitus, range of motion and Ortolani signs must be evaluated. Both stifle joints must be checked and if any doubt also joint fluid aspirated and evaluated. When evaluating hips for possible surgery in young dogs, we take a number of radiographic views. Namely ventro-dorsal, "frog-leg", lateral pelvic and DAR (dorsal acetabular rim) projections. Subluxation and reduction angles can be evaluated to either predict hip osteo-arthritis later in life or plan for possible triple pelvic osteotomy. The best predictor of hip osteo-arthritis at the age of 2 was the reduction angle of the joint evaluated at the age of 6 months. A reduction angle of more than 15 degrees was shown to be a very good predictor of hip osteo-arthritis later in life when measured at 6 months of age. One should keep in mind that there will be breed differences for example GSD's have smaller reduction angles in general even when they develop OA. Rottweilers are more muscular and palpation could be falsely positive when evaluating the hip joints for laxity. The degree of radiographic changes found rarely correlate

with the severity of the clinical signs. It is important to explain this to the owner. The degree of pain and the loss of function are far more important than the radiographs when one decide on which option to take for treatment.

THERAPY FOR CHD

Basically 2 groups of therapy exist when treating a clinical case of CHD. Conservative therapy or surgical therapy. Conservative therapy consists of weight control, exercise modification, physical therapy, pain control medication and joint modification therapy. Surgical options include Juvenile Pubic Symphysiodesis, Triple pelvic osteotomy, Femoral head and neck excisional arthroplasty and Total hip replacement. In the past pectineal tenectomy/tenotomy, Intertrochanteric osteotomy and Shelf arthroplasty were also used for treatment of CHD. These three procedures are very rarely used today as the evidence that they work are very poor.

Conservative therapy

Activity restriction

Conflicting reports exist with low-level evidence on the effect of activity restriction. Some showed improvement in lameness assessments and other showed none.

Weight control and dietary restriction

High-level evidence showed a decrease in severity of radiographic changes and increased lifespan.

Modulation of degenerative joint disease

Good level of evidence exist for using polysulfated glycosaminoglycans in improving lameness scores but they were unable to show that this was an effective treatment for HD. Antioxidant therapy and mesenchymal stem cell therapy both showed improvement in clinical cases. Outcome measures were subjective and we are awaiting objective outcome measurements in the future for these therapies.

Acupuncture

Conflicting results were obtained using gold bead implantation at acupunctures sites. The placebo effect was also seen in these studies.

Surgical therapy

It can be very challenging to decide when to do surgery on hip cases and also which procedure to choose. There certainly is no “one size fits all” approach when it comes to CHD. Approaches differ widely between clinics and clinicians worldwide. The individual patient needs and owner expectations play a very big role. Finances are a very big factor in cases of CHD because of the cost of total hip replacements compared to the other procedures and therapies available to the owner. A very thorough discussion must be held with the client when trying to guide them into making the right decision regarding the treatment of their pet.

Juvenile pubic symphysiodesis (JPS)

Usually performed in puppies between the age of 3 and 5 months. The objective is to slow down growth of the ventral aspect of the pelvis by obliterating the pubic symphysis using electro-cautery. This stimulates overgrowth of the dorsal acetabulae in order to reduce subluxation of the coxo-femoral joint. The difficulty with this procedure is to positively identify dogs with clinical signs of HD at this young age. Patients should be sterilized at the same time, as these dogs should not be used for breeding.

Triple pelvic osteotomy (TPO)

This procedure is performed on dogs between then age of 6 and 12 months usually. Dogs with hip laxity, positive Ortolani manoeuvres, reduction angles suggestive of normal acetabulae and no secondary arthritic changes make good candidates for this procedure. Very good long-term results have been reported and are seen in practice if selection criteria are strictly adhered to. TPO attempts to save the hip joint by eliminating femoral head subluxation. One could argue that some of these patients might have improved spontaneously, but return to function is usually rapid and complication rates low. Also much more affordable compared to total hip replacement.

Femoral head and neck excisional arthroplasty (FHO)
Probably the most commonly performed hip surgery worldwide. This procedure has been reported to give very good results in medium and small breed dogs. Pain relief is usually reliable but full functionality is not always achieved. The procedure is simple but still needs to be performed properly to avoid post-operative pain and complications. Most common mistake is to not remove enough of the femoral neck and thus not eliminating bone-on-bone contact between the femur and the pelvis. It is also very important to provide soft tissue cover of the acetabulum. Using the correct equipment also prevents femoral splitting and insufficient bone removal. This is a salvage procedure and one should explain to the owner what the objective is with this procedure. Typically we expect pain relief after 2 weeks and a gradual return to full use of the limb between 2 and 4 months post operatively. Physical therapy can promote formation of the pseudo-arthrosis and speed up recovery. Muscle mass on the hindquarter also promotes a quicker return to function. The limb can typically not be extended as well after a FHO compared to THR and a normal hip. Some of these patients can become very stiff and painful again later in life. THR after FHO can be very challenging but is possible.

Total hip replacement (THR)
THR is readily available lately with excellent cementless systems now being preferred. Unfortunately a high cost procedure and also higher risk of complications due to the technical demand of the procedure itself. Very good success rate and close to normal return to function in most cases. A review of the long term outcome after Zurich cementless Total hip Arthroplasty in 439 cases revealed a 20 and 13 % complication rate in juvenile and adult dogs respectively. All cases were successfully revised except for 4 explants in total. The biggest factor in determining if a dog will have a complication was change in body condition score post operatively. 5-8 % complication rate is seen in general. The advantage of cementless systems is that they can be revised more successfully than cemented systems. When cement loosens or gets infected, all implants have to be removed usually. The most common complication is aseptic loosening of mostly the cup component. Luxation and femoral fracture are the next most common complications seen. When luxation is encountered, one has to determine the cause for example femoral neck too short or malpositioning of the acetabular cup component. If possible this is then corrected to solve the problem. Post operatively these patients are confined to a small room size area and leash walked for 6 weeks. At 6 weeks post op follow up radiographs are taken under sedation to evaluate the implant position and the bone-implant interface. Preferably no radiolucency should be seen between the implant and the bone but if any, this must be less than 1 mm. With cementless systems the bone has to integrate into the implant at 6-8 weeks post operatively. THR systems have also been developed for small dogs and cats. This is not available in South Africa yet.

CONCLUSION

No surgical procedure has been proven to consistently return dogs to normal function when suffering from CHD. Bergh and Budsberg reviewed peer-reviewed pubilcations between 1948 and 2012 on the surgical treatment of HD. This review found no evidence that FHO, TPO and intertrochanteric osteotomy or gold bead implantation could return dogs to normal function consistently. There was mixed evidence for THR and JPS. They looked at studies from an Evidence-based Medicine point of view and used the grading scheme for quality of evidence. Response to treatment is most often used by clinicians treating CHD to decide what the next step is for the patient. Regular follow ups and critical evaluation of the clinical outcome of conservative or surgical treatment of these cases will probably over time lead to more frequent and earlier surgical intervention in these cases. The choice of surgery must be based on expected outcome, risk factors and what seems best for the patient in the long term.

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SURGERY OF THE HEPATOBILIARY SYSTEM

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ABSTRACT

Cats and dogs commonly present with conditions of the liver and biliary tract that may require surgical intervention. Techniques such as liver biopsy, partial liver lobectomy and simple exploration of the biliary tract can be easily performed in general veterinary practice. It is important to remember that the liver has many diverse functions. It is the primary organ for protein; carbohydrate and fat metabolism, it detoxifies and excretes drugs and toxins; and it is responsible for the formation of bile and coagulation factors. Because of its large functional reserve, clinical signs of liver disease may not be apparent until the disease is advanced and many metabolic abnormalities become apparent. It is extremely important to understand the pathophysiology of the underlying disease processes and to review the anatomy and specific surgical techniques before embarking on hepatobiliary surgery.

THE LIVER

Pre-operative considerations

Liver disease can cause a number of significant metabolic and haematological disorders. Preoperative blood tests should be performed. In particular haematocrit, serum albumin, glucose and electrolytes should be closely monitored. Additionally, liver function tests such as pre- and postprandial bile acids and blood ammonia concentrations provide valuable information prior to surgery.

Hypoglycaemia

If the animal is hypoglycaemic, intravenous fluids supplemented with glucose should be given before and during the surgery.

Anaemia

Mild to moderate anaemia can occur secondary to liver disease due to blood loss (coagulopathy or gastro-intestinal ulceration) or it may be associated with anaemia of chronic disease. Animals with a haematocrit of lower than 20% should be given a pre-operative blood transfusion and blood loss during and after surgery should be monitored closely.

Coagulopathy

Animals with severe or chronic liver disease may bleed excessively. A coagulation profile should be performed in any animal with hepatobiliary disease that is undergoing major surgery. The liver is responsible for the synthesis of all coagulation factors except factor VII. The liver also clears activated clotting factors and fibrinolytic enzymes. Mechanisms of coagulopathy associated with hepatobiliary disease include the decreased synthesis of clotting factors, DIC and vitamin K deficiency. Vitamin K deficiency is usually associated with the malabsorption of vitamin K secondary to complete bile duct obstruction, but can also occur in association with severe hepatic insufficiency. Treatment with subcutaneous injections of vitamin K1 usually results in improvement of the coagulation within a few hours. If the coagulation is abnormal at the time of surgery, fresh whole plasma or fresh frozen plasma should be considered.

Hypoalbuminaemia and ascites

Albumin is synthesized exclusively by the liver. Hypoalbuminaemia does not typically occur until the functional liver mass is reduced by 70-80%. In end stage liver disease there is both decreased albumin synthesis and dilution of serum albumin due to sodium and water retention. Ascitic fluid that accumulates secondary to liver disease and hypoalbuminaemia is usually a transudate. Administration of plasma should be considered if albumin levels are significantly low. A large accumulation of ascitic fluid can displace the diaphragm and restrict the lung expansion. Removal of some of the fluid immediately prior to anaesthetic induction may help to prevent hypoventilation.

Hepatic encephalopathy
Dementia, disorientation, circling, head pressing, hypersalivation, seizures or coma are all clinical signs of hepatic encephalopathy that occurs secondary to severe liver disease or portosystemic shunting of blood. These clinical signs should be stabilized prior to surgery using dietary and medical management.

Anaesthetic considerations
Animals with hepatic disease have impaired ability to metabolize and inactivate some drugs. This results in a prolonged duration of action or altered function of drugs and anaesthetic protocols should therefore be chosen wisely.

Antibiotics
Prophylactic antibiotics are warranted in almost all dog and cats undergoing hepatobiliary surgery. There is evidence to suggest that anaerobic bacteria colonize the canine liver and that these may proliferate if there is hepatic ischaemia or hypoxia. Bacteria can also migrate to the liver via the portal venous system. The reticuloendothelial system of the liver normally clears these bacteria but the mechanism is compromised in animals with severe liver disease or portosystemic shunting of blood. Cephalosporins provide broad-spectrum activity and high tissue concentrations when given intravenously immediately prior to surgery. Penicillin derivatives, metronidazole and clindamycin may also be considered. Continuation of the antibiotics post operatively is determined by the findings at surgery and the results of culture and sensitivity testing.

Surgical management
A large laparotomy incision is required for thorough examination and surgical manipulation of the liver and biliary tract. A ventral midline laparotomy incision should be made from the xiphoid to the pubis. The falciform fat should be removed using either a Metzenbaum scissors or electrocautery by incising the falciform ligament along its attachment. A circumferential suture can be placed around the base followed by transection of the ligament distal to the ligature. Removal of the falciform ligament and fat will greatly improve visibility of the cranial abdomen.

In most dogs, incising up and through the cartilage portion of the xiphoid process provides adequate exposure of the liver and biliary tract. In some cases a caudal sternotomy or a transverse paracostal incision can be made after the abdomen has been opened if further exposure is required. Incising the peritoneal folds that suspend the liver from the abdominal wall and diaphragm can also facilitate manipulation and examination of the liver. By placing a hand over the ventral surface of the liver and gently retracting it caudally, the coronary and triangular ligaments can be seen and carefully transected. Extreme care must be taken not to incise the wall of the vena cava as it courses through the caval foramen of the diaphragm or the left hepatic vein as it drains into the vena cava just before the caval foramen. A ventrodorsal incision into the diaphragm from the xiphoid towards the caval foramen allows further caudal retraction of the liver. Another practical tip to facilitate exposure and manipulation of the liver may be for an assistant to retract the stomach caudally on stay sutures placed in the greater curvature of the stomach. Each lobe of the liver should be visually inspected and palpated and a full abdomen exploration should be performed. The gallbladder should be visualized and palpated and the cystic and common bile duct identified. Identification and retraction of the descending duodenum aids in locating the common bile duct.

Hepatic biopsy techniques, partial and complete liver lobectomies are procedures that may be attempted in private practice and will be briefly discussed in this lecture. Hepatic cysts and abscesses, liver lobe torsion, arteriovenous fistulae and hepatobiliary neoplasia are conditions of the liver that may require surgical intervention.

THE BILIARY TRACT

Extrahepatic bile obstruction and trauma are the two most common indications for biliary tract surgery.

Bile duct obstruction

Biliary disease may be caused by extrahepatic biliary tract obstruction (EHBO), neoplasia, infection, or trauma. Lesions that cause EHBO may be extraluminal or intraluminal. Extraluminal obstruction may be caused by pancreatitis, pancreatic neoplasia, duodenal or pyloric neoplasia, hepatic or biliary neoplasia, diaphragmatic hernia, congenital abnormalities, or pancreatic abscessation. Intraluminal obstruction is less common but may occur in association with cholelithiasis, choledocholithiasis, inspissated bile or neoplasia. Pancreatic disease is the most common cause of EHBO in small animals. Scar formation may occur in or around the duct, or the duct may be compressed by fibrotic or inflamed pancreatic tissue. Animals with EHBO should have electrolyte and fluid abnormalities corrected before surgery. Coagulopathy considerations and peri-operative antibiotic use is similar to what was discussed earlier under hepatic surgery.

Extrahepatic biliary injury may be caused by blunt or penetrating trauma. Common bile duct, gallbladder, cystic duct, or hepatic duct lacerations may cause bile peritonitis or a localized inflammatory process with adherence to surrounding organs. Necrotizing cholecystitis occurs when bacteria damage the gallbladder wall, which often results in peritoneal spillage of bile. This may lead to the development of severe, generalized septic peritonitis. Sometimes bile becomes inspissated before the gallbladder ruptures, and spillage of the relatively thick, gelatinous substance into the cranial abdomen causes a localized peritonitis. Adhesions or fistulous tracts occasionally occur around the gallbladder.

Animals with EHBO should be differentiated from those with intrahepatic cholestasis causing partial obstruction, because EHBO often requires surgery whereas intrahepatic cholestasis can usually be managed medically. The indications for surgery in animals with suspected extrahepatic biliary obstruction are not set in stone. An increasing serum bilirubin level over 7 to 10 days in the absence of primary hepatic disease or pancreatitis, combined with supportive radiography or ultrasonographic evidence of obstruction, is generally considered to be an indication for surgery. Dogs with EHBO due to pancreatitis seldom require surgery, although rare patients that do not respond to appropriate medical therapy may require a drainage procedure.

Surgical anatomy

A thorough knowledge of the surgical anatomy and the anatomical differences between cats and dogs is essential before performing hepatobiliary surgery on these patients.

The hepatic and cystic ducts, the common bile duct and the gallbladder constitute the extrahepatic biliary system. Bile drains from the hepatic ducts into the bile duct and is stored and concentrated in the gallbladder. The gallbladder lies between the quadrate lobe of the liver medially and the right medial lobe laterally. The cystic duct extends from the neck of the gallbladder to the junction with the first tributary from the liver. From this point to the opening of the biliary system into the duodenum, the duct is called the bile duct. The bile duct runs through the lesser omentum for approximately 5 cm and enters the mesenteric wall of the duodenum. The bile duct of cats and dogs is generally approximately 2-3 mm in diameter. The canine bile duct terminates in the duodenum near the opening of the minor pancreatic duct. This combined opening of the minor (accessory) pancreatic duct and the bile duct is the major duodenal papilla. The feline bile duct usually joins the major pancreatic duct before entering the duodenum. Thus, cats with intestinal and hepatic disease may be at increased risk for pancreatitis caused by ascending infection.

Surgical techniques

Exploratory laparotomy should be performed when i) leakage of bile into the abdomen is suspected; ii) biliary obstruction appears to be caused by a condition other than pancreatitis; iii) neoplasia (biliary tract, intestinal, or pancreatic) or iv) biliary calculi is suspected. During exploration, the patency of the common bile duct must be confirmed by manually expressing the gallbladder or by catheterizing the duct, either retrograde (from the duodenum) or in some cases normograde (from the gallbladder). The treatment of animals with EHBO secondary to pancreatic disease initially consists of medical management of the pancreatitis. If clinical or laboratory improvement is not seen within 7 to 10 days of initiation of therapy, cholecystoduodenostomy or cholecystojejunostomy may be considered. In extremely ill patients with biliary obstruction that cannot undergo surgical exploration, temporary decompression of the gallbladder may be considered using ultrasound-guided aspiration.

Tube drainage of the biliary tract, tube chloecystostomy, cholecystotomy, cholecystectomy, choledochotomy and primary repair of biliary trauma trauma are some of the surgical techniques that will be briefly discussed in this lecture.

Suture materials and instrumentation
Absorbable suture material should be used in the biliary tree because non-absorbable suture may act as a nidus for stone formation. Biliary duct surgery is aided by the use of small instruments such as those used for ophthalmic surgery. With biliary diversion surgery, the gallbladder should be emptied with a syringe and needle, or a needle and suction before surgical manipulation, to reduce spillage of bile during the procedure.

Post-operative care
Fluid therapy should be continued until the animal is able to maintain hydration with oral fluids. Electrolytes and acid-base status should be assessed and corrected during the post-operative period. Many patients with bile peritonitis are debilitated before surgery, and nutritional supplementation and temporary biliary diversion may be beneficial. Antibiotic therapy should be continued for 7 to 10 days if cholecystitis was present, or if bile leakage occurred before or during surgery. Open abdominal drainage may be considered in patients with generalized bile peritonitis. Biliary diversion surgery in cats is commonly associated with intraoperative and postoperative hypotension and anemia. Careful monitoring of these cases during intra- and post-operatively is essential.

Complications
Surgery of the extrahepatic biliary tree requires technical competence and sound surgical judgment to prevent serious complications. Potential complications after cholecystectomy (particularly if perforation was present) include generalized peritonitis, shock, sepsis, hypoglycaemia, hypoproteinemia, and hypokalemia. Close monitoring of clinical signs, blood pressure, complete blood count (CBC), biochemical profile, and coagulation tests during the postoperative period is advised to allow diagnosis and treatment at the earliest sign of a surgical complication or systemic inflammatory response syndrome. Stricture, bile leakage, and dehiscence may occur after surgery of the common bile duct. Ascending cholangiohepatitis may occur in some animals after biliary diversion, particularly if the stoma of the enteric-biliary anastomosis is too small and intestinal contents remain in the gallbladder lumen for prolonged periods. Long-term complications after biliary decompression include cholangiohepatitis, recurrence of obstruction, and chronic weight loss.

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SURGICAL MANAGEMENT OF LUMBOSACRAL DISC DISEASE

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ABSTRACT

Lumbosacral disease is a common condition mainly affecting large breed dogs. The common presentation is marked pain over the LS joint. However some dogs can develop neurological derangements in the hindquarters from ataxia to urinary and faecal incontinence. MRI is the gold standard in detecting DLS, however CT can be helpful on its own or in combination with MRI. Medical management should be tried as a first line in patients that present with pain as the only clinical sign. Unresponsive and patients with neurological derangements should have surgery. Surgical treatment is controversial however the general consensus is that a dorsal laminectomy should be performed to provide decompression of the cauda equina. A number of other procedures are combined with a dorsal laminectomy at the surgeon's preference to best treat the condition in their minds. The prognosis is good in patients presenting with pain and mild neurological derangements. The prognosis is poor in patients presenting with urinary and faecal incontinence.

INTRODUCTION

The understanding of lumbosacral disc disease otherwise know as degenerative lumbosacral stenosis (DLS) has changed dramatically in the last few decades. We now have a better understanding of the signalment of the presenting patient, the imaging of the patient and diagnosis of significant disease. Unfortunately we still have no idea as to what is the best method of treating the disease. The condition was though to occur exclusively in the German Sheppard and working dogs. With the increased availability of MRI in small animal practice we are imaging more dogs and finding DLS of a wide range of breeds.

The clinical signs of DLS are very important and form an integral part of the diagnosis of DLS. It is important to understand the clinical signs in relation to the specific anatomy of the lumbosacral joint (LS). The spinal cord in the dog ends within the vertebral body of L6. From there the nerve roots run in the lateral recesses through L7. The LS joint acts s a hinge between the powerful hindquarters and the spine to transfer the propulsion force of the hind legs through the spine to move the body forwards. The LS joint is a high motion joint, which is thought to play a role in the development of the secondary changes that lead to DLS.

The changes obvious changes are spondylosis deformans, hypertrophy of the dorsal annulus, hypertrophy of the synovium of the facet joints, collapse of the disc space, telescoping of S1 into L7 and narrowing of the intervertebral foramin. These changes all lead to a decreased range of motion in the LS joint and compression of the nerve roots. Radicular pain from compromise to the vascular supply has been reported but is difficult to demonstrate.

The generally accepted theory is that this is a disease of instability at the LS joint. This is however impossible to convincingly demonstrate in any imaging technique. Given this it is still unknown what role the instability post surgery plays and how to best manage these cases. Recent studies using positional CT are showing some improvement in our ability to determine this instability

Clinical signs are imperative to the accurate diagnosis of DLS. A patient without clinical signs should not have surgery or medical management of this condition. The two most important factors that indicate treatment is needed for DLS are clinical signs of pain, ischiadic nerve involvement and advanced imaging of LS compression.

Lumbosacral lesions can cause abnormal gait in the hindquarters, this is due to the lower motor neuron type lesion of the ischiadic nerve. This leads to a relative hyper-reflexia of the femoral nerve reflex test (patella reflex) due to loss of the inhibition from the caudal thigh muscles innervated by the ischiadic. There will then on testing the ischiadic nerve reflexes be a hyporeflexia. However pain on

direct palpation of the LS joint or extension of the LS joint is the most common clinical sign seen. Incontinence results from damage to the pudendal and pelvic nerve. The bladder is generally lower motor neuron and easily expressed. Faecal incontinence is form loss of anal tone. Regardless of any other clinical signs once the patient develops incontinence the chance of a return or continence is poor, around 50%.

Working dogs can show a increase in cinical signs after working. This is thought to be due to claudication of the LS area. This is failure of effective vasodilation of the vessels in the affected nerve roots.

Radiographs can give an indication of possible LS disease. However DLS can be seen in patients with normal survey radiographs. In any case radiographs should be taken to rule out any other conditions of the spine and hips. Normal radiographic myelogram is seldom helpful as the subarachnoid space narrows extremely over L7-S1 leading to poor contrast filling. This leads to poor image quality that is most often non-diagnostic.

Computed tomography can be helpful in the case of mineralised disc material in the canal. This can be detected on CT easily. Computed tomography is excellent to view the narrowing of the vertebral canal, the facets and the intervertebral foramina.

Magnetic resonance imaging is the gold standard in making a diagnosis of DLS. This will detect the soft tissue compression of the cauda equina. A recent study has shown the potential of MRI to image the intervertebral foramina accurately. Ideally one would like to combine MRI with CT in evaluation of these patients. The big concern with MRI is that it can lead to over diagnosis and this is the reason why it should always be combined with clinical signs of LS disease before recommending surgery.

TREATMENT

Medical treatment is the first starting point for these cases when they are mild. Dogs non-responsive to medical management or with neurological signs should be treated with surgery as soon as possible.

Medical management entails oral administration of NSAIDS, Tramahexal and 3-4 months of activity restriction with physiotherapy. A new approach has recommended a corticosteroid epidural of Celstone soluspan repeated 2 weeks then 6 weeks. A dose of 1ml for a small dog and 2ml for a large dog is the recommended dose (personal communication).

If the clinical signs return then surgery is indicated. In patients with incontinence surgery carries a guarded prognosis of 50% recovery.

Surgical treatment is simple and complex all in one sentence. Almost all authors will agree that a dorsal laminectomy forms the basis of surgical intervention. This entails removal of the dorsal lamina of L7 and S1 leaving the articular facets intact. This will allow space for the trapped nerve roots to move and stop the telescoping of S1 into L7 by removing this section of bone.

Now the story becomes complicated. It was found that dogs with incontinence responded poorly to dorsal laminectomy alone and better to laminectomy combined with traction-fusion. The traction-fusion theory is that is addresses the instability, which is cause or consequence of the DLS? Further concerns are is there entrapment of the nerve root leaving the foramin? Do we need to remove the type II disc if we have removed the lamina? This has created a whole host of procedural combinations used with a dorsal laminectomy.

Discectomy

Removal of the type II disc using a scalpel blade. The nerve roots are gently retracted out the way with a nerve root hook and the disc cut out. One must be very careful not to damage the nerve roots with the scalpel. Discectomy is thought to worsen the instability and collapse of the intervertebral foramina and will often be combined with a stabilisation technique.

Foraminal decompression or facetectomy
An upcut spinal ronger or Kerrison bone punch is used to remove soft tissue and bone believed to be obstructing the nerve root passage through the foramin. This is a danger area any false move and the nerve root is gone. A facetectomy entails removal of the articular facet on that side. This allows excellent visualisation but massively decreases stability and is not recommended.

Dorsal traction-fixation-fusion
The goal of this is to alleviate the compression from the telescoping of the S1 into L7 and open up the collapsed intervertebral foramina. There are a few ways of performing this. The main goal is to pull L7 and S1 into the normal position as determined by the articulation of the articular facets then stabilising them in place. The first is to place pedicle screws in the vertebra and then connect these screws with bone cement or rods. This provides good stability but recent studies have show accurate placement of the screws is only achieved in 70% of the screws. Little clinical effect is however seen from screws not placed perfectly. The other is to place trans articular screws through the facet joints. This is technically much easier but the merits are questionable. It has been shown that on follow up at 12 months 5 of 17 dogs had implant failure with little or no clinical signs. This begs the question as to what function the screws were serving in the first place.

Some authors have advocated ventral disc removal and traction stabilisation but this has not been fully explored as yet.

The current thinking is less is more and most authors will perform a dorsal laminectomy with removal of the disc as a standard. If they feel the need on that day they will adopt one of the stabilisation techniques in combination with the dorsal laminectomy and discectomy.

Postoperative care
This entails rest for 3 months with slow controlled walking and physiotherapy. Hydrotherapy can be helpful to make a full recovery. A slow return to normal activity over a period of a month is recommended. I will generally send them home on a course of NSAIDS and Tramahexal.

PROGNOSIS

The prognosis is generally good in most patients that maintain continence. Dorsal laminectomy provides immediate relief of pain dogs that had foraminal decompression showed no difference from dogs that had laminectomy alone. Traction fusion cases seemed to do as well as laminectomies alone. The general success rate ranges from 73-90% in the literature.

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MEDIAL PATELLA LUXATION IN CANINES

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ABSTRACT

Medial patella luxation is an extremely common condition of both small and large breed dogs. It is most commonly seen in small breed dogs but the prevalence is increasing in large breed dogs. Medial patella luxation usually occurs together with a conformational deformity of the femur. This needs to be taken in to account in the correction of this condition. Surgical correction consists of soft tissue reconstruction and bone correction to correct the alignment of the patella mechanism. The complication rate with surgical correction remains high with the most common complication re-luxation of the patella. The current thinking is to investigate correction of the underling conformational deformity to prevent this re-luxation. Correction of this femoral deformity should be made on measurements of the centre of rotation of the femur to quantify the degree of the corrective osteotomy. Femoral corrective osteotomies are technically difficult surgery but the recent results of success are very promising.

INTRODUCTION

Medial patella luxation (MPL) is a common developmental orthopaedic condition of the canine patient. Small breed dogs are more commonly affected by the condition than large breed dogs. However MPL is more commonly detected in large breed dogs than lateral patella luxation.

Patella luxation is easily detected clinically with palpation of the patella luxating medially from the trochlear groove with the leg in extension. The condition is more complicated than just a patella luxation. These patients will usually have associated musculoskeletal abnormalities. These include, medial displacement of the quadriceps muscle group, distal femoral varus, hypoplasia of the medial condyle and rotation tibial deformity. Clinically these patients have been graded as to the degree of patella luxation through full range of motion (Table 1). This grading system takes into account the effects of the other components of the condition by their effect on the patella. The grading system does not quantify the underlying components of the condition that have lead to the patella luxation. The big concern is that a grade 2 or 3 luxation can be present in a patient with a marked femoral varus and another patient with a mild femoral varus. This does not allow us to accurately determine the best way forward in regards to surgical treatment of each individual patient. Grade 4 patients mostly have moderate to severe osseous deformities that need correcting.

Table 1.

Grade	Description
Grade 1	Patella can be luxated but spontaneous luxation seldom occurs. Once the patella has been luxated during clinical exam, the patella spontaneously reduces when the examiner releases pressure. The patella is stable in the trochlear in full range of motion
Grade 2	Angular and rotation deformities of the femur may be present. The patella can be luxated and remains luxated through full range of motion unless reduced by the examiner. The animal can clinically reduce the patella and this is seen with the skipping gait. Once reduced the patella remains in the trochlear groove for the full range of stifle motion.
Grade 3	Angular and rotational deformities are often present. The patella remains luxated most of the time, The patella can be reduced by the examiner however normal range of motion of the stifle lead to luxation of the patella.
Grade 4	Moderate to severe angular limb deformities are often present. The patella is permanently luxated and cannot be reduced to the trochlear groove at any point in the normal rang of motion of the stifle. The trochlear groove itself is often shallow or convex.

Adapted from Fossum Et al.

There are many suggestions put forward to explain how MPL occurs. Some authors suggest that the pathogenesis in small breed dogs is different from large breed dogs. A reasonable suggestion is that MPL develops form a decreased angle of inclination of the femoral neck, coxa vara. This leads to marked angular deformities of the distal femur from bowing of the distal femur, genu varum. This causes a relative tibial varus and internal rotation of the tibia on the femur. The patella is then forced laterally due to the pull of the medial thigh muscles and hypoplasia of the medial condyle. Other studies have shown an increased angle of inclination of the femoral neck is associated with patella luxation in small breed dogs. Large breed dogs seem to have a relatively normal conformation of the femur when compared with small breed dogs. However large breed dogs with MPL tend to have a relative degree of patella alta when compared to the conformation of normal large breed dogs, this may be cause or consequence of MPL in large breed dogs.

SURGERY

Surgical repair of patella luxation should be performed on animals showing clinical signs of lameness associated with the patella luxation or in young animals to prevent the long-term complications later on in life form the patella luxation. The main goal of surgical repair is re-alignment of the patella quadriceps mechanism leading to normal sliding of the patella in the patella groove. Surgical repair has two categories, release or augmentation of the soft tissue components and corrective distal femoral osteotomy (CDFO) or tibial crest transplant (TCT). It has been shown that soft tissue procedures performed without osseous correction have a high failure rate and should never be used as a sole method of repair. Surgical complications using the current techniques are reported as high as 85-48%. Surgical complications using the TCT in combination with soft tissue repair techniques are reported to be as high as 20%. These complication rates should be considered to high for a condition that is relatively common. Surgical repair most often utilizes a combination of soft tissue and osseous repair to reposition the patella in the trochlear groove. The trochlear groove can then be deepened using one of the many described techniques for a trochlearplasty. Only techniques that salvage the hyaline cartilage in the trochlear groove should be used. Other methods of creating a deeper trochlear groove have been developed in recent times. These consist of RidgeStop^{tm} Developed by Orthomed UK. This uses a high-density polyurethane implant placed on the medial trochlear ridge to aid in the treatment of patella luxation. The current recommendation is that is should only be used alone in cases of mild patella luxation with no marked bone deformity of the leg. However it can be used together with the osseous corrective techniques to augment repair. Ridge stop offers a less invasive method to deepen the patella groove to aid in tracking of the patella in the normal alignment. It does not require the removal of a cartilage wedge to deep in the groove but does require accurate placement of 3 bone screws in the medial condyle to secure the implant. Kyon have developed an entire groove replacement made from titanium that the patella slides in. The author has no experience with this technique. The disadvantage to both these techniques is the increased cost to the client for the surgery. The implant is significantly more than expensive than a trochlearplasty. This should be discussed with the client on a case-to-case basis. Our recent experience with Ridgestop^{tm} is that is provides an adequate method for deepening the trochlear groove with less damage to the cartilage.

Given the high complication rates of soft tissue procedures performed on their own, it is recommended that they should always be combined with re-alignment of the osseous structures. A tibial crest transplant has been the standard operation used for re-alignment of the osseous structures. This technique is simple to perform but can have catastrophic complications. The complication rate varies from 9-50% for a tibial crest transplant. A corrective distal femoral osteotomy was until recent times only used for severe cases of patella luxation with a severe femoral varus and severe patella luxation. In these cases a guarded prognosis was given even though the animals did improve clinically but were never normal. However recently CDFO has gained popularity in correction of patella luxation with a moderate femoral varus. The challenge comes in patient selection for a TCT or CDFO. Most patients presenting with a patella luxation will have plain film radiographs as an initial step after the clinical exam. Plain film radiographs were shown to be 96% accurate in ruling a patient out of having a CDFO but only 76% accurate in patient selection for a CDFO. This was performed measuring the R-aLDFA of the femur, which was found to be an acceptable measurement to assess the varus deformity. R-aLDFA is a measurement made on radiographs to determine the centre of rotation of the distal femur, CORA in order to perform a corrective osteotomy. It stands for the anatomic lateral distal femoral angle. The technique for measurement can be found in most surgical texts.

It is recommended given the extra cost to the client that patients that are selected for a CDFO on radiographs should have a computed tomography scan performed of the femur and the R-aLDFA should be measured on the CT images. This gives the most accurate measurement of the femoral deformity in all planes. This allows the surgeon accurate planning for the surgery and gives the best possible outcome.

The reality of patella luxation surgery is that we don't understand the cause of the disease in these patients hence the conflicting literature on how to fix it. Current evidence from 2- 3 small studies are pointing to wards a lower complication rate with CDFO than TCT. This needs to be evaluated in the future to best help us treat our patients. The reality is that the TCT with associated soft tissue procedures is a great surgical procedure in 80% of cases. However in the 20% of cases we see major complications it can be a nightmare to repair. The 60% of dogs that develop a recurrent patella luxation at 4-6 weeks post TCT often are asymptomatic. The underlying question is what harm is this recurrent patella luxation doing to the articular cartilage down the line?

The big challenge and hope of this lecture is that the surgeon will no longer apply 1 surgical technique to all patients with patella luxation. Instead the surgeon should assess the underlying anatomical deformities of the patient and plan the surgery from there. The author suspects the high failure rate for TCT up to 50% is caused by this technique being used in all patients presenting with medial patella luxation without thought to the underlying anatomical deformities leading to or exacerbating the patella luxation.

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HOW TO ENSURE THE BEST POSSIBLE OUTCOME IN INTESTINAL SURGERY

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INTRODUCTION

There are many indications for surgery of the small intestine. Surgical intervention with supportive, diagnostic or therapeutic intent is routine in both general veterinary practices and referral hospitals.[1] Although commonly performed, outcomes for small intestinal surgery can be greatly improved if certain pre-operative considerations are taken into account and the basic principles of small intestinal surgery are followed.

ANATOMY

A thorough understanding of anatomy is essential when performing abdominal surgery. A systematic exploration of the entire abdomen is required when performing small intestinal surgery. The small intestines are approximately 3.5 times the body length of dogs and cats. The small intestine is divided into a fairly immobile duodenum, a mobile jejunum and a short ileum. A full anatomical description of the abdominal organs is beyond the scope of this lecture. The small intestine is structurally composed of four layers: the mucousa, submucousa, muscularis and serosa. The submucousal connective tissue is the holding layer of intestine and therefore the key structure to suture when performing intestinal surgery.[2]

INTESTINAL HEALING

Generally speaking, intestinal wounds gain strength more rapidly than skin wounds. Healing may however be delayed by a number of factors (hypovolaemia, hypoproteinaemia, anaemia, concurrent infection, immunosuppression, metabolic/endocrine disorders and malnutrition) in compromised animals. Failure to recognize and address these “at risk” patients will result in increased morbidity and mortality due to wound dehiscence and subsequent peritonitis.[3] The omentum also plays a valuable role in intestinal healing. It provides a seal to the anastomosis, enhances the vascular supply, establishes lymphatic drainage, stimulates granulation tissue and helps to prevent and control infection.[4]

PATHOPHYSIOLOGY OF INTESTINAL OBSTRUCTION

Intestinal obstruction may be mechanical (e.g., foreign body, intussusception or neoplasia) or functional (e.g., idiopathic pseudo obstruction) and causes profound effects on body fluid balance. Consequences include excessive fluid secretion; malabsorption of water and solute; fluid, electrolyte and acid-base disturbances; and proliferation and translocation of luminal bacteria.[5] Loss of intestinal alkaline fluids typically results in metabolic acidosis. If the obstruction is located in the proximal duodenum, however, gastric fluids that are rich potassium, sodium and hydrochloric acid are vomited, resulting in a hypochloraemic, hypokalemic metabolic acidosis. Because of these pronounced consequences, volume loading and circulatory support are critical in affected patients.[6]

Large fluid losses from vomiting, diarrhoea and sequestration within the intestinal lumen invariably lead to a dehydrated patient on presentation. Vomition from a proximal small intestinal obstruction typically presents with severe dehydration and a hypokalaemic, hypochloaemic, hyponatraemic metabolic acidosis. Patients with a more distal or partial obstruction usually present dehydrated, with a variable acid-base and electrolyte status.[2,5]

PRINCIPLES OF INTESTINAL SURGERY

Fluid therapy

Fluid, electrolyte and acid base imbalances should be assessed and treatment initiated immediately with intravenous therapy. It is advised to attempt to correct 50—75% of the deficit prior to surgery, unless an acute abdomen indicates immediate surgical intervention.[7] Most intestinal surgery cases can be managed with chrystalloids alone, but severe acute cases often require a combination of colloid, chrystalloid and blood component therapy to support blood volume, maintain systemic pressures and provide adequate oxygen delivery to the cells.[6] When protein concentrations are low, healing will be delayed. A serum albumin concentration < 20g/l constitutes a serious risk for delayed wound healing. In these cases, suture line re-inforcement should be strongly considered.[2] Once the patient is under anaesthesia, fluid deficits will worsen due to the hypotension associated with anaesthetic drugs, and with the evaporation from the anaesthetic circuit and open abdomen. Surgical fluid losses occur from tissue damage during dissection. Fluid therapy must be continued throughout surgery and the post-operative period. To maintain circulatory volume, an appropriate chrystalloid (10ml/kg/hr) should be administered during surgery. Oncotic support may be indicated in critical cases. In the post-operative period, fluid rates can be adjusted to the patient's condition, usually at twice maintenance in the non-complicated case until hydration balance is normal.[5] Weighing the animal once or twice a day can provide valuable information regarding haemodynamic stability of these patients.[7] The measurement of central venous pressure may also be helpful.[1]

When to operate?

For lower or partial obstructions, surgery should be performed within 12 hours of diagnosis. This allows times for correction of fluid, acid-base and electrolyte abnormalities. Most of these imbalances can be partially corrected and these patients are good surgical candidates within several hours. Immediate surgical intervention is indicated for complete intestinal obstruction, intestinal perforation, strangulation, mesenteric volvulus or penetrating abdominal wounds. Bolus fluid therapy and other emergency managements are instituted simultaneously.[1]

Antibiotic prophylaxis

The small intestine of dogs normally contains gram-positive and gram-negative organisms. Although the bacteria present in the small intestine represent a possible source of post-operative infection, the need for antibiotic coverage during intestinal surgery is still being debated. If prophylactic antibiotics are used, a number of factors need to be considered:

- The antibiotic should be appropriate for the bacteria that are suspected at the surgical site.
- The antibiotic should be in the tissues at the time of surgery
- A total of one or two doses are administered, or at most, antibiotics are continued for up to 24 hours unless ongoing infection is present

Extended use of antibiotics does not prevent infection and increases the incidence of resistant bacteria.[2]

Instrumentation

Small intestinal surgery can be performed without specialized instrumentation, but certain instruments greatly facilitate most procedures. A Poole's suction tip, DeBakey forceps and self-retaining abdominal retractors are essential. Hand held retractors are also very useful to expose deeper structures. Doyen non-crushing intestinal forceps are useful for occluding the lumen of the bowel without compromising perfusion of the intestinal wall.

Surgical considerations

The intestines should always be handled gently. Excessive handling and drying of the intestines may result in vagal response and post-operative ileus. The intestines should be kept moist at all times because surgery lights are very desiccating to these delicate tissues. Hands/fingers are the best "instruments" for examining the intestines and occluding the bowel lumen. Only the tips of the forceps should be used on the serosa of the bowel, as opposed to grabbing and crushing all the layers of the intestine. Similarly the adventitia of any blood vessel should be grasped, and not the vessel itself. Dissection should always be sharp and precise. Electrocautery should not be used on the bowel wall. Haemorrhage from transected or incised bowel will soon clot with gentle pressure from a moistened gauze swab. Likewise, bleeding from the vasa recta or arcuate vessels should be ligated

with fine ligatures of absorbable monofilament suture material and not electrocautery. Minimal tension should be applied to the suture line.[3]

Assessment of Intestinal Viability

Determining bowel viability is difficult because many clinical criteria can either overestimate or underestimate the amount of intestine that may recover from an ischemic insult. The standard subjective criteria for viable intestine include colour, arterial pulsation, peristalsis and bleeding from the cut edge.[8] The bowel should be moistened and warm when assessing these criteria. These clinical factors do not necessarily correlate with the histological severity of intestinal damage or the survival of the animal.[8] Clinicians may be misled by intramural haemorrhage and oedema which invite a positive prediction of bowel viability.[9] Fortunately, overestimation of an area of resection is not usually detrimental based on experienced clinical judgement. If viability is questionable, intestinal resection is the best choice.[3] Around 75-80% of the small intestine can be resected before permanent adverse effects are seen (short bowel syndrome). When short-bowel syndrome is a concern the use of more objective measurements of vitality, such as surface oximetry or fluorescein infusion may be helpful in the decision making process.[9,10]

Fluorescein is an organic dye that emits a gold-green fluorescence when exposed to ultraviolet to ultraviolet. Intravenous infusion results in wide distribution within minutes. Using a Wood's lamp to illuminate the intestines in a darkened surgery suite, a homogeneous gold-green pattern of fluorescence can be seen if the intestinal blood supply is patent. Non-viable intestines have patchy areas of non-fluorescence greater than 3mm, indicating loss of vascularity.[9] Surface oximetry may also be useful for the assessment of bowel perfusion and has shown reliable results. One limitation is however that the surface oxygen tension electrode measures a serosal surface area of a few millimetres in diameter and patchy segments of ischaemia can be missed.[2]

Exclusion draping

Following the initial exploration of the abdomen, the affected areas of bowl should be isolated and excluded (packed off) from the remaining abdominal contents. This is achieved by careful placement of at least four large laparotomy swabs moistened with warm sterile saline. The swabs act to protect the abdomen form contamination in case of inadvertent leakage from an enterectomy or enterotomy. This also keeps the abdominal content moistened and decreases heat loss. Small gauze swabs should not be used in the abdomen unless the surgeon is always prepared to perform a pre, intra and post-operative swab count.[11]

Suture material

The selection of suture material should be based on the known biological properties of the suture and the particular clinical situation in which it is going to be used. Monofilament synthetic absorbable (polydioxanone, polyglyconate) or non-absorbable (nylon or polypropylene) sutures are excellent choices for enteric closure. The use of multifilament suture materials have been described but they produce more tissue drag than monofilaments and may potentiate infection in the presence of contamination.[12] Monofilaments are less susceptible to bacterial adhesion and allow easier clearance of bacteria by host defence mechanisms.[13]

Factors that induce inflammation at the incision site prolong the lag phase of wound healing and delay wound healing and return of strength.[14] Inflammation activates bowel wall collagenase, which degrades collagen within the wound and erodes the foundation in which sutures are anchored. All suture materials produce an inflammatory reaction after placement because of their foreign body nature and the mechanical disruption and manipulation of tissues that occurs with their placement. Multifilament suture materials produce a greater inflammatory response than monofilaments.[13] Catgut stimulates an inflammatory response during its absorption which occurs very quickly when it is exposed to proteolytic action of gastrointestinal secretions.[14] A fine suture material is always indicated, usually 4/0 or 5/0 and occasionally 3/0 in size. A common error is to use too large a size of suture material.

Stay sutures are atraumatic and can be used to manipulate the bowl as needed. As early as 1887, submucosa was recognised to be the strongest part of the intestinal wall. It is therefore critical to include this layer into the enteric closure. Good submucousal apposition can result in primary intestinal healing with direct bridging of the defect. Poor submucosal apposition results in healing by second intention with indirect bridging of the submucousal layer and a prolonged presence of an

epithelial defect.[15] Submucousal apposition is poorer with two layer closure than single layer closure.[16] Two layer closures also result in avascular necrosis of the inverted cuff of tissue which prolongs the lag phase of wound healing. These closures also result in more intraluminal protrusion of tissues making the animal more prone to obstruction. Single layer closure, with direct apposition of the submucousa is therefore preferred in dogs and cats. Everting patterns are also best avoided as they are more likely to elicit adhesion formation.[11] Approximating suture patterns avoid these potential complications. Although approximating patterns often show some degree of eversion, they offer consistently good results and do not obstruct the lumen. The mild degree of eversion seen with these patterns does not obstruct the intestinal lumen and does not pose a significant clinical concern in small animal intestinal surgery. Accurate apposition is difficult to obtain, due to the tendancy for the redundant mucousa to bulge outward from the lumen. Mucousal eversion can be minimized by mucousal trimming with metzenbaum scissors, modifying a simple bite to a Gambee suture and by using a simple continuous suture pattern.[1]

Knot tying force

The amount of force applied to the knot is applied naturally by the experience of the surgeon. The rule of thumb is that tissues should be well opposed without being crushed. Crushing the tissues between the sutures inhibits angiogenesis and impedes wound healing.[2]

Suture line re-inforcement

The omentum is a mesothethelial membrane that has been referred to as the “abdominal policeman”.[4] It has extensive vascular and lymphatic supply and has proven angiogenic, immunogenic and adhesive properties that assist in controlling infection, restoring blood supply and establish lymphatic drainage.[17] Studies have shown that when the omentum is wrapped around an avascular small intestinal anastomosis it is capable of establishing an adherent sheath which prevents perforation and fatal leakage as well as re-vascularizing the region. The omentum can easily be positioned to allow complete coverage of almost any small intestinal anastomosis site. Unfortunately the omentum will not prevent leakage in all cases and serosal patching may need to be considered in some patients.[18]

The jejenal serosal patch has been referred to as the surgical parachute: “It is rarely needed, but when the occasion arises, nothing else can take its place”.[19] It has been clinically shown to reliably seal contaminated and grossly infected intestinal perforations in dogs. A loop of healthy jejenal that can easily reach the surgical site in question is selected. The antimesenteric border is placed over the questionable suture line and secured with a row of simple interrupted sutures of 3-0 or 4-0 monofilament suture material along either side. Each suture should include the submucousa of both segments of bowel. Although this process is time consuming, its use may prevent serious complications after surgery.

Abdominal lavage and suction

Thorough lavage of the abdomen with copious amounts of warm ringers lactate and diligent suctioning before closure are considered to be essential for improved outcome. Repeated irrigation and suctioning of the abdomen until the fluid is almost clear acts to reduce contaminating bacteria and debris, removes residual blood, warms the abdomen, moistens all the organs and enable a final check of the cavity.[11]

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SURGICAL MANAGEMENT OF THORACOLUMBAR INTERVERTEBRAL DISC DISEASE

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ABSTRACT

Dogs presenting with acute onset of spinal pain and/or neurological deficits are often suffering from a condition called Intervertebral Disc Disease (IVDD) and showing the early signs of Intervertebral Disc Prolapse (IVDP). This disease is hereditary and more frequently found in small breed dogs with the Dachshund being overrepresented. Pekingese and Maltese and are often also affected. The condition occurs due to an early chondroid degeneration of the nucleus pulposis in the intervertebral disc space. In some cases protrusion and extrusion of this central disc material occurs and results in entrapment of the spinal cord and adjacent nerve roots. The thoracolumbar region carries by far the highest incidence of disc protrusion and will be the emphasis of this article. Clinical signs include change in behaviour; yelping when picked up, tense abdominal and paraspinal muscles and hind limb ataxia. Acute paralysis may be immediate in cases where prolapse of the central nucleus pulposis is explosive or it may become apparent with time as more disc material leaks out through the rupture. Surgical decompression is indicated in almost all cases of disc prolapse where neurological signs are evident and no improvement occurs within a 24-48 hours period. Success rate with the procedure is high and many dogs regain full function following surgery.

INTRODUCTION

Intervertebral Disc Disease (IVDD) is a hereditary condition and the most common cause of myelopathy in dogs. It is found in all breeds, but particularly prevalent in the Dachshund and other chondrodystrophic types. It is caused by an early degeneration of the nucleus pulposis of the intervertebral disc. The loss of ability of the disc to withstand pressure prevents them from acting like a shock absorber and may result in secondary degeneration and rupture. The nucleus sits eccentrically within the disc space and thus excessive pressure results in a dorsal rupture of the annulus followed by protrusion of the nucleus into the vertebral canal. This is known as Intervertebral Disc Prolapse (IVDP). Compression of the spinal cord within the canal, as well as entrapment and irritation of the adjacent nerve roots, results in the clinical signs that we are frequently see.

Disc degeneration in dogs has been categorised as either Hansen Type I (Chondroid degeneration), which occurs largely in small breeds of dogs and results in a frank prolapse with extrusion of the calcified disc material into the vertebral canal or Hansen type 2 (Fibrinoid degeneration), which is more prevalent in large breed dogs and presents as a chronic protrusion resulting in progressive nerve compression, pain and neurological deficits.

This paper will concentrate largely on Hansen type 1 degeneration and in particular of the thoracolumbar spine. This is a condition that affects young, chondrodystrophic breeds. One study indicated that only 5% of dogs with thoracolumbar disc disease are older than 8 years.

HISTORY AND CLINICAL EXAM

The clinical signs of thoracolumbar IVDP can look deceptively like early gastrointestinal disease. Dogs appear hunched and uncomfortable, they frequently yelp when they are picked up. Behavioural changes like not wanting to jump, abnormal micturition and defecation and general exercise avoidance are common. Once neurological signs like hind limb incoordination, ataxia, and even paresis and paralysis show, the cause becomes more apparent. Paralysis may progress very rapidly and it is well documented that a fast progression of clinical signs correlates to more severe IVDP and a poorer prognosis, especially if surgical decompression is not achieved rapidly. A useful guideline for poorer prognosis without surgery is more than 12 hours from loss of hind limb motor function and more than 6 hours from loss of deep pain sensation.

The first step in diagnosis is always to obtain a good clinical and treatment history. Record the age of the patient; establish how long ago the problem started and what the patient was doing when clinical signs were first noticed. There is often little or no history of trauma involved. We need to know how fast the clinical signs have progressed and what previous treatments were given, as these could influence our prognosis. We need to determine whether the dog has had any previous incidents of spinal problems or of showing similar clinical signs.

Clinical examination should always begin with observing the patient's mental and ambulatory status. Place the animal on the floor of the consulting room and get an idea of whether you are dealing with an orthopaedic or neurological problem. If neurological, are clinical signs related to the central or peripheral nervous system? Use this time to assess the level of pain and degree of motor function. Even dogs with bilateral pelvic limb paresis, may show slight motor function. This provides us with more information on the functional status of the spinal cord and greatly improves the prognosis. Large breed dogs are often easier to examine outside the consulting room on a "non-slip" surface, where they can be trotted out and turned in circles. Carefully note any muscle atrophy, asymmetry, ataxia, high stepping gait abnormalities or dragging of the claws. "Root signs" caused by compression of specific nerve roots will manifest as pain or lameness in a limb and provide us with further valuable information. Remember that treatment with an anti-inflammatory could mask the clinical signs and make the assessment of the hyperpathic level inaccurate. It is important to know this information up front.

A "screening" neurological examination for hind limb paresis / paralysis consists of assessing conscious proprioception and testing hind limb reflexes including patella and cranial tibial reflexes. Cutaneous and perineal reflexes may also help to localise the lesion. Palpation with light pressure on the dorsal spinal processes is likely to elicit a hyperpathic level and this can be surprisingly accurate in determining the level of the disc prolapse. Assessing and understanding conscious deep pain response is vital to the ultimate prognosis and owners usually require this information before conceding to the costs of the diagnostic modalities and treatment options suggested. A simple withdrawal reflex is often confused with conscious deep pain sensation and is not an indicator of the same favourable prognosis.

DIAGNOSTIC IMAGING

In most cases, clinical assessment provides enough clarity to diagnose the condition of IVDP, but it gives no clear direction with regard to actual disc space affected or lateralisation of the prolapsed material. This information is required for surgical decompression. Good quality survey radiographs give some idea as to number of calcified, dehydrated disc spaces present and may even show comparably narrowed disc spaces, but is unable to confirm herniation. Lumbar myelography, MRI or CT scans are still needed for accurate localisation in order to achieve decompression.

Advanced diagnostic imaging modalities have greatly assisted the practitioner in making diagnoses in spinal patients; however these techniques are not a substitute for a thorough clinical work-up and good quality survey radiographs. Magnetic Resonance Imaging (MRI) and Computer Tomography (CT) examinations are much more likely to be diagnostic if the clinical examination has narrowed down the area of interest to a small segment of the spinal cord. Narrower image slices can then be utilised to identify and quantify the lesion. Understanding the scope of MRI/CT verses conventional myelography is important.

Collection of cerebrospinal fluid (CSF) at time of myelogram is easy and can be useful especially for differentiating between inflammatory, infectious and neoplastic conditions. It should routinely be sent for analysis, unless the diagnosis of IVDP can be confirmed by the myelogram.

TREATMENT OPTIONS FOR IVDP

To date, surgical decompression of confirmed IVDP in cases with significant neurologic deficits is the only feasible treatment option. In some cases, veterinarians or owners may get away with conservative or medical options, but the risks of this far outweigh the benefits. Owners need to have it explained to them, that prolapsed disc material is essentially a foreign body to the dog's immune system. The inflammatory response mounted by the body, as well as the neurogenic pain caused by the physical compression from prolapsed disc material, is extremely painful and recovery is

statistically prolonged without surgery. Furthermore the risks of the residual compression causing ongoing chronic damage to the spinal cord and nerves needs to be considered.
When medical management is instituted for financial reasons or in animals with mild, non neurological clinical signs, it involves a combination of enforced rest, use of analgesics and muscle relaxants, as well as physical rehabilitation. Previous studies suggest that medical management can be successful in up to 80% of ambulatory dogs, but only 45% of non-ambulatory dogs. These results are extremely optimistic when it comes to dogs that have lost motor function or conscious deep pain sensation.

The preferred technique for surgical decompression in the thoracolumbar region differs marginally based on the location of the disc material and the surgeon's preferred approach. Variations include dorsal laminectomy, hemilaminectomy, mini-hemilaminectomy, pediculectomy and lateral corpectomy. Dorsal laminectomy, which removes the dorsal lamina, is a slightly easier approach to the cord, but only gives limited access to the disc material trapped ventral to the spinal cord. Pediculectomy, which as its name suggests, involves the removal of the lateral wall of the vertebral canal, is technically more challenging, but leaves facet joints and dorsal spinal processes unharmed and therefore causes the least iatrogenic instability. A modification of both techniques is used when surgeons remove the facet joint and in this way gain better visualisation of the cord, but marginally increase instability.

More recently, an article has described a technique known as corpectomy. In this technique, the surgeon works ventral to the vertebral canal and longitudinal ligament and selectively drills away the vertebral floor adjacent to the affected disc space. Whilst some degree of weakening of the vertebral body may occur, the incidence of vertebral fractures post surgery is uncommon and the technique is particularly useful for chronic and adhered disc protrusions.

In the caudal lumbar region, where the wings of the ilium make the approach to the pedicles virtually impossible, most surgeons prefer a dorsal laminectomy technique. The nerves of the *cauda equina* allow for removal of disc material from between them, meaning that minimal damage to the distal spinal cord occurs.
Intervertebral disc fenestration is a technique described to remove nucleus pulposis material via a lateral approach to the disc and hopefully avoid its herniation into the vertebral canal at a later stage. It is generally not recommended alone as a treatment for disc herniation, as it doesn't remove the already herniated material and it is controversial whether it should be used prophylactically in dogs without clinical evidence of disc herniation.

POST OPERATIVE CARE

Neurosurgical patients are often significantly debilitated following decompression and until their normal range of function returns, they require a huge amount of nursing care in order to allow them to recover optimally. It is important that the hospital nursing staff has a high level of training for this and are properly informed of which parameters need to be monitored. The post-operative care of spinal patients can basically be divided into pain control, the nutritional management, bladder management, physical rehabilitation and the management of pressure sores and other complications.

PROGNOSTIC INDICATORS

In general the outcome in dogs with chronic annular protrusions (Hansen type II) is significantly worse than in dogs with nuclear extrusions (Hansen type I). Factors that influence prognosis include amount of disc material extruded and rate with which it was ejected. Rate of deterioration of the dog's clinical signs, presurgical neurologic status and amount of spinal cord swelling noted on myelographic study. Obesity, which is common in the chondrodystrophic breeds, is a significant negative factor and owners should be warned accordingly that their dogs will suffer higher post operative morbidity.
Functional recovery is defined as being ambulatory with normal urination post surgery. This may occur despite some residual disc material remaining in the vertebral canal. Although studies have been done, it is unclear what reserve capacity the spinal cord has to cope with this material and the resultant compression.

CONCLUSION

IVDP is the most common cause of myelopathy, especially in small breed, chondrodystrophic dogs. In the early stages, the clinical signs are often misinterpreted as having gastrointestinal origin. Due to

the potential for rapid and catastrophic deterioration in acute cases of disc extrusion, it is important that vets are aware of the signalment, recognise the condition and give owners the option of decompressive surgery. To date decompressive surgery is the single best treatment option available, especially in cases showing severe pain and neurologic dysfunction. An inability to recognise the condition and act accordingly can significantly decrease the patient's chances of making a full functional recovery.

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HOW TO DEAL WITH A BLOCKED URETER

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ABSTRACT

Ureteral obstruction can be acute or chronic in nature. Cats are most often affected but it can be seen in dogs. The most common cause is obstruction due to urolithiasis. However obstruction can be due to stenosis trauma or neoplasia. These patients often present in an azotaemic crisis due. The reason for this is that often obstruction and damage to one of the kidneys go unnoticed. The remaining kidney will hypertrophy and take over renal function. The crisis presents when the remaining kidney obstructs. Traditional surgical methods are fraught with complications and a high rate of recurrence due to stricture from fibrous tissue. The introduction of ureteral stents has dramatically increased the survival time in these cases and decreased the recurrence of urolithiasis. Stents can be combined with traditional surgery or lithotripsy. The addition of the subcutaneous ureteral bypass system has decreased surgical time in these compromised patients and further decreased complications. However subcutaneous bypass being a new technique needs to be carefully evaluated in the long term.

INTRODUCTION

The diagnosis of a ureteral obstruction can be very difficult. These cases are often detected in retrospect. The so called big kidney small kidney syndrome represents obstruction of both kidneys, a chronic obstruction leading to severe fibrosis and atrophy in the small kidney the second being acute obstruction with marked swelling oedema and hydronephrosis in the large kidney. This patient now starts off with only one functional kidney that has now obstructed. This represents an emergency to salvage renal function.

Ureterolithiasis is an uncommon syndrome affecting mainly cats but can affect dogs in some cases. However the consequences of a complete ureteral obstruction are catastrophic on the kidney. Complete obstructions need to be decompressed as a matter of urgency to salvage kidney function. Following ureteral obstructions there is a dramatic increase in ureteral pressure. This leads to marked changes in glomerular filtration rates (GFR) and renal blood flow. The GFR is decreased due to the backpressure, which is transferred to the entire nephron system. This leads to a release of vasoactive compounds, leukocyte infiltration and early fibrosis. This cascade of events further decreases GFR and leads to irreversible damage of the kidney. Obstruction for 7 days leads to a loss of 34% of renal function and obstruction for 14 days leads to loss of 54% of renal function in a study performed on healthy dogs. This damage cannot be reversed.

A recent report showed up to 95% of ureteroliths are made up mainly of calcium oxalate. These stones cannot be dissolved medically but have to be passed into the urinary tract and excreted.

MEDICAL MANAGEMENT

Medical management of the ureteral obstruction has value in the patient that has early detection of the obstruction and can be useful in partial obstruction of the ureter. The initial therapy is intravenous fluid therapy to try “push’ the ureterolith into the bladder and alleviate the obstruction. A constant rate infusion of Mannitol to increase the pressure in the ureter to move the stone through can be used if fluid therapy fails. Given the risk benefit ratio of surgery in the past partial obstructions were often left in place. With recent advances it is best to manage these obstructions to try maintain long term renal function.

SURGICAL MANAGEMENT

Surgical options allow immediate decompression but are invasive in an already critical patient. There are many options available to surgically decompress the kidney and divert urine away from the

blocked ureter. Some of these provide a temporary solution some provide a permanent solution. A nephrostomy tube can be placed directly into the pelvis of the kidney to relieve pressure and divert urine away this is obviously a temporary procedure used in patients that will then require another surgery to remove the obstruction. The reality of this in animals is that they will seldom tolerate this for a long period of time leading to patient interference of the tube.

Traditionally celiotomy and ureterotomy was the surgical procedure of choice in patients with an obstructed ureter. There is a high risk of stenosis of the ureter at the site of ureterotomy. This would however still be the first choice of surgery with neoureterocystostomy being second choice depending on where the stone was located in the ureter. To perform neoureterocystostomy the obstruction would have to be in the distal third of the ureter to allow re-implantation to the bladder. Ureteronephrectomy removes the obstruction but the kidney and its renal function together with the kidney. This is obviously not an option in a cat with questionable renal function already. That then necessitates renal transplantation, which carries a big ethical problem for the donor cat and a high cost factor and life long immunosuppressive treatment.

The complications with these surgical procedures of 30% depending on the procedure selected. Most complications were from odema, stenosis of the ureter at the surgical site, reformation of stones then moving into the stenosis and forming an obstruction again. These surgeries take a significant length of time to perform which can further affect the outcome of the already critical patient.

In one study on ureteral surgery it was found that up to 40% of cats had recurrence of urolithiasis leading to ureteral obstruction and over 75% had persistent azotemia from initial diagnosis to follow up. However patients treated surgically performed better than patients treated with medical management alone. Medical management should be considered, as a first choice but is at best effective in 17% of cases. It works best for stones less than 2-3 mm in diameter and located in the distal third of the ureter.

In recent times ureteral stenting subcutaneous ureteral bypass have become the treatment of choice in these patients. In humans stenting can be performed endoscopically with minimal surgical morbidity. This is often combined with Holmium: YAG laser lithotripsy to give an excellent success rate in removal of ureteral stones. Ureteral stenting is then performed post ureteroscopy for post procedural swelling and spasm of the ureter. Stents and subcutaneous bypass systems have been used to treat both malignant and benign obstructions in human patients. The subcutaneous bypass system has been used to treat post renal transplant strictures with excellent success in human patients.

Ureteral stenting has been well documented in veterinary medicine. Stents do not generally remove the stone they bypass it in the ureter. The stone can be removed via lithotripsy or surgery if needed. Stents allow passive drainage of urine into the bladder. They can be performed via cystoscopy in a retrograde manner in female cats and dogs. This requires expensive equipment and a step learning curve. They can be passed in an antegrade fashion via a ventral midline celiotomy. This is the general method for placement. The kidney is isolated and a over the needle catheter is placed into the lateral surface of the kidney. A guide wire is placed down into the urethra and kidney. A mini cystotomy is performed to find the guide wire and the stent passed over the wire. The wire is removed.
The technique is difficult in the stents are difficult to handle and require a steep learning curve to place. The procedure of placing the stent is longer which has been suspected to increase mortality in these patients. Given this the success rate of stent placement is around 95%. Cases with a stricture of the ureter have a higher chance of complications. For this reason ureteral bypass is recommended for cases with a stricture.

Subcutaneous ureteral bypass (SUB) is an indwelling device that completely bypasses the ureter and drains into the kidney. The device is placed via a ventral midline celiotomy. A locking loop nephrostomy tube is inserted into the caudal pole of the kidney. This is performed by directing an 18 gauge over the needle catheter in to caudal pole of the kidney aimed at the pelvis. The position is confirmed with fluoroscopy ideally. A guide wire is then gently passed into the catheter taking care not to damage the renal pelvis and perforate the opposite side. The locking loop catheter is then passed over the wire and into the pelvis of the kidney. This is confirmed with fluoroscopy. The Dacron cuff is then sutured onto the renal capsule to prevent leakage and help secure the catheter. The cystotomy tube in placed through a stab incision in the apex of the bladder and secured in place with a purse

string suture. These tubes are then passed through the ventro-lateral musculature of the body wall into a gently created subcutaneous pocket. These tubes are then connected to the port and the celiotomy closed up. The surgical time is shorter than placement of a ureteral stent. There is an easier learning curve to use the implants. Recent studies have shown a lower complication rate and mortality rate than seen with other alternatives. The long-term results are not certain, as this is a new device with the longest one in place for 6 years at the time of writing this. However two studies performed by independent people apart from the developers have shown excellent results in a large number of cats. The system can then be flushed out every 3-6 months via the port to prevent more stone formation.
There are a few complications seen with the SUB leakage at the nephrostomy/cystostomy tube or shunting port is most common and is seen in less than 5%. The newer design has all but eliminated this and I have not seen it in any of the clinical cases I have done. Haemorrhage during nephrostomy tube placement occurs in less than 1% of cases. This can lead to occlusion of the system with a blood clot. Obstruction can occur due to debris, purulent material or stones. This can be prevented with the recommended flushing. The catheters can kink during placement. Urinary tract infections can occur but can easily be treated.

Extracorporeal shock-wave lithotripsy is a commonplace therapy in human medicine. It can be used in animals but is expensive and difficult to get access to. A stent should still be placed to aid in drainage and removal of debris post therapy. The author has no clinical experience with this.

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ARTIFICIAL URETHRAL SPHINCTER PLACEMENT FOR URINARY INCONTINENCE

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ABSTRACT

Urinary incontinence is a common disorder mainly of the spayed female dog. It is generally caused by a loss of tone of the proximal third of the urethra leading to an intermittent incontinence. However it can be due to a multitude of other factors that need to be ruled out. The common term is urethral sphincter mechanism incontinence. The initial step of USMI is medical management with α-adrenergic drugs or oestrogens. If medical management fails surgical treatment is the next step. An intensive investigation should be undertaken prior to surgical treatment to rule out all other causes for the incontinence. In the past surgical management has provided poor results of around 50% return of continence. With the advent of new techniques the successes rates have increased into the 90% range. Correct patient selection is essential in the decision to correct USMI surgically and with what technique.

INTRODUCTION

Urinary incontinence is a relatively common condition affecting mainly large breed spayed female dogs. The condition is often seen in middle age to older dogs. Until now the ability to deal with this condition proved frustrating and unrewarding.

The causes of incontinence are linked to many underlying conditions. These may be neurogenic abnormalities, anatomic outflow obstruction, hormonal responsive such as urethral sphincter mechanism incontinence (USMI), inflammation, anatomic abnormalities such as ectopic ureters or behavioural. For the purpose of this communication we will discuss USMI this will include patients that have had corrective surgery for ectopic ureters and are still incontinent due to a poorly developed bladder neck. We will discuss USMI in regards to the female patient, which is the most common presentation. However USMI and ectopic ureters can occur in male patients, be it very uncommon management is similar.

The entire urethral wall is made up of smooth muscle in a circular fashion. This is 25% thicker in the proximal urethra. In the distal third of the urethra there is an interdigitating of the smooth muscle with the striated muscle of the urethralis muscle making up the so-called external sphincter. Urine continence is maintained by a number of factors working together in the pelvic canal. These factors include the tone of the smooth muscle in the urethra, the tone of the striated muscle, the natural elasticity of the urethral tissues, the length and diameter of the urethra and the degree of engorgement of the sub-urethral plexus.

There are a number of factors that predispose to USMI, however, the role-played by each of these factors and the importance of each is unknown. This explains why surgical treatment of this condition has proved to be frustrating and often not effective. The identified factors are urethral hypoplasia, intra-pelvic bladder, brick or gourd shaped bladders, obesity and ovahiohysterectomy (OVH). Incontinence post ectopic ureter surgery is suspected to be from the fact that the bladder neck did not form properly due to the intra-mural ureter in the bladder neck. This leads to incontinence that can be managed as for USMI. Female dogs have a significantly higher amount of collagen in the proximal third of the urethra when compared to male dogs. This is regardless of whether they have undergone OVH or not. However there is a significant change in the balance of the ratio between connective tissue and muscle of the proximal third of the urethra in spayed female dogs. The female urethra is shorter and wider than the male urethra, these factors are highlighted as the main reason most presenting dogs are females.

DIAGNOSIS

Full work up of the lower urinary tract should be done in patients that don't respond to medical management, patients with a lifelong history of incontinence, patients that don't respond to medical management or in the patient that the owner requests it.

Diagnosis of USMI is basically a diagnosis of ruling out all conditions that can lead to incontinence. These include urinary tract infection, urine pooling and ectopic ureters, even in an older dog. History is important in the diagnosis; generally these are middle-aged dogs with a history of OVH and incontinence starting any time from there. The incontinence is generally worse when the patient has been lying down sleeping. Patients with a lifetime history of incontinence may still have USMI but should be examined for other congenital conditions.

Ultrasound is essential in the first step of diagnosis. This allows one to look at the urethral neck, get a sterile urine sample for urinalysis and examine the pelvic cavity for any other abnormalities. Ultrasound can detect ectopic ureters in most cases but will not rule them out in all cases. The urinalysis should never be forgotten, as it would be very embarrassing to perform corrective surgery for a non-responsive incontinence when there is an active urinary tract infection. Once gross abnormalities of the lower urinary tract are ruled out and there is no urinary tract infection one can decide on two possible options for the next step. In an otherwise normal adult dog with a incontinence that has developed in adulthood that has had an OVH medical management can now be attempted.

Contrast radiography is the next step. Retrograde vagino-urethrogram is the best to evaluate the vagina, urethra, ureters and bladder. It entails retrograde injection of contrast through a Foley's catheter inserted just past the vulva. The cuff is then inflated and contrast injected under pressure. Radiographs are then taken while keeping the pressure on the Foley's. This allows assessment of the shape of the bladder, position of the bladder, length of the bladder neck. Excessive pooling of contrast can give an idea of urine pooling in the vagina. Retrograde vagino-urethrogram will easily detect ectopic ureters. However if ectopic ureters are expected from the history a computed tomography excretory urography (CT EU) should be chosen as the next diagnostic test. Computed tomography excretory urography allows the ureters to be assessed with no overlying tissue. If the results of the CT EU are non-diagnostic then a retrograde vagino-urethrogram should be performed to correlate with the results of contrast radiology.

Cystoscopy is only possible in the female patient in the veterinary setting. This entails the passage of a rigid endoscope into the vagina and urethral opening under direct visualisation. This allows examination of the vagina for urine pooling, examination of the mucosal surface of the urethra and neck of the bladder. One can often visualise the openings of the ureters in the trigone or vagina in the case of ectopic ureters.

MANAGEMENT

Medical management consists of treatment with sympathomimetic drugs such as the α - adrenergic stimulants or oestrogen supplementation. This is started on a trial basis with the dose being titrated to effect. This should be given at least 4 weeks to see if there is any response.

Surgical management of USMI and post ureteral ectopica surgery can be classified in the same group f procedures. In the past the outcome with these procedures was poor. The main goal of all of these procedures was to increase the resistance provided by the urethra or increase urethral length. The main concern with these procedures is the potential to make an incontinent animal dysuric from to much resistance in the urethra. The currently available surgeries that will be discussed here include urethral bulking agents, colposuspension, urethropexy, transobturator vaginal tape and artificial urethral sphincter placement. The general Feeling is that urethral lengthening procedures are very invasive and should only be used in the rare case of extreme urethral hypoplasia. This will not be discussed here.

Urethral bulking agents are injected via cystoscopy to narrow the urethral lumen and increase resistance. The most effected and safest agent is collagen. This shows good results with most owners reporting an increase in the continence score and less need for medication. The duration of the effect

is questionable and varies from 3 months to 15 months in some reports. This would obviously have to be repeated

Colposuspension has been one of the older techniques used for many years to manage this condition. It entails anchoring of the vagina or remnant form OVH to either side of the pre-pubic tendon thus suspending the urethra. This moved the bladder cranially and increases resistance in the urethra. The current feeling is that around 50% of dogs are continent after surgery. The remaining dogs required medical management after surgery to maintain continence. However these dogs seemed more responsive to medical management. Complications are few with colposuspension the main being to much resistance leading to dysuria.

Urethropexy entails relocation of the bladder neck to a more cranial position. Given that the bladder in secured to the body wall a kink is seen in the urethra, which is suspected to increase the resistance of the urethra. The success rate of urethropexy alone was about 57% similar to that of colposuspension. Some patients developed a gradual deterioration in continence and needed repeat surgery. When combined with colposuspension the outcome was considered good in 70% of patients.

Transobturator vaginal tape is a new technique with a cadaver and a small live study supporting it. The study showed that 6 out of 7 dogs remained continent after surgery and a major complication was seen in one dog. My understanding is that umbilical tape is passed paravaginal with the help of an episiotomy to surround the urethra and increase resistance. The results show promise and hopefully more will be done to advance this technique.

Artificial urethral sphincter placement is a new technique that shows great promise in maintaining continence. This entails placement of a silicone cuff around the neck of the bladder connected to a silicone tube. This connects to a port that one can access under the skin in the groin. This allows the level of resistance in the urethra to be adjusted with ease in the awake patient. This is a major advantage that makes a repeat surgery unnecessary. The volume of fluid can be adjusted with a simple injection into the port. Recent studies show 25 out of 27 dogs were continent at 2 years post surgery. No major complications were seen intra-operatively. The only complication was partial urethral obstruction that required AUS removal 5 and 9 months after surgery. This was similar to the initial study performed for development of the device where 92% of compliant owners dogs remained continent after surgery. Both studies have shown that 30% of dogs become continent after placement of the AUS alone with no inflation. One should only inflate the device 2-3 weeks after the surgery if needed. Placement of the device is simple and quick to do and can be done without extensive equipment.

As one can see there are many options for surgical management of USMI and the good news is that the success rates are increasing from 50%!

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ANTIBIOTIC RESISTANCE

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ABSTRACT

Antimicrobial resistance is one of the greatest challenges currently facing small animal veterinary medicine. During the past decade, various multidrug-resistant bacteria (MDR) have emerged and spread among dogs and cats on a worldwide basis. The major current MDR organisms of concern are methicillin-resistant *Staphylococcus pseudointermedius* and *Escherichia coli* producing extended-spectrum β-lactamase. However, these bacteria are just the tip of the iceberg because multidrug resistance has diffused in other common bacterial pathogens encountered in general practice, such as *Pseudomonas aeruginosa* and enterococci. Understanding why and how antimicrobial resistance develop is very important in everyday practice. Certain bacteria have intrinsic resistance for certain types of antibiotics and this should at all times be considered when planning the treatment protocol. Recent antibiogram data will also be discussed. Measures to prevent resistance should be implemented in all practices.

ANTIBIOTIC THERAPY[2,5,6]

The aim of antimicrobial therapy is to assist the host's specific and non-specific defense mechanisms in containing and eliminating invading microorganisms. The ability to do this is also enhanced when therapeutic drug concentrations are rapidly produced at the site of infection and maintained for a sufficient length of time. In doing so the pathogen's ability to replicate is reduced or eliminated.

Antimicrobials may be classified according to three basic features:

- Antibacterial Activity
- Bacteriostatic or bactericidal activity
- Time or concentration dependant activity

Antibacterial activity

Antibiotics can be considered as being narrow-spectrum if they inhibit only Gram + OR Gram – bacteria, whereas broad-spectrum drugs inhibit both Gram + and Gram – bacteria. Broad-spectrum antibiotics are more numerous these days and these terms are seldom used.

Bacteriostatic or bactericidal activity

Some antimicrobials inhibit the growth of a bacterium at one concentration, the minimum inhibitory concentration (MIC), but require a higher concentration to kill it, minimum bactericidal concentration (MBC). An antibacterial agent that exhibits a large difference between inhibitory and cidal effects is considered to be bacteriostatic drug. An antibacterial agent that kills the bacterium at or near the same drug concentration that inhibits the growth is considered to be bactericidal drug. Benzyl penicillin is bactericidal at at usual therapeutic concentrations and bacteriostatic at low concentrations. Other groups of antimicrobials also act in this manner is fluoroquinolones.

Time or concentration dependant activity

The most important factor determining the efficacy of time-dependant activity of certain types of antibiotics is the length of time that serum concentrations exceed the MIC of a given pathogen. Increasing the concentration of the drug several fold above the MIC does not significantly increase the rate of microbial killing. Rather, it is the length of time that bacteria are exposed to concentrations of these drugs above the MIC that dictates their rate of killing. It is for this reason that frequent administration is needed. Eg. beta-lactams, macrolides, tetracyclines, sulpha's.

Concentration-dependent antimicrobials' rate of killing increases, as the drug concentration increases above the MIC for the pathogen, and it is not necessary or even beneficial to maintain drug levels above the MIC between doses. Optimal dosing would therefore be high doses at long dosing intervals. Eg. Aminoglycosides, fluoroquinolones and metronidazole. Some antibiotics exert characteristics of both time- and concentration-dependent activity.

MECHANISM OF ACTION OF ANTIMICROBIAL DRUGS

Antimicrobial drugs can work in one of five ways:

- Inhibition of cell wall synthesis – beta-lactam antibiotics
- Damage to cell membrane function – polymyxins
- Inhibition of nucleic acid synthesis or function – quinolones
- Inhibition of protein synthesis – aminoglycosides, macrolides, tetracyclines
- Inhibition of folic acid synthesis – sulphonamides, trimethoprim

Knowledge of the different antimicrobial mechanisms of action is important when combining agents to determine their interaction with each other.

ANTIBIOTIC RESISTANCE[2,3,6]

The increasing frequency with which new resistant bacteria pathogens in veterinary and human medicine develop is of great concern worldwide. Therefore successful management of antimicrobials and the awareness of the mechanisms of resistance formation are critical. Several mechanisms can frequently be responsible for resistance to a single antimicrobial agent.

Antimicrobial resistance mechanisms can be classified into four major categories:

- Reducing penetration of the agent into the bacterial cell
- Excretion of the agent through efflux pumps
- Inactivation of antimicrobial agent by modification or degradation, either before or after penetrating bacterial cell
- Antimicrobial target can be modified or protected by another molecule, preventing access of the antibiotic to its target

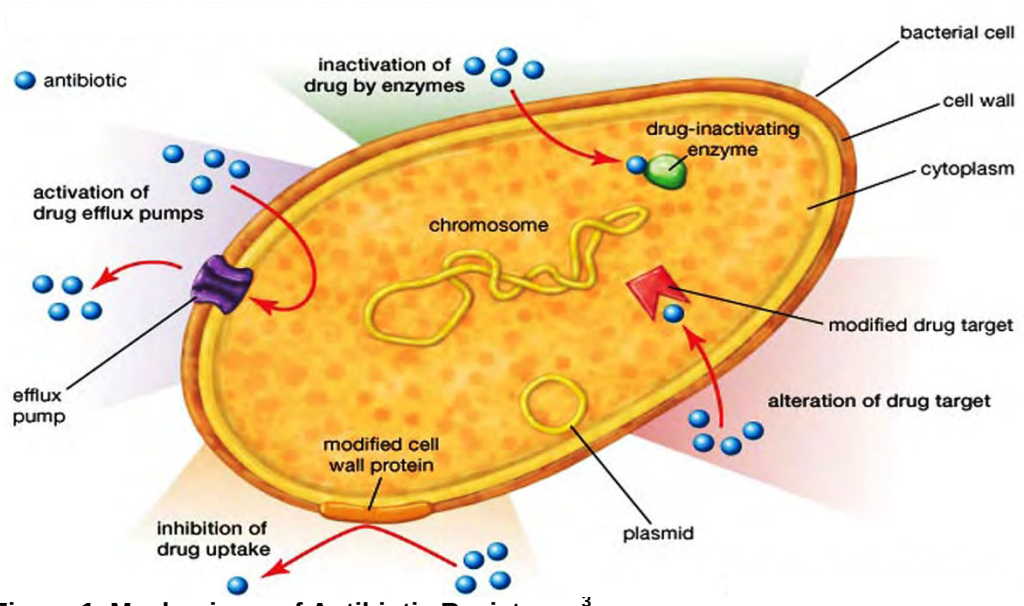

Figure 1. Mechanisms of Antibiotic Resistance[3]

Intrinsic Resistance[2]

Bacteria can display fundamental phenotypes of resistance to antibiotics. One of the most important phenotypes is intrinsic resistance. Intrinsic resistance is natural to all the members of a specific bacterial taxonomic group. This type of resistance is most often through structural or biochemical characteristics inherent to the native microorganism. For example, many Gram-negative bacteria are naturally resistant to the activity of macrolides since these chemicals are too large to cross the cell wall and to gain access to their cytoplasmic target. Knowledge of intrinsic resistance of specific organisms is very important, as it will influence our choice of antibiotic.

Bacterial antibiotic resistance can also result from the mutation of genes involved in normal physiological processes and cellular structures, from the acquisition of foreign resistance genes, or from a combination of these mechanisms. Mutations occur continuously but at a relatively low frequency in bacteria, thus leading to the occasional random emergence of resistant mutants. However, under conditions of stress, bacterial mutations can increase. This so-called mutator state has been suggested to be involved in the rapid development of resistance in-vivo during treatment with certain antimicrobials such as fluoroquinolones. However, for the majority of clinical isolates, antimicrobial resistance results from acquisition of extra chromosomal resistance genes.

Bacteria can acquire foreign DNA in three different ways;

- Transformation – uptake of naked DNA present in environment
- Transduction – transfer of DNA from one bacterium to another by bacteriophages
- Conjugation – transfer of plasmids between bacteria through a mating-like process

ANTIMICROBIAL SELECTION AND PRINCIPLES OF THERAPY[1,2,4]

The aim of antibiotic therapy is to assist the host's immune system in containing and eliminating invading organisms. The ability to do this is enhanced when therapeutic drug concentrations are rapidly produced at the site of infection and maintained for a sufficient time. Knowledge of when and where antibiotics work will make the selection much easier and efficient. In order to make the correct choice of which antibiotic to use, we should always do a bacterial swab for culture and antibiogram. This is not always possible and empirical treatment is then validated.

In making the decision as to which drug to use, the clinician should also keep in mind that bactericidal drugs are required

- For serious life threatening infections
- When host defences are compromised
- For infections of vital tissues such as CNS, cardiovascular and skeletal system
- Immuno-deficient or immune compromised animals.

For infections of a less severe nature, bacteriostatic agents may be more or as useful than bactericidal drugs.

Antibiotics should be classified as "Protected" or "Avoided" antibiotics in practice to ensure least resistant formation and to protect our future use and effectively of antibiotics

- Protected antibiotics – Macrolides, fluoroquinolones, 3rd and 4th generation cephalosporins
- Avoided antibiotics – Vancomycin, Carbapenems, Non-ocular chloramphenicol

REAL-TIME ANTIBIOGRAM RESULTS[7]

Antibiogram results to the most common bacteria will be presented on day of presentation.

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BRUCELLOSIS KNOWLEDGE, ATTITUDES AND PRACTICES OF CATTLE KEEPERS IN A RURAL COMMUNITY IN THE EASTERN CAPE, SOUTH AFRICA

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ABSTRACT

Brucellosis has been identified globally as a neglected zoonosis.[1] Poverty is a strong risk factor for brucellosis, especially in resource-limited areas that still rely heavily on livestock keeping for survival.[1,2] In South Africa, bovine brucellosis is currently not well controlled and poses a threat to both livestock production and public health.[3] The aim of this study is to determine the current Knowledge, Attitudes, and Practices (KAP) of cattle keepers, regarding bovine brucellosis, in a rural communal farming community in the Eastern Cape Province.

INTRODUCTION AND BACKGROUND

Brucellosis has been identified globally as a neglected zoonosis. Poverty is a strong risk factor for brucellosis, especially in resource-limited areas that still rely heavily on livestock keeping for survival. In South Africa, bovine brucellosis is currently not well controlled and poses a threat to both livestock production and public health. There is a paucity of verified and published information on brucellosis in South Africa.

The aim of this study is to determine the current Knowledge, Attitudes, and Practices (KAP) of cattle keepers, regarding bovine brucellosis, in a rural communal farming community in the Lukhanji municipal area of the Eastern Cape, South Africa. This study seeks to understand the zoonotic potential of bovine brucellosis in this community and to explore potential risk management and disease control interventions.

METHODS

An outbreak of bovine brucellosis occurred in 2008-2009 in the Lukhanji municipal area, and this was used as the sampling frame for our study. The 2008-09 outbreak was successfully eradicated through test and slaughter principles and extensive awareness campaigns on brucellosis were held in the affected area. The 9 positive villages from the 2008-2009 outbreak and 9 negative “control” villages were selected to participate in our study. A descriptive cross-sectional study was conducted from 6-10 March 2017 to investigate the current knowledge, attitudes, and practices among cattle keepers in the identified community regarding brucellosis and associated risk factors. A convenience sample of cattle keepers were interviewed at pre-arranged meetings in these villages using individual KAP questionnaires. The questionnaire was divided into 5 sections and includes questions on basic demographics; knowledge on brucellosis as a disease; attitudes towards animal health, human health and brucellosis; practices relating to meat and dairy consumption; and practices relating to cattle husbandry.

Knowledge, attitudes and practices regarding bovine brucellosis will be described using frequency tables as a representation of the Whittlesea community. KAP scores will be compared between villages. The Chi square test or the Fisher’s exact test will be used to test associations between categorical variables for statistical significance. Data will be compared between previously positive and negative control villages to detect any potential differences in responses. Data will also be compared according to whether individual brucellosis awareness communication has been received to detect any potential differences in responses.

RESULTS AND DISCUSSION

These will be discussed during the presenation

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RECOMMENDED READING

1. Department of Agriculture Forestry & Fisheries, 2016, *Discussion paper on the review of bovine brucellosis control in South Africa,* draft document. (final document to be published in the Government Gazette soon)

2. Department of Agriculture Forestry & Fisheries, 2016, *South African Veterinary Strategy (2016-2026),* from http://www.daff.gov.za/daffweb3/Branches/Agricultural-Production-Health-Food-Safety/Animal-Health/information/press-release

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HEIFER AND CALF NUTRITION: MUCH MORE THAN GROWTH

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ABSTRACT

For decades we have been spending efforts in making sure that we satisfy nutritional requirements of lactating animals, with, at times, ground breaking research that has changed and greatly improved the way we manage and feed dairy cows. We often talk about fine-tuning diets or precision feeding and if milk yield, milk composition, reproduction or health is not up to the genetic potentials we tend to blame or praise the nutrition. Exactly, what potentials are we talking about? We have started to realize that those are not only dictated by the DNA of the animal but they are set not only at breeding but also weeks after conception up to few days before milking. A central theme is therefore the identification of critical windows in development, during which the supply of nutrients to the whole animal, its tissues, organs and even cells plays pivotal roles in the expression of immediate and subsequent responses that affect processes involved in milking, reproduction and immune system. There are still many gaps in our knowledge, but new opportunities for developing a more holistic approach to the subject are emerging from the acceptance that, in addition to its fairly immediate effects on adult performance, nutrition at earlier stages may have consequences for the whole animal productive career. We can therefore try and do much more than just feeding the best possible ration during lactation.

HEIFERS AND CALVES NUTRITION

Assuming the foetus has an ideal uterine environment where the expression of the genetic material is optimized, we will focus on how nutrition after parturition can affect the long-term productivity of the future cow. Because ruminants are born with a lack of serum Igs, they completely rely on colostrum ingestion in the first few hours of life to absorb them among other components that are fundamental for their survival and future production performance. To maximise calf survival and growth, plasma immunoglobulin (Ig) status and thus colostrum management is therefore of utmost importance. The optimal level of serum IgG in calves is 10 g/L below which mortality rate increases[1,2] , hence, hindering of the absorption process predisposes the newborn to debilitating digestive and/or respiratory infections that may lead to septicaemia and early mortality[2,3]. After ingestion of colostrum, the intestinal epithelium of the newborn (unlike the placenta) has the capacity to absorb macromolecules until approximately 48 hours after birth allowing for transportation of Igs, growth factors and hormones into the newborn lymphatics and general circulation[3,4,5]. As demonstrated by Yang and collaborators[6] in a study in which the passive immune transfer and intestinal development on calves was evaluated comparing different feed sources namely colostrum, transitional milk and bulk tank milk, these processes are favoured by a high quality colostrum ingested the day of birth, leading to better Igs and growth factors absorption and increased intestinal crypt and villi development. According to Maunsell (2014)[7] in order to declare colostrum as high quality it has to contain at least 50 g/L total IgG with adequate fat (20-30%), protein (15%), vitamins and minerals levels. Additionally, it must have low bacterial contamination (<100.000 cfu/ml total bacteria count and <10.000 cfu/ml coliform count) and be free from colostrum transmissible pathogens (i.e. *Mycobacterium spp., Mycoplasma spp and Salmonella spp*)[7]. Colostrum plays therefore a fundamental role on the stimulation of adequate development of the post-natal animal. Establishment of well-developed and protective epithelium linen in the digestive tract ultimately leads to enhance feed efficiency, growth, puberty onset and younger age at first calving. Several nutritional strategies related to improvement of calf growth have been applied at diverse stages of their development. Pre-weaning use of milk replacements containing 20% protein and fat and fed on a proportion of 15% of body weight plus a high quality protein calf starter (19 to 20%) ration has shown to double calf's size by 2 months of age influencing average daily gain and growth rate[8]. From the literature, in general, positive effects have been found mostly through allowing suckling on increased weight gain, feed intake, development rate of the mammary gland, reduced age at puberty and earlier age at first calving (27 days earlier) and improvements on overall health, reinforcing the notion of applying high-quality nutrition to shape the productive future of replacements[9]. It has also been shown that antibiotics can be replaced by probiotics in milk replacers without affecting calf performance [10]. In

other cases, probiotics actually improved growth and health of calves affected by diarrhoea or respiratory diseases [11,12].

Even though there has been a lot of focus on feeding milk-fed calves, little research information is available regarding the best strategies for feeding post-weaned dairy heifers. During this phase our main objective remains making them grow consistently at a specific rate so they are ready for breeding and that they efficiently utilize the diets they are fed. As feed costs are the greatest expense for raising dairy heifers, nutritional strategies to encourage growth and development while improving feed efficiency will be beneficial for both animals and farmers. We well know how a growing animal does differ in terms of bones, fat and protein accretion, depending on how close to maturity the animal is. However, nutrition of the post-weaned animal is often referred to as a whole, without differentiating in relation to age and growth stage and puberty makes things more complex as well.

Similarly to lactating cows in various stages of lactation, nutrients' requirements of dairy heifers vary substantially during their 2 years of development. Especially during the first 6 months after weaning, heifers will differ from older ones not only because of different requirements but also because of different gastro-intestinal tract development and therefore different efficiency of nutrient utilization. Younger heifers will therefore require greater dietary nutrient concentration than older ones and, therefore, need to be fed differently. Starter intake does help to promote the growth and development of the rumen in calves, but making the assumption that weaned calves are fully functional ruminants is not correct. Therefore, continuing to pay close attention to how post-weaned heifers are fed will allow for the rumen to continue to develop and will maximize growth and development. When managed in conventional feeding systems, calves are typically provided ad libitum access to a high-energy grain concentrate alongside restricted quantities of milk. Early intake of concentrate is critical for rumen development, as rumen papillae development occurs in response to butyrate produced through fermentation of carbohydrates [13, 14]. Provision of forage has long been discouraged, out of concern that it will displace concentrate intake and, consequently, impair rumen development[15,16]. However, there is evidence to suggest that forage provision does not need to reduce concentrate intake[17,18] and, further, may positively impact ruminal environment, reducing acidity of ruminal fluid[17,19] and improving feed efficiency (Coverdale et al. 2004). Provision of chopped forage has also been noted to reduce non-nutritive oral behaviour of the calf [18, 20] suggesting that it may satisfy a motivation to perform oral foraging-type behaviour. Results of feeding hay seem to depend on the form and type of hay. The positive effects of hay intake on nutrient digestibility are reduced when hay is finely ground, suggesting that benefits of hay are, in part, due to its physical effectiveness [20]. It has also been shown that providing alfalfa hay may reduce concentrate intake, as calves consumed larger amounts of alfalfa hay compared to other types of hay, such as ryegrass [18]. It is interesting to note that when offered a choice of hay and concentrate, calves selected a proportion of hay ranging between 5 and 30% of total DM intake[17,18,21], depending on the type of hay provided and, potentially, other nutritional factors such as milk intake. Selection in favour of hay has been found to decrease after weaning, suggesting that calves may alter dietary selection patterns in response to energy requirements [21]. All these research results indicate that, in addition to provision of a high-quality starter concentrate, offering limited amounts of a physically effective fibre from forage (limited to 5 to 10% of total DMI) may also be ideal for calf growth and development.

A more intensive feeding approach should be continued post-weaning as well. Feeding rations that achieve a greater protein-allowable growth than energy-allowable growth helps to promote greater lean tissue accretion, improved frame growth and less risk for excessive body condition. This approach will cost more per day but will result in significantly fewer total days on feed, less total feed consumed over the heifer's growth and development, and actually results in a lower total cost of production [22]. Over-conditioned heifers can result in extra costs, calving problems, reduced fertility, premature culling and lower yields. On the other hand, reducing the length of the growing period by decreasing age at first calving below recommendations (22-24 months), by increasing pre-pubertal average daily gain (ADG), has been shown to negatively affect mammary development and first lactation milk yield[23,24]. In a summary of recent literature, it seems that first lactation milk yield would be maximized with a prepubertal ADG of 800 g/d for Holstein heifers [25]. Values will change based on mature body weight.

A typical dairy heifer is fed a ration in which the majority of its nutrition is derived from forages as opposed to concentrates. However, there is a large inefficiency associated with this method of feeding due to lower digestibility of most forages, greater metabolic protein and energy requirements

associated with digesting forage, and higher feed costs per unit of energy as compared to concentrates. To address this concept for raising dairy heifers, a series of experiments have recently been conducted to evaluate heifer growth characteristics and nutrient utilization when given 75% forage or 75% concentrate rations at restricted intakes to achieve a similar ADG. Increasing energy density, while restricting intake, has shown improved feed efficiency and no differences in development and first and second lactation yields [26]. Higher concentrate diet also resulted in higher nitrogen (N) retention and improved environmental N load, with lower manure output between 12 and 40%. In the same studies, feed costs dropped between 3 and 16%. The potential therefore exists to replace a significant proportion of the forage dry matter (DM) in a ration with concentrate DM, reducing the inefficiency associated with raising dairy heifers, between 4 and 22 months, while maintaining similar ADG. Unfortunately restricted diets have also resulted in reduced lying and resting time and increased aggressiveness probably due to lack of satiety[27,28]. These behaviours may result in negative health implications, like risk of hoof pathologies or sub-acute ruminal acidosis. One option is therefore to add a low-nutritive, low-value feedstuff to the diet that would satisfy the natural feeding behaviour patterns of limit-fed animals. Straw in the diet (10-20%) for example has shown to increase feeding time and meal duration and to target caloric intake for desirable weight gain and development while allowing heifers to engage in more foraging behaviour [29].

Studies on precision feeding heifers, since 2000, for improving feed efficiency and reducing nutrient waste have resulted in suggesting 14-15% CP for pre-pubertal heifers based on 2.15% BW DMI/d and 13-14% for post-pubertal heifers based on 1.65% BW DMI/d, and in both cases 30-35% of soluble CP. Research has also shown that added rumen undegradable protein (RUP) is of limited value to the heifer beyond of what found in common feedstuff. Rumen undegradable CP levels in excess of 25 to 30% are therefore not required so feeds specifically designed for high bypass protein can be avoided. We have mentioned how energy and intake should be monitored and recording weights is a must if heifers want to be fed successfully. Anytime a heifer is handled, or ideally once a month and always at the same time of day, heifers should be weighed to prevent rapid gains and fat heifers or gains lower than desired. In large herds, weighing a representative group may be sufficient.

Lifetime performance in dairy cows, and also other species, is influenced by early life development and dairy producers have the ability to manipulate this early life programming via nutrition. We now know that this manipulation must start immediately after birth and continue for at least 5 wk and must be in the form of liquid feed to have a positive influence on lifetime performance. Studies on post-weaned heifers have shown that these animals should be fed considering that their GI tract is not fully developed. However, feeding them high energy density rations has the potential to increase feed efficiency and reduce costs and nutrients waste, but behaviour needs to be considered by possibly adding low-nutritive feedstuff (e.g. 10-20% straw). Also, having homogenous growth rates and mature body weights allows to further reduce waste and ensure optimal feeding for all growing animals.

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COMPARISON OF IMMUNOLOGICAL RESPONSES IN ANCIENT AND MODERN TICK-HOST INTERACTIONS

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ABSTRACT

The objective of the current study was to compare tick counts, inflammatory cell counts and histopathological scores in skin biopsies from feeding sites of larval R. *decoloratus* and *R. microplus* ticks in Angus, Brahman, Nguni cattle to establish immunological responses at the tick-host interface of ancient and modern tick host associations. The Brahman breed displayed lower ($p<0.01$) tick counts compared to both the Nguni and Angus breeds. No differences between breed or tick species groups were observed within the number of cellular infiltrates or histopathology scores. It was concluded that a specific evolutionary relationship is not necessarily the primary contributor to the manifestation of the resistant phenotype and a high level of cross resistance is possible. Immunological parameters are important when assessing tick-host relationships, but the influence on the host includes a wider range of factors. The 12-hour interval is promising for further investigations, but higher intensities of infestation are recommended to increase the reliability of assessments.

INTRODUCTION

It has been well illustrated that local, cutaneous cellular responses play a key role in the bovine host's manifestation of resistance to infestation (Veríssimo *et al.*, 2008; Carvalho *et al.*, 2010; Constantinoiu *et al.*, 2010; Piper *et al.*, 2010; Marufu *et al.*, 2014). These previous studies have not only shown that the cellular responses between resistant and susceptible comparisons differ, but also that the animal's genotype, represented by breed, plays an important part in determining its response. Infiltrates of neutrophils, eosinophils, basophils, mast cells and lymphocytes contribute to the host's responses to tick attachment (Veríssimo *et al.*, 2008; Carvalho *et al.*, 2010; Constantinoiu *et al.*, 2010; Wada *et al.*, 2010; Marufu *et al.*, 2014). It is possible that certain breeds have a superior ability to maintain an extracellular matrix that is less susceptible to tick feeding. Recent studies have indicated that susceptible breeds have more pronounced subcutaneous changes than resistant cattle (Piper *et al.*, 2010; Marufu *et al.*, 2014). Piper *et al.*, (2010) also reported increased expression of collagen transcripts in the skin of indicine Brahman cattle compared to the Holstein Frisian cattle, which supported suggestions by Piper *et al.*, (2009) that Brahman cattle might possess an ability to modify the extracellular matrix in a way that increases resistance to infestation. Marufu *et al.*, (2014) associated the more severe cutaneous changes to differences to the type of hypersensitivity response of host, suggesting that the more severe necrosis and oedema is due to an acute, type I hypersensitivity reaction.

A comparison of ancient and modern parasite-host relationships could thus benefit from an assessment of the cutaneous environment and level of cellular infiltration during early infestation. Furthermore, previous studies included limited breed comparisons and focus was only on a single tick species, which could be a disadvantage in an attempt to elucidate host responses. As the majority of previous studies has focused on cellular reactions to *R. microplus* by B. taurus and B indicus breeds, there is a need to investigate the cutaneous changes between a wider scope of breeds and tick species. Parallel artificial infestation of *B. taurus*, *B. indicus*, and *B. t. africanus* breeds with both tick types *R. microplus* and *R. decoloratus* thus allow for the comparison of 'ancient' and 'modern' relationships while allowing for tissue sampling during the same phase of infestation over all treatment groups. The initial reaction from the host is essential for the manifestation of resistance (Tatchell & Moorhouse, 1970) and because there is less value in the assessment of ticks that have already successfully fed to maturity, sampling is taken at 12 h post infestation in order to assess the cutaneous reaction before or during possible tick detachment within the resistant hosts. Sampling of attachment sites at 12 h, although providing a good indication of the early host response to infestation, does not allow for determination as to whether the attachment would ultimately be a successful one. Hence interpretation is limited to relating the characteristics of an attachment site at

12 h with the average success rate of the breed, tick type or treatment group. The study's objective is to compare cutaneous responses between treatments in order to assess if certain breed and tick species interactions can be regarded as specific to that particular relationship.

MATERIALS AND METHODS

The trial was conducted at the Agricultural Research Council Animal Production Institute (ARC-API) in Irene, which is located 25 53' 59.6" S 28 12' 51.6" E. Two tick species from the genus *Rhipicephalus*, subgenus *Boophilus*, were obtained from Clin Vet International Laboratory for use in the study. A total of 36 cattle (12 from each of Angus, Brahman and Nguni breed) were sourced from a selection of extensively managed farms. The Nguni and Brahman cattle came from Mpumalanga while Angus came from the Free State Province of South Africa, both of which are areas where the *R. microplus* and *R. decoloratus* tick species are endemic.

Approximately 100 unfed tick larvae (UFL) of *R. decoloratus* or R microplus were infested on each of the three breeds by means of calico feeding bags. Each breed group of the experimental animals was split in half, with six animals per breed undergoing artificial infestation with the *R. microplus* species while the remaining six were infested with the *R. decoloratus* species.

Using a disposable 5 mm biopsy punch, three skin biopsies were taken with a 5 mm diameter and a depth of 10mm. The three skin biopsies were, two from non-parasitized skin prior to infestation and one from parasitized skin from identifiable tick feeding sites 12 hours post-infestation. Biopsy samples were fixed in neutral buffered formalin (pH 7) for 48 h to ensure thorough fixation of the 5mm samples and processed according to routine histological techniques. Sample section analysis and cell counts were conducted under a light microscope by a technician blind to breed and tick type. Histopathology score was performed on H&E stained sections according to Piper *et al.* (2010).

The Linear Models procedure was used to perform ANOCOVA (Type III) analysis of the effects of treatments, breed and tick species on the respective square root transformed tick counts. The pre-infestation values were used as the covariates in all ANOCOVA models. The adjusted mean effects of treatments were determined using LSMEANS option and compared using Bonferroni t-tests. Correlations among variables were determined using the PROC CORR function. Associations between histopathology score and treatment groups were done using The FREQ Procedure. Due to low cell frequencies, P-values presented were calculated using Fisher's Exact Test.

RESULTS

There were no significant ($p>0.05$) differences in square root-transformed infestation site dermal cell counts between breeds. Table 1 presents the adjusted least square means as well as standard deviations for all breed groups. There were no significant ($p>0.05$) differences in the transformed dermal cell counts among breeds. The parasitized skin displayed a higher level ($p<0.05$) of dermal cell counts than the non-parasitized skin in all three breeds. Table 2 presents the least square means as well as standard deviations for skin samples before and after infestation within all breed groups. No significant ($p>0.05$) associations between histopathology scores and breed groups were observed within any of the time points. Table 3 presents the frequencies of histopathology scores between before and after infestation within all tick species groups.

DISCUSSION

The results observed in this study for total cell counts partially agree with observations by Marufu et al., (2014), who reported increased total cell counts in parasitized skin compared to normal skin within breeds. It can be assumed that the presence of the cells within the post-infestation time point is due to their role as inflammatory infiltrates in response to larval infestation in the host's skin. The presence of infiltrates within parasitized tissue is not surprising considering the presence of leukocytes at infestation sites has been well documented (Veríssimo et al., 2008; Carvalho et al., 2010; Constantinoiu et al., 2010; Wada et al., 2010) and their presence will likely have an influence on the tick's ability to infest and feed. However, a unique aspect of the current study is the 12-hour post infestation sampling time point. The high density of infiltrates is thus an indication that the host response, and possibly the determining factor between successful or unsuccessful attachment, is already activated at very early stages of infestation. The results support early suggestions that the

larval stage of the life cycle is the most critical for the manifestation of resistance (Roberts, 1968; Kemp et al., 1976). It is not possible to distinguish between which of the sampled infestation sites would have developed into a successful tick feeding episode, but if the cellular differences between susceptible and resistant hosts are to be elucidated, it is possible that the 12-hour post infestation point could provide the optimal platform for comparisons of responses.

No differences in the total cell counts were observed between breeds and among tick species in the current study, a finding which was not expected. It is important to note that the cellular composition of infiltrates play a key role in the host's resistance or susceptibility to ticks, as neutrophils and eosinophils have been associated with increased susceptibility to ticks (Wada et al., 2010; Marufu et al., 2014), while mast cells and basophils have been shown to increase resistance (Veríssimo et al., 2008; Carvalho et al., 2010; Marufu et al., 2014). It is thus important to note that differential assessment of infiltrates will provide an improved insight into the cellular influences at a cutaneous level compared to the total cell counts. The current results did, however, disagree with observations by Marufu et al., (2014) who reported higher levels of total cell counts in the more susceptible cattle breed than the more resistant counterpart. Findings from the present study may be an indication that skin sections are not always an accurate representation of a tick-host interaction. It should be borne in mind that sampling was conducted early on in the infestation sequence. Damage due to tick mouthparts will likely be very little at this time point, so a difference between the tick species might be clearer at a later stage of infestation.

CONCLUSION

The lack of differences between breeds or tick species does not support the hypothesis that immunological reactions are influenced by the tick-host association. Possible differential reactions in parasite-host relationships that have had the advantage of co-evolution is therefore considered inconclusive. Regarding the 12-hour time point, the possible increase in the biological accuracy of elucidating the cellular response of the resistant host has a corresponding increase in risk of sampling error. The earlier the time point, the less pronounced the formation of the tick feeding lesion will be and the greater the risk of a section that is a poor representation of the tick-host interaction

Table 1 Infestation site dermal cell counts (LS means and SD) for all breed treatment groups

Parameter	Brahman	Nguni	Angus
Dermal cell counts(SQRT)	46.79 ± 7.54	44.79 ± 3.64	46.01 ± 7.92

a,b Means with different superscripts in the same row differ significantly ($p < 0.05$)

Table 2 Dermal cell counts for pre-and post-infestation samples (LS means and SD) within all breed groups

	Brahman		Nguni		Angus	
Parameter	**Non-parasitized**	**Parasitized**	**Non-parasitized**	**Parasitized**	**Non-parasitized**	**Parasitized**
Dermal cell counts (SQRT)	18.81 ± 3.62[b]	42.18 ± 7.54[a]	18.35 ± 13.47[b]	44.86 ± 3.63[a]	20.31 ± 13.81[b]	43.10 ± 7.92[a]

[a,b] Means with different superscripts in the same row differ significantly ($p < 0.05$)

Table 3 Frequency of histopathology scores between breed groups within pre- and post-infestation time points.

Time point	**Non-parasitized**			**Parasitized**		
P-Value	**p<0.34**			**p<0.98**		
Histopathology score	**Brahman**	**Nguni**	**Angus**	**Brahman**	**Nguni**	**Angus**
0	7	9	6	1	1	1
1	4	1	4	1	2	2
2	0	1	2	3	2	2
3	0	1	0	5	7	7
4	0	0	0	1	0	0
5	0	0	0	0	0	0
6	0	0	0	0	0	0

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CRYPTOSPORIDIUM, AN UPDATE ON DIAGNOSIS, TREATMENT AND CONTROL IN SOUTH AFRICA.

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ABSTRACT

Many cases of *Cryptosporidium* have been reported to the Faculty of Veterinary Science, Onderstepoort in recent years, in wildlife, bovines, ovine and caprine neonates. Severely affected individuals often succumb to hypovolaemic shock as a result diarrhoea-induced dehydration. Diagnosis and treatment can be frustrating. In this article, diagnostic tools and treatment options are discussed.

INTRODUCTION

Numerous *Cryptosporidiosis* cases have been reported to the Production animal clinic of the Onderstepoort Veterinary Academic Hospital (OVAH) recently involving neonatal antelope, bovine calves, lambs and kids, indicating an increased incidence of *Cryptosporidium* associated cases in recent years.

Cryptosporidium spp. are more closely related to protozoa than coccidia[4]. Two species, *Cryptosporidium parvum and Cryptosporidium andersoni* have been isolated from cattle. The bovine *Cryptosporidium parvum* genotype also infects other mammals, including humans making it an important zoonosis, especially to immunocompromised people[11]. There seems to be a number of genetically distinct intraspecies variants of *Cryptosporidium parvum* many showing host specific tendencies[8].

Cryptosporidium spp. have *a* direct life cycle. In cattle, *Cryptosporidium parvum* invades the superficial cells of the intestinal mucosa, while *Cryptosporidium andersoni* invades the superficial cells of the abomasum. Asexual and sexual stages occur extracytoplasmically within enterocytes[8]. Individuals become infected by oral ingestion of encysted, sporulated oocysts, which had been excreted in the faeces of infected cattle that then contaminated food or water sources. *Cryptosporidium* spp. oocysts can survive for long periods in the environment, but are susceptible to high temperatures and desiccation[8].

VETERINARY SIGNIFICANCE

Calves are usually infected between 7-30 days of age, with infections lasting up to 14 days[9]. *Cryptosporidium* spp. induced diarrhoea is mainly the result of malabsorption with intestinal epithelial injury characterised by villus atrophy, epithelial brush-border absorptive surface occupation by organisms and inflammatory mediator induced mucosal secretion[1]. Affected calves present with a severe watery to mucoid diarrhoea, depression, anorexia and dehydration[1]. In severe cases, affected neonates succumb to dehydration with cardiovascular collapse. Other enteric viral and bacterial pathogens may result in co-infection with cryptosporidiosis complicating diagnosis and treatment. At OVAH, enteric *E. coli* is most commonly isolated in the affected cases.

Cryptosporidiosis affected neonates may take up to six weeks to fully recover, impacting negatively on growth and weight gain. *Cryptosporidium* spp. cases are more commonly associated with intensive farming systems and less commonly seen under extensive conditions[8]. Individuals with poor colostrum intake within the first 24 hours of life are at increased risk as colostrum derived antibodies reduce the infection rate by blocking the invasion and inhibiting adhesion to host intestinal epithelium[8]. Any immunity compromising stressor within the first month of life will also predispose to clinical

infection. In Canada, calves born in selenium deficient herds, showed higher cryptosporidiosis associated mortalities, suggesting that certain trace elements might play a role in the immune status and clinical outcome in these calves[6].

DIAGNOSIS

Cryptosporidium spp. should be a differential diagnosis for any ruminant neonate between 7 days and 4 weeks of age that present with severe diarrhoea.

The *Cryptosporidium* spp. oocysts can be identified on a faecal float, especially if dichromate flotation fluid is used[13]. A faecal smear stained according to the safranin-methylene blue technique may also help to identify *Cryptosporidium* oocytes[7]. At necropsy, samples of intestine should be collected on ice for bacterial culture to rule out enteric bacterial pathogens and to exclude bacterial co-infection. Faecal samples can be sent for electron microscopy for identification of *Cryptosporidium* oocysts and enteric viral pathogens such as Rota- and Corona virus. Samples of intestinal mucosae, especially from distal small intestine, colon and caecum should be submitted in 10% buffered formalin for histopathology, as histopathology has a high sensitivity and specificity for *Cryptosporidial* infection, provided the infected regions were included during sampling. (J. Steyl pers. comm).

CONTROL

Contamination of water sources with *Cryptosporidium* spp. oocytes is a potential major source of infection, thus preventing faecal contamination of water sources is imperative in the face of an outbreak. Similarly, to coccidial infections, the practice of pasture fertilization using fresh animal manure or irrigation with slurry is also a source of infection. *Cryptosporidium parvum* oocytes are relatively stable in the environment. At low temperatures oocysts can survive in soil, water or faeces for >12 weeks, with degradation of oocytes accelerating at temperatures exceeding 25°C[8]. *Cryptosporidium* oocytes are deactivated at temperatures below -20°C and above 55°C, and are able to survive the fermentation process within silage[2]. Oocysts are strongly resistant to most commonly used disinfectants and chlorination of water, making sterilizing of intensive neonatal holding pens and other equipment difficult.

Presently there is no effective vaccination protocol for the control of *Cryptosporidium.* In addition to basic hygiene and cleanliness of neonate housing facilities, ensuring that neonates ingest adequate volumes of good quality colostrum within the first 24 hours of age is essential. Faecal waste management is important to limit exposure doses within a herd or flock. The isolation of any infected animals from the rest of the herd or flock is critical to limit the spread.

TREATMENT OPTIONS

Most young animals severely affected with cryptosporidiosis succumb due to the effect of dehydration leading to hypovolaemic shock. Fluid therapy is essential in the treatment of these cases. If dehydration is mild to moderate (5-8% dehydration), oral dosing of fluids can be attempted. Electrolytes can be added to water and dosed to the patient *per os*. If the level of dehydration is >8%, intravenous fluid administration is warranted. Dehydrated neonates will often suffer from metabolic acidosis. If facilities are available to test venous blood gas (VBG) to assess the level of metabolic acidosis, this is useful to monitor response to treatment. If VBG indicates metabolic acidosis is present, Sodium Bicarbonate (NaHCO3) should be administered intravenously.

The amount of $NaHCO_3$ that needs to be administered can be calculated by:

Bicarbonate (mmol) =Body weight (kg) x 0.6 x Base deficit (mmol/L)

Lactate levels can also be used to assess response to fluid therapy and prognosis.

Many chemotherapeutic drugs have been attempted for the treatment of *Cryptosporidium* spp. but very few have shown promise. To date, anti-coccidial drugs have been used with limited success. Decoquinate (Deccox, Zoetis) administered at 2.5mg/kg/24 hours for the first 15 days of life has proven to be relatively effective in one study[10]. Azithromycin and clarithromycin has been used to treat

children and affected animals with mixed success[3]. Tulathromycin (Draxxin, Zoetis) belonging to the same group of antibiotics may be an alternative treatment option.

Halofuginone lactate (Halocur, MSD) has been used with good success overseas at a dose of 0.1mg/kg OID for the first seven days of life. In a trial, treated calves shed significantly less *Cryptosporidium* oocytes and their mean growth rates were consistently higher than controls[12]. At present, Halocur is not available in South Africa, registration is pending, but could be imported under a Section 21 permit, if a laboratory has confirmed cryptosporidiosis on a farm.

CONCLUSION

There appears to be an increased incidence of Cryptosporidiosis in South Africa in recent years. Neonatal ruminants between 3-30 days of age are typically affected. Clinical signs include watery to mucoid diarrhoea, depression, anorexia and terminal hypovolaemic shock resulting from dehydration. Adequate ingestion of colostrum within 24 hours of birth and adequate hygiene of areas where neonates are housed within the first month of life are important prophylactic actions to consider. Treatment of affected individuals includes appropriate fluid therapy, controlling any enteric co-infections, and the use of Halofuginone lactate if available.

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Figure 1: Photomicrograph of cross sections of intestinal villi from a sheep lamb. Multiple basophilic round and crescent shaped *Cryptosporidium* spp. organisms (arrows) populating enterocytic brush borders. Note the absence of significant proprial inflammation. Haematoxylin & eosin staining. 400x magnification.

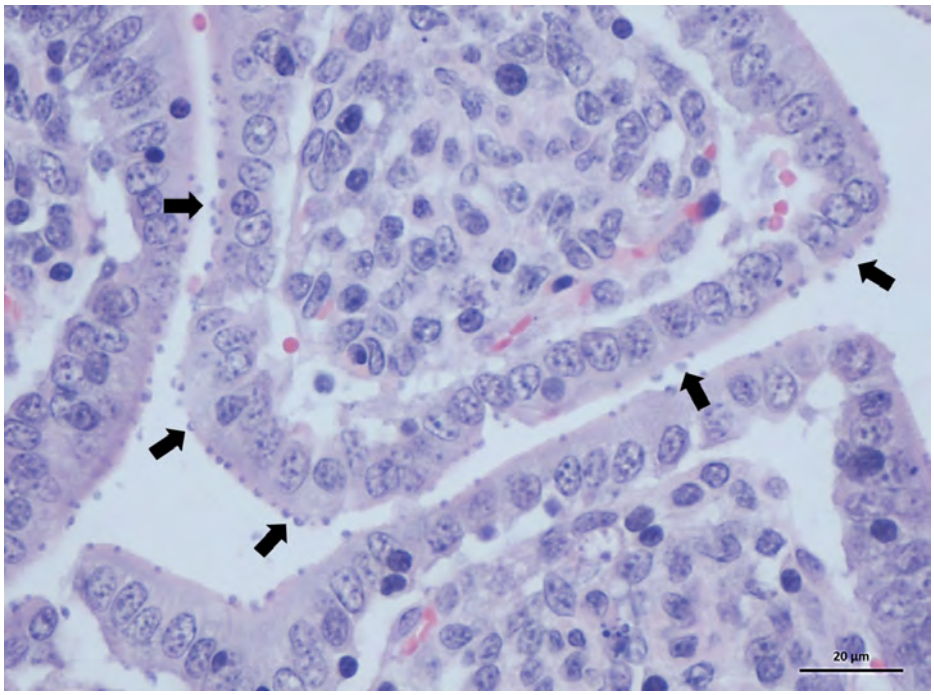
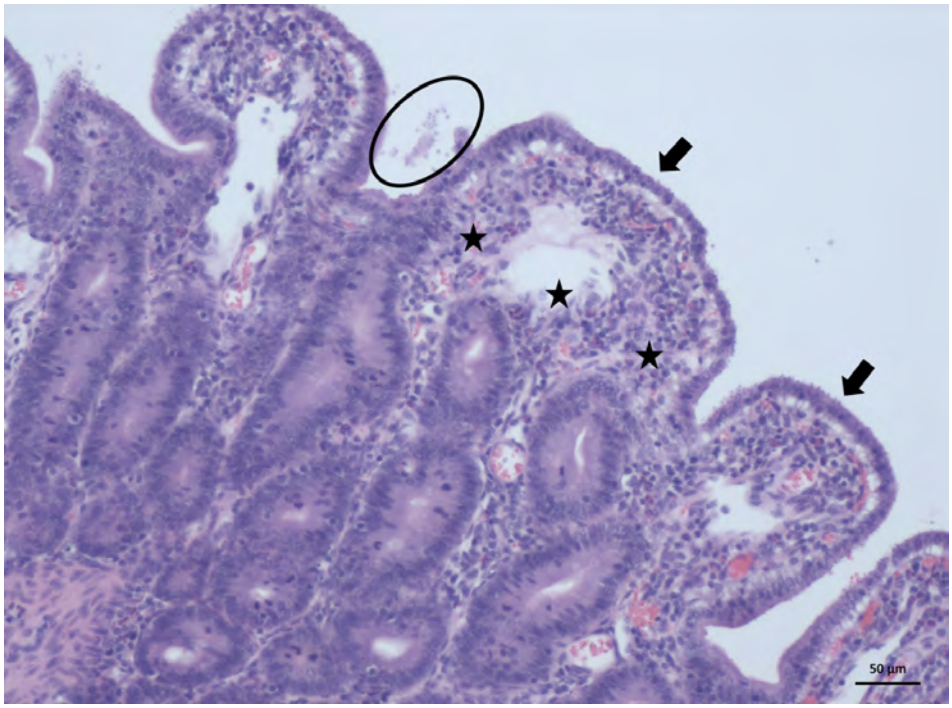


Figure 2: Photomicrograph of intestinal mucosae from a sheep lamb suffering from cryptosporidiosis. Marked mucosal villous atrophy (arrows) and fusion (stars). Note the presence of small basophilic round organisms populating the enterocyte brush borders, some of which are free in the lumen (circle). Mucosal glandular epithelium shows evidence of immaturity characterised by increased mitotic activity and lack of goblet cell maturation. Haematoxylin & eosin staining. 100x magnification.



NUTRITION OF THE DAM DURING PREGNANCY AFFECTS FUTURE OFFSPRING PERFORMANCE

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ABSTRACT

The increased animal production due to more intensive farming and strong genetic selection, has led to the need of more fine-tuned diets for each stage of the production cycle. Lately, more attention has been payed to the specific nutrient requirements during pregnancy on the base of the recently developed concept of foetal programming. In fact, there is growing evidence that foetal programming can alter postnatal development, growth, and disease susceptibility of the offspring. In ruminants, the main "critical windows" for foetal programming have been identified. The best strategy to obtain highly productive offspring is certainly to provide adequate dam nourishment during the all pregnancy in order to meet the specific requirement of each critical window. However, when the conditions do not always allow to provide the best diet, several strategies, when applied timely, can be used to aid in preventing or compensating the negative effects induced by undernourishment of dams during pregnancy. In conclusion, taking advantage of pregnancy as the first available and possibly most influential developmental window by providing and/or reinforcing adequate nutritional and energy requirements will positively impact future offspring performance by unlocking full genetic potential.

PREGNANCY AND NUTRITIONAL REQUIREMENTS

Fertilization can be regarded as the event that marks the start of a new organism. In the developmental process, by which the new organism growths and matures in preparation for extra uterine life, three stages (trimesters) are classically recognized.

During the first trimester, life supportive systems develop within the uterus in order to provide nutrients, oxygen and protection for the growing embryo. Recognition of pregnancy occurs around 17 days after mating, when the conceptus produces and liberates interferon *tau* (INFT), which contributes to *corpus luteum* (CL) development and progesterone production essential for pregnancy maintenance in bovines. By day 19, placentation starts and endometrial attachment will occur at about the end of the first month of pregnancy. At the same time, different organ systems start developing, the limbs appear by the end of the first month, hoof vestiges are present by day 60, by day 90 the four stomach chambers are developed and the scrotum is visible on ultrasonography.

By the end of the second trimester the foetus will weight approximately 4 to 5 kilograms, the testicles descend into the scrotum and the eyelashes are visible. Even though, by the end of the second trimester, the foetus is a completely developed organism, over 75% of the total foetal weight gain and growth will take place during the last trimester of gestation and especially between 240 and 280 days when pregnancy is about to terminate.

Allocation of energy and nutrients requirements for dairy cows during the dry period tends to focus on the transition period, because it represents the moment of higher metabolic demands. In fact, this period coincides with accelerated growth and weight gain of the foetus and the beginning of lactogenesis few days before parturition, considered as the start of the productive cycle.

This practice has led to neglect cows 'requirements during late lactation and early dry period, when the female is regarded as unproductive while, in fact, is gestating a calf that will represent an income for the enterprise, either as an abattoir-sell or as a replacement for the herd.

When referring to beef production, the focus becomes again the period near parturition in an attempt to improve dams' nursing ability and advance the probability of pregnancy during the next mating to fulfil the one calf per cow per year golden goal. Once more, nutritional requirements are often neglected over early and mid-gestation negatively affecting the offspring growth and development before and after birth adding rearing costs to compensate growth, organ and metabolic performance defi-

ciencies later during productive life. Moreover, most of the times, these negative effects are carried over to the next generation as epigenetic modifications often occur.

RECOGNIZING DEVELOPMENTAL WINDOWS

In the past few decades, the increased animal production due to more intensive farming and strong genetic selection, has led to the need of more fine-tuned diets for each stage of the animal production cycle. Furthermore, in the recent years the concept of foetal programming has been developed, stressing even more the need of highly fine-tuned diets. According to Nathanielsz and collaborators (2007), foetal programming can be defined as "The response to a specific challenge to the mammalian organism during a *critical developmental time window* that alters the trajectory of development qualitatively, quantitatively, or both, with resulting persistent effects". In animal and biomedical science, there is growing evidence that foetal programming can alter postnatal development, growth, and disease susceptibility of the offspring.

During pregnancy, the main "*critical windows*" have been identified as follow:

- Placentation
- Immune system development
- Growth and attainment of puberty
- Muscle and fat development.

Placentation

Senger describes the placenta as a transient organ of pregnancy that provides an interface for metabolic exchange between the dam and the foetus. It starts developing soon after the recognition of the conceptus and, in cows; it is fully developed by 40 days (Senger, 2003). Placental surface growth, vascularisation and secretion of growth factors never cease during pregnancy paralleling increasing foetal demands. Thus, it is clear how nutrient restrictions during this phase can dramatically affect placentation and foetal development. In fact, it was observed that nutrient restriction during early gestation, followed by re-alimentation during d 125 to 250 affected placental angiogenesis caused decreased development in the cotyledonary and caruncular portions *(Vonnahme et al. 2007*). Compromised placental development translates in reduced vascularity and uteroplacental perfusion. In studies on rats, in which the investigators used a model of reduced uteroplacental perfusion (**RUPP**), to restrict foetal growth during the last third of gestation, RUPP dams had reduced litter size, placental weight and individual foetal weight, confirming the restriction of intrauterine foetal growth.

These data clearly demonstrate the central role of uteroplacental blood flow in programming not only foetal growth but also cardiovascular function in offspring; furthermore, the negative effects of RUPP were observed also in F2 generation proving that these postnatal consequences are transmitted across generations (Anderson et al., 2005).

Transport of nutrient through the placenta increases throughout gestation primarily because of increased uteroplacental blood flow rather than increased nutrient extraction from each unit of blood (Reynolds et al., 2010). Thus, supporting the hypothesis that uteroplacental perfusion plays a central role in foetal growth.

Immune system development

In ruminants, passive immunity depends mainly on quality and quantity of colostrum available and on the new-born absorption capacity. However, nutrition of the dam during the late gestation will affect absorption of immunoglobulins G (IgG) in the offspring. In fact, linear decrease in protein intake during the last 100 d of gestation results in linearly impaired serum IgG concentrations in the calf, despite the fact that colostrum IgG concentration and amount of colostrum consumed by calves were not affected (Blecha et al., 1981). In another study, calves born to non-restricted cows but receiving colostrum from restricted dams tended to have lesser serum IgG concentrations at 24 h of life than the calves receiving colostrum from well-nourished cows (Hough et al. 1990). One of the reason could be find in the decreased colostral tri-iodothyronine concentrations exhibited by the nutrient-restricted mothers. In fct, tri-iodothyronine plays an important role in IgG absorption at the intestinal level (Boland et al., 2008).

Growth and attainment of puberty

At the dam level, Insulin Growth Factors (IGFs) influence maternal tissue growth and metabolism and, thereby, modulate nutrient availability for conceptus growth. At the placental level, IGFs regulate placental morphogenesis, substrate transport and hormone secretion. Apparently, increased IGF-I, secondary to maternal GH treatment in early pregnancy, increases maternal nutrient concentrations and sometimes increases foetal weight at the expense of maternal tissue mass *(Koch et al. 2010).* The effects of the IGFs on foetal growth must occur indirectly either affecting maternal metabolism and nutrient partitioning, and/or placental development and function, since IGFs and GH do not cross the placenta in significant quantities (Davenport et al. 1990).

The major factors controlling the onset of puberty are body weight and growth rather than age (McDonald, 1980). Several studies in cattle have demonstrated the importance of dam nutrition on growth, attainment of puberty and reproductive performance of the offspring. A recent study proved that heifers born to dams supplemented with protein during the last third of pregnancy had increased pregnancy rates compared with heifers born to non-supplemented dams (Martin et al., 2007).

Muscle and fat development

Skeletal muscle development is initiated in the embryonic stage (Cossu and Borello, 1999) during which, primary myofibres form during the initial stage of myogenesis. During this phase, maternal nutrition has negligible effects on foetal skeletal muscle development. The majority of muscle fibres form during secondary myogenesis in the foetal stage between 2nd month and 7th or 8th months of gestation in cattle (Russell and Oteruelo, 1981). Post-natally, muscle growth is mainly due to an increase in muscle fibre size rather than new muscle fibres formation (Karunaratne et al., 2005). Adipogenesis is initiated around mid-gestation (Muhlhausler et al., 2007), which overlaps with the period of secondary myogenesis. The amount of intramuscular fat is determined by the number and size of intramuscular adipocytes within foetal skeletal muscle. These are affected by maternal nutrition.

When considering dam nutrition, skeletal muscle and fat have less of a priority in nutrient partitioning during foetal development when compared with organs such as the brain, heart, and liver. As a result, skeletal muscle development is particularly vulnerable to nutrient availability (Zhu et al., 2006). Therefore, the effectiveness of nutritional management on altering marbling is fetal stage > neonatal stage > early weaning stage (i.e., 150 to 250 d of age) > weaning and older stages.

SEIZING THE OPPORTUNITY

The best strategy to obtain highly productive offspring is to provide adequate dam nourishment. However, especially in African countries, where the harsh conditions do not always allow to provide the best diet, several strategies, when applied timely, can be used to aid in preventing or compensating the negative effects induced by undernourishment of dams during pregnancy.

Protein supplementation of the maternal diet, for instance, proved to have a positive effect on neonates' birth weight, further attainment of puberty and first pregnancy rate when provided over the last trimester to grazing beef dams (Martin et al., 2007); the administration of vasoactive agents, such as Sildenafil, to induce vasodilation and increase nutrients, oxygen and hormones transport between mother and foetus has been used on nutrient-restricted ovine models resulting on greater foetal weight on supplemented ewes (Zoma et al., 2004); the use of amino acids such as Arginine and Methionine and/or B vitamines has been seen to improve blood flow into organs and stimulate tissue growth (Kwon et al., 2004); dams 'supplementation with conjugate linoleic acid isomers showed increase growth rate of the offspring (Cardoso et al., 2017 in press).

CONCLUSION

Taking advantage of pregnancy as the first available and possibly most influential developmental window by providing and/or reinforcing adequate nutritional and energy requirements will positively impact future offspring performance by unlocking full genetic potential. Improvement in carcass quality in the case of beef production or mammary gland development and milk production capability in dairy cattle plus enhancement of reproductive performance in both cases are amongst the effects to look forward to.

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DIFFERENTIAL EXPRESSION OF TICK-RESISTANCE RELATED GENES FOLLOWING ARTIFICIAL INFESTATION WITH *R. MICROPLUS* AND *R. DECOLORATUS* TICKS

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ABSTRACT

The objective of the current study was to conduct gene expression analyses using real-time PCR data of RNA extracted from skin biopsies collected 12-hours post artificial infestation with *R. microplus* and *R. decoloratus* ticks. Expression profiles of 17 previously identified immune function-related genes were studied in Angus, Brahman and Nguni cattle. The panel of genes included cytokines (*TLR5, TLR7, TLR9, TRAF6, CD14*), chemokines and their receptor (*CCR1, CCL2, CCL6*), toll-like receptors (*IL-1β, CXCL8, IL-10, TNF*) and other candidate genes (*BDA20, OGN, TBP, LUM, B2M*). The expression level displayed by the Brahman cattle differed significantly from those of the Angus cattle for genes *LUM, TBP, TRAF6* and *B2M*. Most of the differences were of genes encoding products of the extracellular matrix primarily involved in tissue repair. Important among which was *LUM*, and to a lesser extent *B2M,* which had expression levels significantly higher in the Brahman and Nguni cattle as opposed to the Angus cattle, thus presenting *LUM* as a potential biomarker for tick resistance. There was no evidence of breed by tick species interaction, implying that the effect of host-tick association was not responsible for gene expression post infestation.

INTRODUCTION

The devastating economic and environmental effects resulting from infestations by the cattle ticks (*Rhipicephalus microplus* and *Rhipicephalus decoloratus*) have been the driving force behind the global movement of the beef industry towards developing strategies with the potential to effectively combat tick burdens. While acaricides and vaccines have been the dominant tick control methods (Jongejan & Uilenberg, 1994), the host's resistance to ticks offers an opportunity that can be exploited to develop alternative tick control methods that can complement the existing methods. In previous studies, *Bos indicus* cattle were reported to have a higher degree of resistance than the *Bos taurus*, while *Bos taurus africanus* had intermediate resistance (Wilkinson, 1955; Frisch & O'Neill, 1998; Mapholi *et al.*, 2014). While some studies, reported significant within-breed variations in the expression of the extracellular matrix genes OBP, BDA20 and dendritic cell protein HFL-B5 between high and low resistance cattle (Wang *et al.*, 2007), others have reported significant between breed differences for toll-like receptors (TLR5, TLR7, TLR9, NFKBp50, MyD88, Traf-6, CD14 and IL-1b), chemokines and chemokine receptors (CCL2, CCL26, and CCR-1) between resistant and susceptible breeds (Piper *et al.*, 2008; 2009).

It has been hypothesized that breeds which may have experienced a long period of evolution in the presence of a particular tick species, and are resistant to that tick species, have accumulated genes affecting resistance to that tick species (Frisch, 1999; Marufu *et al.*, 2014). In South Africa, the Nguni (*Bos taursu africanus*) breed is thought to have a long term association and co-evolutionary status with the tick species, *R. decoloratus*. The Brahman (*Bos indicus*) breed, on the other hand, is thought to have long-term association and co-evolutionary status with the *R. microplus* in Asia. In contrast, the Angus (*Bos taurus*) breed lacks co-evolution or long-term term association with either of the two cattle tick species. Therefore, the study investigated the gene expression profiles generated at the host-tick interfaces of two closely related tick species in association with three cattle breeds of different origins. It was hypothesized that the ancient host–tick associations would result in the development of a more superior degree of resistance in these breeds against the tick species of common origin.

MATERIALS AND METHODS

The trial was conducted at the Agricultural Research Council Animal Production Institute (ARC-API) in Irene, which is located 25 53' 59.6" S 28 12' 51.6" E. Thirty-six cattle from 3 breed Angus (n = 12),

species *R. decoloratus* and *R. microplus* were infested on each of the three breeds by means of calico feeding bags. Animals from each breed were split in two, with six animals per breed undergoing artificial infestation with the *R. microplus* species while the remaining six were infested with the *R. decoloratus* species.

Three skin biopsies of 5 mm diameter and a depth of 10mm from the anaesthetized cattle. The three skin biopsies were, two from non-parasitized skin prior to infestation and one from parasitized skin from identifiable tick feeding sites 12 hours post-infestation. The biopsies were then directly placed into 15 ml tissue protect, RNase- and DNase-free tubes (CELLSTAR® tubes, Greiner Bio-One) and completely immersed in 5 ml RNAlater® RNA stabilization Reagent (Qiagen) and then transferred to -80^{o}C for archival storage in the reagent. RNA was precipitated, washed, cleaned and checked for quality according to Qiagen (2013). Using real-time PCR analyses, the gene expression profiles of the panel of genes listed in Table 1 were examined.

The threshold cycle (C_T) values generated by the ABI real-time cycler were used to calculate the expression level of each gene using the RT^2 Profiler PCR Array Data Analysis Webportal (SABioscience - Qiagen). The fold change value of each gene was calculated using the $\Delta\Delta C_T$ method (Livak & Schmittgen, 2001; Wong & Medrano, 2005). The mean and standard errors for each gene were generated in every treatment combination group. From this output, it was then determined whether each of the genes were up- or down-regulated, with mean fold regulation > 0 indicating upregulation and mean fold regulation values < 0 indicating down-regulation of the specific gene (Qiagen, 2015).

RESULTS

None of the genes expressed in the animal exhibited significant interaction between the main effects, breed and tick species. While the expression of the majority of the genes did not differ significantly according to breed, the expression levels of genes *TRAF6, TBP, LUM* and *B2M* were significantly different according to breed with P-values 0.039, 0.026, 0.012 and 0.023, respectively (Table 2). A Bonferroni pairwise comparison combined with a One-Way ANOVA of the breed types (Figure 1) revealed significant differences between the Nguni and Angus for *TBP* (P = 0.008) and *TRAF6* (P = 0.016), as well as between the Brahman and Angus for *LUM* (P = 0.003) and B2M (P = 0.007). None of the genes produced significant P-values (P > 0.05) for the main effect tick species.

Table 3 lists the mean fold regulation values for each gene in the respective treatment groups. In the treatment group Angus-*R. decoloratus*, genes *IL1B, IL10, CCL2, CCL26, CCR1, TLR5, TLR7, CD14, TRAF6, OGN, TBP, LUM* and *B2M* were upregulated following infestation, with genes *CCL2, CCL26, CCR1, TLR5, CD14, TRAF6, OGN, TBP* and *B2M* showing over-expression. However, genes *CXCL8, TLR9, TNF-α* and *BDA20* were downregulated with *CXCL8* and *TLR9* showing under-expression. Treatment groups Brahman-*R. decoloratus*, Nguni-*R. decoloratus* and Nguni-*R. microplus* exhibited similar expression patterns to the Angus-*R. decoloratus* group. The exception was that genes *IL10* and *TLR7* were downregulated in the Brahman-*R. decoloratus* group, while *B2M* was downregulated in the Nguni-*R. decoloratus* and Nguni-*R. microplus* groups. Treatment group Angus-*R. microplus* had genes *CCL2, CCL26, CCR1, TLR5, CD14, OGN, LUM, B2M* and *BDA20* upregulated, among which *CCL2, CCL26, CCR1, CD14, OGN, LUM, B2M* and *BDA20* were over-expressed. The genes *IL1B*, *CXCL8*, *IL10, TLR7, TLR9, TRAF6, TNF* and *TBP* were downregulated with *IL1B, TLR7, TLR9* and *TNF-α* showing under-expression. An expression pattern similar to that of group Angus-*R. microplus* was observed in the group Brahman-*R. microplus* with the only difference being that gene *CCR1* was downregulated while *TRAF6* and *TBP* were upregulated in the latter group. Also presented in Table 3 is the trend that the Angus treatment groups, more specifically the Angus-*R. microplus* group produced the minimum expression values for all, but two (*CCR1* and *CD14*) of the genes of interest, while the Nguni groups produced maximum values for most of the genes with no specification of the tick species.

DISCUSSION

No significant differences (P > 0.05) were observed in the expression patterns of all the genes of interest in cattle infested with either the *R. microplus* or the *R. decoloratus*. These two tick species have been shown to share numerous morphological characteristics (Jongejan & Uilengberg, 2004),

which might explain the lack of difference in their feeding signature and subsequently host gene expression profiles.

Piper *et al.* (2008) and Wang *et al.* (2007) support the data observed in the current study which shows significant differences in the expression profiles among breeds. Significant differences were observed between the Angus and Brahman breeds for genes *LUM* (P = 0.003) and *B2M* (P = 0.007). In addition, significantly different expression levels were detected between the Nguni and Angus for genes *TBP* (P = 0.008) and *TRAF6* (P = 0.016). While this is in contrast with what other researchers have reported, Piper *et al.*, (2008) also reported to have observed breed-associated differential expression of gene *TRAF6*. The genes encoding the extracellular matrix constituents, most importantly LUM and B2M, were upregulated at much higher levels in the high (Brahman) and intermediate (Nguni) resistance breeds than the genes involved in immune system regulation and inflammatory responses. This was consistent the results by Piper *et al.* (2010), where a microarray study showed upregulation of genes encoding constituents of the extracellular matrix in the tick resistant Brahman cattle in comparison to the susceptible Holstein-Friesian cattle. Furthermore, Kongsuwan *et al.* (2010) highlighted the importance of the epidermal permeability barrier of the skin as an important component of resistance in cattle against ticks, which explains the heightened expression of these genes in the tick-resistant Brahman cattle. Numerous studies describe the up- and down-regulation of chemokines and their receptors in inflammatory and autoimmune diseases (Navratilova, 2006). The expression profiles observed for the two chemokine ligands (*CCL2, CCL26*) and the one receptor (*CCLR1*) forming the panel of genes were consistent with those reported by Piper *et al.* (2008, 2009). All the three genes were upregulated in all six treatment combination groups, with the exception of *CCR1,* which was downregulated but stable in the Brahman – *R. microplus* group and stable in group Brahman – *R. decoloratus.*

CONCLUSIONS

There were more similarities than differences in the gene expression profiles of the different breeds and tick species studied. Furthermore, no important tick species × breed interactions that would suggest differences according to the co-evolutionary history of tick species and cattle breeds were observed. Nonetheless, breed variations only accounted for approximately 30 percent of the observed variation in gene expression in all treatment groups. This suggests that the majority of the differential gene expression profiles produced in cattle post infestation with *Rhipicephalus (Boophilus)* ticks was likely due to a complex array of other factors in addition to variations in breed and tick species.

Table 1: Description of the 17 genes of interest and their gene product functions

Gene Symbol	Gene name	RefSeq Number		Function of gene product
		UniGene	GenBank	
IL-1β	Interleukin 1, beta	Bt. 4856	NM_174093	Pleiotropic; pro-inflammatory
CXCL8	Interleukin 8	Bt.49470	NM_173925	Chemo-attractant for effector blood cells
IL10	Interleukin 10	Bt.4723	NM_174088	Anti-inflammatory
CCL2	Chemokine (C-C motif) ligand 2	Bt.2408	NM_147006	Recruitment and activation of immune effector cells; inflammatory response
CCL26	Chemokine (C-C motif) ligand 26	Bt.23451	NM_001205635	Recruitment and activation of immune effector cells; inflammatory response
CCR1	Chemokine (C-C motif) receptor 1	Bt.62596	NM_00107739	Recruitment of immune effector sells to site of inflammation
TLR5	Toll-like receptor 5	Bt.66307	NM_001040501	Pathogen recognition and activation of innate immunity
TLR7	Toll-like receptor 7	Bt.111931	NM_001033761	Pathogen recognition and activation of innate immunity
TLR9	Toll-like receptor 9	Bt.12810	NM_183081	Pathogen recognition and activation of innate immunity
CD14	Cluster of differentiation 14	Bt.4285	NM_174008	Confers lipopolysaccharide sensitivity to neutrophils, monocyte & macrophages
TRAF6	TNF receptor-associated factor 6	Bt.9201	NM_001034661	Mediates signal transduction from the TNF receptor family
TNF-α	Tumor necrosis factor – alpha	Bt.12756	NM_173966	Cell signalling protein (cytokine) involved in systemic inflammation
OGN	Osteoglycin	Bt.5341	NM_173946	Corneal keratan sulfate proteoglycan; regulates collagen fibrillogenesis in skin
TBP	TATA box binding protein	Bt.22662	NM_001075742	General transcription factor
LUM	Lumican	Bt.2452	NM_173934	Collagen fibril organization; epithelial cell migration; tissue repair
B2M	Beta-2-micropglobulin	Bt.64557	NM_173893	Formation of amyloid fibrils in some pathological conditions; presentation of peptide antigens to the immune system
BDA20	Bovine dander allergen 20	Bt.550	NM_174761	Weak inducer of both humoral and cellular responses

Table 2: P-values and R^2 values produced by the general linear model for the gene of interest when investigated for the main effects breed and tick species.

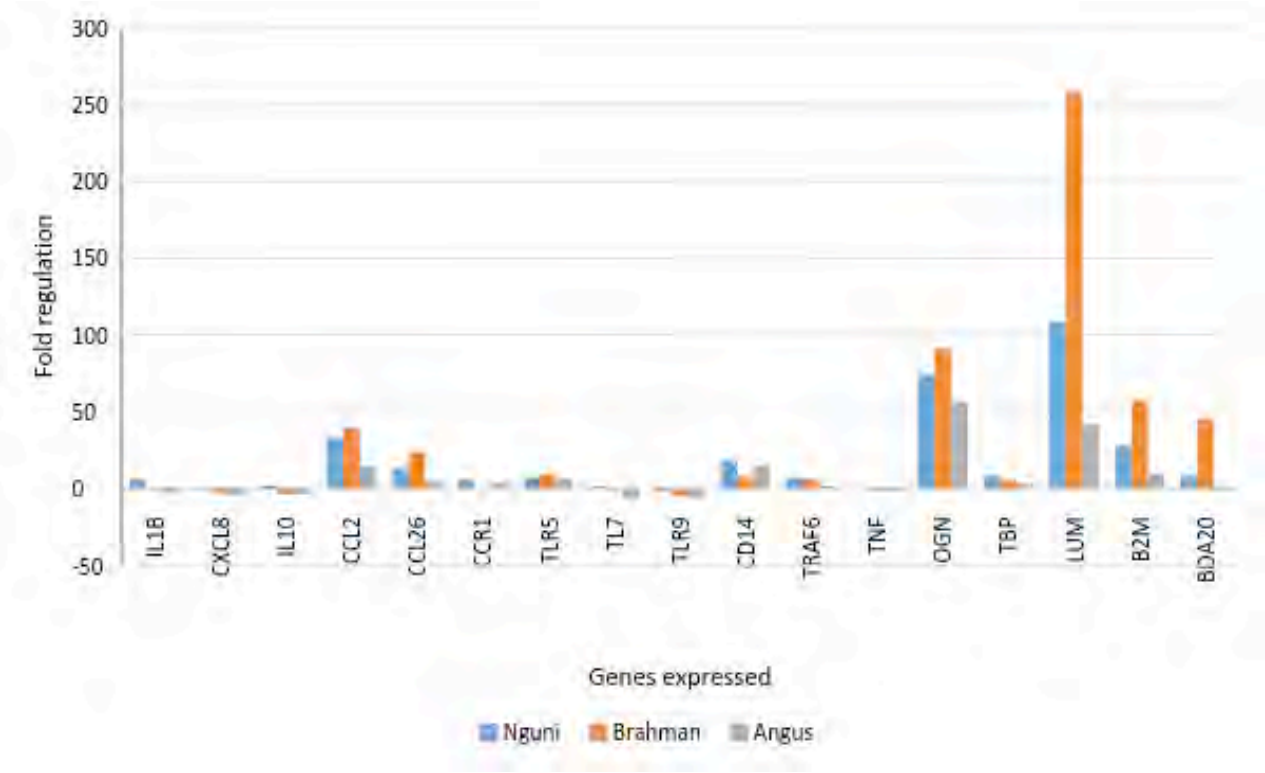
Gene Symbol	P-values		R^2-value
	Breed	Tick species	
IL1B	0.162	0.244	0.122
CXCL8	0.498	0.841	0.049
IL10	0.229	0.113	0.100
CCL2	0.402	0.164	0.063
CCL26	0.164	0.190	0.121
CCR1	0.419	0.809	0.060
TLR5	0.760	0.182	0.019
TLR7	0.172	0.349	0.118
TLR9	0.135	0.487	0.133
CD14	0.315	0.626	0.079
TRAF6	**0.039**	0.909	0.206
TNF	0.521	0.263	0.045
OGN	0.528	0.659	0.045
TBP	**0.026**	**0.053**	0.229
LUM	**0.012**	0.472	0.280
B2M	**0.023**	0.351	0.236
BDA20	0.115	0.244	0.126

Table 3: Mean normalised fold regulation values for 17 genes of interest in each of the six treatment groups

	Angus-*R. decoloratus*	**Angus-*R. microplus***	**Brahman-*R. decoloratus***	**Brahman-*R. microplus***	**Nguni-*R. decoloratus***	**Nguni-*R. microplus***
Host-tick associations	**Modern**	**Modern**	**Modern**	**Ancient**	**Ancient**	**Modern**
IL1B	0.578 ± 1.017	-6.075 ± 8.051	1.088 ± 1.215	-1.855 ± 2.799	8.600 ± 6.513[b]	3.700 ± 2.434[b]
CXCL8	-6.144 ± 2.724[a]	-1.810 ± 5.707	-3.852 ± 1.447[a]	-2.728 ± 0.384[a]	1.960 ± 4.885	-1.682 ± 1.190
IL10	1.440 ± 1.565	-9.590 ± 5.718[a]	-0.872 ± 1.159	-7.240 ± 8.456[a]	1.042 ± 0.622	2.593 ± 1.140[b]
CCL2	8.458 ± 1.137[b]	20.008 ± 14.723[b]	28.872 ± 9.652[b]	50.195 ± 31.677[b]	16.154 ± 7.806[b]	49.570 ± 27.943[b]
CCL26	5.622 ± 1.651[b]	4.365 ± 6.819[b]	13.610 ± 4.042[b]	34.388 ± 25.918[b]	8.236 ± 1.631[b]	18.442 ± 3.575[b]
CCR1	3.970 ± 1.309[b]	4.038 ± 8.707[b]	0.798 ± 1.487	-0.853 ± 2.191	3.540 ± 0.821[b]	7.850 ± 2.579[b]
TLR5	12.500 ± 6.521[b]	1.123 ± 4.208	11.128 ± 2.790[b]	7.095 ± 4.789[b]	6.338 ± 1.309[b]	8.713 ± 1.385[b]
TLR7	0.516 ± 1.127	-13.965 ± 9.647[a]	-0.844 ± 1.022	-0.518 ± 1.144	-0.112 ± 1.031	2.873 ± 0.767[b]
TLR9	-2.544 ± 0.867[a]	-10.005 ± 3.982[a]	-6.338 ± 2.704[a]	-3.585 ± 1.141[a]	-2.320 ± 0.378[a]	-1.547 ± 0.170
CD14	13.710 ± 3.127[b]	15.575 ± 12.499[b]	8.602 ± 1.865[b]	7.240 ± 3.032[b]	14.568 ± 1.864[b]	23.092 ± 8.015[b]
TRAF6	3.718 ± 1.896[b]	-0.702 ± 2.893	5.274 ± 1.168[b]	7.283 ± 5.018[b]	6.378 ± 0.577[b]	8.090 ± 1.993[b]
TNF	-0.308 ± 0.910	-2.895 ± 2.791[a]	-1.042 ± 1.053	-2.148 ± 0.468[a]	0.122 ± 0.677	-0.448 ± 0.548
OGN	94.832 ± 49.478[b]	19.308 ± 16.549[b]	86.660 ± 33.819[b]	95.198 ± 59.177[b]	58.086 ± 4.828[b]	90.150 ±15.200[b]
TBP	5.426 ± 1.216[b]	-0.405 ± 2.497	6.416 ±1.767[b]	4.743 ± 3.702[b]	9.372 ± 1.408[b]	6.898 ±0.952[b]
LUM	49.326 ± 21.347[b]	33.507 ± 15.499[b]	218.752 ± 52.470[b]	297.033 ± 172.467[b]	83.512 ± 16.906[b]	133.278 ± 46.208[b]
B2M	7.108 ± 3.057[b]	12.157 ± 6.034[b]	45.954 ± 10.639[b]	69.000 ± 43.197[b]	23.478 ± 6.944[b]	32.900 ± 14.198[b]
BDA20	-1.156 ± 1.462	4.227 ± 1.706[b]	17.444 ± 2.780[b]	73.210 ± 67.850[b]	8.176 ± 2.190[b]	8.428 ± 3.377[b]

[*]a: values below the cut-off threshold of -2 are categorised as under-expressed genes
[*]b: values above the cut-off threshold of +2 are categorised as over-expressed genes

Figure 1: LS means, using fold regulation as a measure of the expression levels of 17 genes of interest in the Angus, Brahman and Nguni following tick infestations.



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PREVALENCE OF METHICILLIN RESISTANT *STAPHYLOCOCCUS AUREUS* (MRSA) AMONG COMMERCIAL PIG HERDS IN SOUTH AFRICA

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ABSTRACT

Nasal carrier status of MRSA in pigs has been described elsewhere but is unknown in South Africa. To address concerns that exist over the zoonotic risk positive carriers pose to workers, the prevalence of methicillin resistant *Staphylococcus aureus* (MRSA) among twenty-five commercial pig herds in South Africa was determined. Of each herd, 18 finisher pigs' nasal contents were sampled at the abattoir, pooled into three pools and selectively cultured to determine the presence of MRSA. A herd was classified as MRSA positive if one or more of the three pooled samples cultured positive for MRSA. Three out of the 25 herds tested positive for MRSA, equating to a 12% herd prevalence (95% CI: 2.5 - 31%) among South African commercial piggeries. The prevalence of nasal MRSA carriers amongst large commercial pig herds in South Africa was low compared to what has been reported in other parts in the world and suggests a low zoonotic MRSA risk to workers in South African commercial piggeries and abattoirs.

INTRODUCTION

Methicillin resistant *Staphylococcus aureus* (MRSA) is one of the leading nosocomial human pathogens causing hospital-associated infections in humans worldwide (Bell et al., 2002; Dantes et al., 2013; De Kraker, Wolkewitz, Davey, & Grundmann, 2011; Hoyert & Xu, 2012; Köck et al., 2010). Nasal carriage is one of the major risk factors for developing staphylococcal infection (Peacock, De Silva, & Lowy, 2001). The ability of MRSA to acquire different mechanisms of antibiotic resistance through plasmids and/or chromosome cassettes (Lowy, 2003) realises its potential to be a truly multidrug-resistant potential pathogen.

Three MRSA epidemiological reservoirs are currently recognised: hospital associated (HA-MRSA), community associated (CA-MRSA) and livestock associated (LA-MRSA) (Köck et al., 2010). LA-MRSA, carried in pigs, is seen as a potential risk factor for people working with pigs as carrier status might be transferred to them. The first human case of LA-MRSA from pigs was detected in a Dutch hospital in 2004 from a six-month-old baby hailing from a pig farming family (Voss, Loeffen, Bakker, Klaassen, & Wulf, 2005).

Prevalence and association studies of LA-MRSA in both pigs and humans working with pigs have been conducted in several countries such as the Netherlands, Denmark, Canada and the United States over the past few years (Broens et al., 2011; de Neeling et al., 2007; Khanna, Friendship, Dewey, & Weese, 2008; Smith et al., 2009; Verhegghe et al., 2013; Voss et al., 2005; Wulf et al., 2008). De Neeling et al. (2007) screened 540 pigs from nine slaughterhouses in the Netherlands. The MRSA prevalence amongst the 540 pigs tested was 39%. A subsequent study concerning herd prevalence in the Netherlands among 202 herds, which were studied from 2007 to 2008, concluded that of 171 breeding herds, 67.3% were MRSA positive, and of 31 finisher herds, 71% were MRSA positive (Broens et al., 2011).

In South Africa, the prevalence of LA-MRSA in pigs and people working with pigs is unknown. Introducing LA-MRSA into human hospitals is potentially dangerous in a country with a high prevalence of HIV infections, which is one of the risk factors associated with an increased probability of acquiring an MRSA infection (Shisana, 2005). Since several studies elsewhere found MRSA in pigs and their caretakers, it was appropriate to determine the prevalence of pig carriers. This study aimed to determine the prevalence of LA-MRSA among commercial pig herds in South Africa.

MATERIALS AND METHODS

Study design

A cross-sectional survey with two-stage sampling was used. A random sample of 25 large commercial pig herds of over 500 sow units was selected to represent the country's commercial pig sector. From each herd pig nasal swab samples were taken at the abattoir to determine the herd's MRSA infection status. Prevalence among large commercial piggeries in South Africa was determined in terms of the proportion of large commercial pig herds, which were positive for MRSA. A herd was defined MRSA positive if one or more pooled samples were MRSA positive.

Sample size calculation

In order to calculate the number of herds needed to participate an expected prevalence of 50% was used with the following equation:

$$n = \frac{1.96^2 PQ}{L^2}$$ (Thrusfield et al. 2001)

$$n = \frac{1.96^2 * 0.5 * 0.5}{0.15^2}$$

= 42.7 herds

The population (N) of herds with ≥ 500 sow units in South Africa were only 56 herds; the calculated sample size, n, was larger than 0.1N. A new sample size n* was calculated as the reciprocal of $\frac{1}{n} + \frac{1}{N}$

$$\frac{1}{n*} = \frac{1}{42.7} + \frac{1}{56}$$

n* = 25 herds

Therefore 25 herds were to partake in this study.

Free Calc2 software (www.ausvet.com.au) calculated the sample size needed to determine the presence of MRSA in a herd using the weekly batches of pigs sent to the abattoir as the population size, an alpha value of 0.05 and a beta value of 0.05 was applied. The minimum expected prevalence was taken as 20% with a test sensitivity of 80% and specificity of 100%. The sample size calculated to detect freedom of colonization among the largest batch of 2 500 pigs from a 5 000 sow-unit was 18 pigs.

Sample collection

The nasal contents of 18 finisher pigs per participating herd was taken at the abattoir between stunning and sticking. A single sterile swab (Copan Transystem®, Copan Diagnostics Inc, Murrieta, USA) was used to collect the contents of both nares per pig.

Laboratory procedures

Each herd's 18 samples were pooled into three pools of six swabs per pool. The pooled samples were directly swabbed onto chromID™ MRSA agar plates (bioMérieux SA, Marcy-l'Etoile, France) used to screen for MRSA. With each batch of chromID™ MRSA agar plates processed, one plate was inoculated with an authentic MRSA strain as a positive control. These plates were incubated in aerobic conditions at 37 C for 48 hours.

Positive plates were selected for a rapid slide agglutination test (Staphaureux*,Remel Europe Ltd, Kent, UK) to confirm whether the colonies were truly *S. aureus*. An authentic MRSA strain was used as a positive control for each batch of suspected colonies tested with Staphaureux*.

If a sample was both positive on the chromID™ MRSA agar plate and on the Staphaurex* slide, colonies were then sampled from the MRSA agar plate for further identification confirmation via mass spectrometry. Therefore, a pooled sample could only be MRSA positive with 100% specificity after it was positive on the chromID™ MRSA agar plate, positive on the slide agglutination test and identified as a S. aureus on mass spectrometry.

A random selection of positive samples' resistance to cefoxitin was confirmed with an antibiogram. To control for the possible effect of pooling on the results, 24 swabs from 4 herds of which two tested positive and two negative were individually inoculated directly onto the chromID™ MRSA agar plates. To ensure that the absence of a pre-enrichment broth was not affecting results, samples were individually enriched with thioglycollate broth for 24 hours at 37 C after which an inoculum was then

inoculated onto the chromID™ MRSA agar plate which was read after 24 hours. Lastly, to rule out the possibility that Gram-neagtive bacteria were inhibiting the growth of MRSA, individual swabs were used to inoculate 5% sheep blood agar swabs containing colistine for 24 hours at 37 C where-after the colonies were then inoculated onto the chromID™ MRSA agar plates.

Data analysis
A herd was classified MRSA positive if at least one pooled nasal sample tested positive for MRSA. MRSA herd prevalence was calculated with an exact binominal 95% confidence interval.

RESULTS

Out of the 25 participating herds only seven pooled samples, from three herds, were positive on both the selective chromID™ agar plate and the Staphaureux* agglutination test. All positive colonies were identified to be *S. aureus* on mass spectrometry. MRSA herd prevalence among large commercial piggeries in South Africa was calculated with an exact binominal 95% confidence interval to be 12%. All positive colonies tested on an antiobiogram were resistant to cefoxitin.

From the 24 individual swabs from 4 herds, all 12 from the two negative herds' tested negative while of the two positive herd's all six individual swabs from one herd and three individual swabs from the other herd tested positive on the chromID™ MRSA agar plates. All 12 swabs from the negative herds tested negative after pre-enrichment while all six swabs from one positive herd and three swabs from the other positive herd tested positive after pre-enrichment. After pre-inoculation onto 5% sheep blood agar plates containing colistine, the two negative herds' swabs all tested negative on the MRSA selective plates while all six swab samples from the positive herd and three swabs from the other positive herd tested positive on the MRSA plates.

DISCUSSION AND CONCLUSION

This study found a 12% (95% CI: 2.5 – 31%) herd prevalence of MRSA in large commercial piggeries in South Africa. In total, 450 slaughter pigs' nasal contents were sampled from 25 large commercial pig herds and MRSA was detected in only 7 samples from 3 herds. Despite the relatively large range of the estimate, it can be concluded that substantially at most one third of piggeries appear to have MRSA carriers. This relatively low prevalence was unexpected compared to the study of Broens et al. (2011) in the Netherlands where a herd prevalence of 67.3% among 171 breeding herds and a 71% herd prevalence among 31 finisher herds were observed. However, in both Nigeria and the USA low MRSA herd prevalence's were estimated. In Ilora, Nigeria, MRSA herd prevalence from 11 participating herds was 9% (Okunlola & Ayandele, 2015). The latter study correlates well with the 12% MRSA herd prevalence found in South Africa in this study. A more recent study in the USA found no MRSA in 36 herds across 11 states (Sun, Yang, Sreevatsan, & Davies, 2015). It is therefore still possible to have a low herd prevalence of MRSA in pig herds despite the high prevalence found in other pig-dense countries in the world. One may speculate that all three countries with low MRSA herd prevalence have relatively healthy herds due to being less pig-dense than countries such as the Netherlands. It may furthermore be speculated that increased distances between piggeries increases overall biosecurity resulting in less spread of disease between herds with consequently healthier herds and an overall reduction in the use of antimicrobial and other drugs to curtail disease. The influence of pig density on MRSA herd prevalence warrants further investigation.

The prevalence of MRSA in infected herds could have been underestimated if the herd-level sensitivity of the tests was low. In this study the specified minimum expected within-herd prevalence was 20% which was far less compared to true prevalence which Verhegghe (2013) determined to be at least 75% in finisher pigs. In the Eastern Cape of South Africa, Adegoke and Okoh (2014) sampled 64 pigs from different ages of which 15 pigs were MRSA positive giving a within-herd prevalence of 23%, which correlates with the in-herd specified minimum expected prevalence of 20% used. It has to be borne in mind though, pigs from different ages (including piglets) were sampled in the Eastern Cape herd. Therefore, the true within-herd prevalence in South African pig herds and the age of pigs with the highest MRSA prevalence are areas in need of investigation.

Herd prevalence might have been over-estimated due to possible cross contamination between pigs from different herds or environmental contamination as the pigs did spend time in the lairage before sampling which would significantly increase the risk of transfer of LA-MRSA between pigs

(Schmithausen et al., 2015). MRSA transmission via nose to nose contact was possible at one abattoir due to non-solid partitions between pig pens, but only one herd out of a total of six herds at this abattoir tested positive which might indicate that this was not a significant risk factor. Environmental contamination is suspected to be low due to lairages at all abattoirs being washed and disinfected between batches of pigs. Therefore, the chance of an over-estimation of prevalence seems limited. Environmental swabs from the abattoir would have to be taken to estimate the risk of environmental contamination.

Herds were only sampled once off and not repeatedly which might influence the results to either an over- or an underestimation of MRSA herd prevalence. The risk of herds acquiring MRSA is not only dependant on transmission between carriers and non-carriers, but is significantly increased via the administration of antimicrobials to groups of pigs (van Duijkeren et al., 2008). Group antimicrobial administration might change periodically within a herd depending on the treatment programme followed on the farm prior to sampling. It could also be seasonal if one considers an increase in severity of respiratory diseases during autumn and winter. The influence of antimicrobial usage patterns was not investigated in this study.

Selection for MRSA in finisher pigs in South Africa is potentially decreased due to non-availability of registered in-feed or in-water formulations of penicillin-based or cephalosporin antibiotics for pigs, which would have increased the risk of pigs becoming MRSA carriers (van Duijkeren et al., 2008). These substances can however be used either off-label or compounded on special request by the herd veterinarian, but practices such as this in finisher pigs are mostly not done due to strict annual on-farm food safety audits on the majority of commercial piggeries. There are however injectable formulas available, but strict meat withdrawal periods have to be adhered to.
Our results indicate that LA-MRSA in pigs in South Africa is likely of limited significance as a general public health threat to workers on farms and abattoirs. Further research should focus on quantifying the true public health risk through establishing within-herd MRSA prevalence and LA-MRSA carrier status of personnel working with pigs on farms and in abattoirs.

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CREATING RABIES AWARENESS IN A RURAL COMMUNITY

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INTRODUCTION

Rabies is an endemic disease in South Africa, killing thousands of people every year in Africa and Asia. Rabies control requires regular vaccinations of dogs, as dogs are the main source of rabies in South Africa. In spite of the rabies vaccine being readily available free of charge, and regular vaccination campaigns by the state and private sector, some communities are not taking responsibility for rabies vaccinations.

OBJECTIVES

Participatory reflection and action (PRA) is a collaborative engagement methodology that aims to enable communities to take ownership of and responsibility for addressing challenges, using assets already present in the community. This study utilised PRA in a community in the Hluvukani village in Mpumalanga province, South Africa, in an attempt to raise awareness and action with regards to rabies control.

METHODS

The community was visited several times over a period of 10 months. Each visit comprised of a series of participatory engagements, including amongst others, asset mapping, small group discussions, transect walks and poster making.

RESULTS

Rabies awareness was initially not a priority in this group of participants, due to more pressing socio-economic issues as well as traditional and cultural beliefs. Over time, rabies control was brought to the foreground resulting in active participation in a rabies awareness project.

CONCLUSIONS

This study proved that trust and confidence needed to be built up with repeated participatory engagements in order for change to take place in people's perceptions and their resultant actions. Community involvement to promote animal health and welfare is essential and its success depends on the utilisation of proven community development principles. Successful veterinary community engagement is enhanced by collaboration between different sectors within the profession and other organisations.

AN UNUSUAL CASE OF RABIES IN MUTTON MERINO SHEEP IN THE NORTH WEST PROVINCE, SOUTH AFRICA

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There have been recent outbreaks of rabies in South Africa, mainly in the Kwa-Zulu Natal province but also elsewhere. Infections in sheep in South Africa are rare. The following wildlife have been identified as carriers that transmit the virus to humans: yellow mongooses, genets, unspecified wild cats, caracals, honey badgers and chacma baboons. Jackals and bat-eared foxes have also been identified as carriers that may transmit the virus to domestic animals. An outbreak of rabies in a Mutton Merino flock as a result from a predator attack is described. The vector is identified and the behaviour of the predator is described as well as how it could have gained entry. All seven sheep that survived the predator attack died within 38 days. Forty-five days after the predator attack, one sheep that had not been attacked by the predator died and tested positive for rabies suggesting horizontal transmission in this flock. Differential diagnoses, post-exposure prevention of infection and future prevention are discussed.

ECONOMIC SUCCESS FARMING WITH SHEEP AND CARNATIONS

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Maluvha Nursery is a 30 hectare carnation producing farm. Buildings cover seven and a half hectares: greenhouses, offices, storerooms and staff living quarters. Ten hectares are outside the security perimeter and only the firebreak needs to be maintained because it borders a nature reserve. All other open land is covered with a variety of veld grasses and kikuyu that need constant maintenance to keep pests from the grass affecting the flowers in production.

Maintenance entails daily trimming with brush cutters or “weed eaters” controlled by 3 individuals. There is a lot of trimming close to the greenhouses and hard to reach places that took 5 days a week, 9 months of the year. During the remaining 3 months of winter, the grass could be trimmed every 4 weeks, giving time for machinery maintenance. The larger areas between greenhouses were trimmed every second week with a tractor driven slasher/mower. Again, during winter, maintenance of the implements and tractor took place. During 2012, the annual expenditure to maintain the open land grasses amounted to 125000 SA Rands. The costs were expected to increase drastically as labour costs and maintenance and replacement costs of machinery keep increasing. This system requires 6 individuals for different tasks: three for “weed eater” work; one for operating the tractor with mower; and a further 2 individuals remove carnation “wastage” daily form the packing store to the composting heaps.

Three hundred sheep were introduced to the farm during 2013 and since then, all the above tasks have been carried out by 3 individuals, employed on a part time basis. The sheep are kraaled at night and let out for grazing in the morning by 9:00. In the afternoon, the sheep are brought back in the kraal by 16:00. The rest of the day the workers are tending to carnation harvesting and plant health. The removal of the carnation wastage is also done by the same 3 workers. The carnation wastage is fed to the sheep on returning to the kraal and excess wastage is composted with sheep faeces. The workload of the 3 individuals increases during the six-week lambing season once a year. Since 2014, the cost of keeping the sheep amounts to 45000 SA Rands per annum and remains stable and includes 2016. This figure takes the following sheep related costs into account: labour; supplement feed, veterinary costs and maintenance of sheep facilities.

Maluvha Nursery now has the advantage of well-maintained grass all around the farm with 90% less labour. Good quality “kraal manure” is compost in half the time that it took the carnation waste to compost. The carnation has high protein content and as such is a palatable protein feed supplement for the sheep. The carnation planting season starts at early winter (April/May) and ends in summer (December/January). That is exactly when the grazing on the farm is in short supply. Carnations are also rich in minerals and receive fertilizer in a semi hydroponic system.

TECHNOLOGY AND NUTRITION: UNDERSTANDING GADGETS AND NUMBERS

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ABSTRACT

With advances in technologies, we are all surrounded by devices that should make our life easier. The agricultural sector has already started to use many of these technologies years ago. Compared to other agricultural sectors, dairy farming has lagged behind also because of a world dairy crisis. Milk prices and feed stock instability and continuous need for increases in quality from consumer's present new challenges. However, scientific and technological knowledge have sufficiently advanced over the years to enable feeding management and husbandry practices to overcome the challenges and allow sustainability.

TECHNOLOGY AND NUTRITION

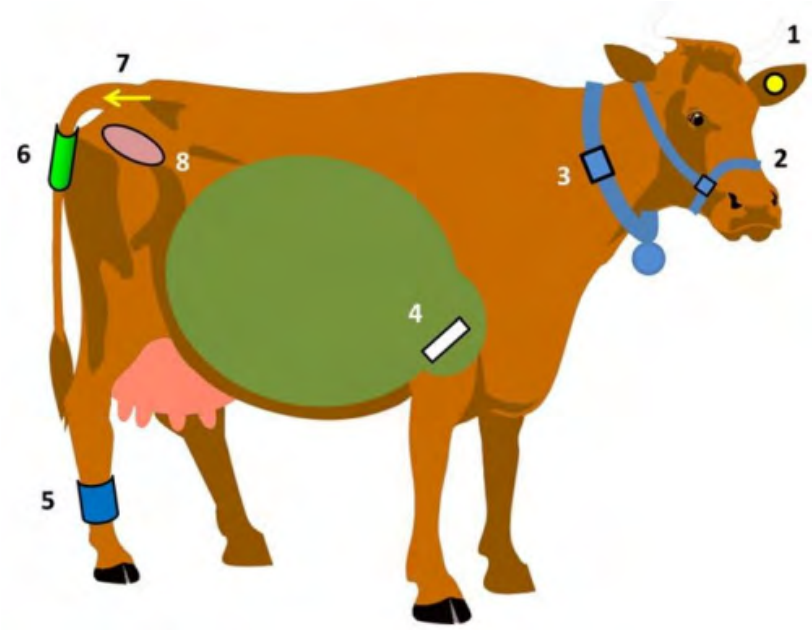
As soon as we talk about technology in dairy farms many of us think about robotic milking, but there is now much more than such an expensive investment that can assist in carefully managing these high value dairy cows. Engineering advances and the decreasing cost of electronic technologies has allowed the development of "sensing solutions" that automatically collect data, such as physiological parameters, production measures and behavioural traits. Body related parameters, even before milk yield and composition, are the first ones to be affected as soon as the complex "dairy cow system" is not balanced or tuned. Such data can potentially help the decision making process, enabling early detection of health or wellbeing problems in individual animals and, hence, the application of appropriate corrective husbandry practices.

Recent technology has developed mainly towards welfare biomarkers (e.g. stress and metabolic diseases), activity based welfare assessment (e.g. oestrus and lameness detection) and sensors of temperature and pH (e.g. calving alert and rumen function). Many of these technologies have been available for research, but because of lower costs, enhanced data acquisition and storage, faster processing speeds and improved wireless communication, they are now available to dairy farmers for the daily management. The main problematic consequence is that these units can generate a huge amount of information that only a well-prepared technician and farmer can interpret. There are, however, specific software that can assist in the difficult task. To make things more complex, science has been showing how cows have a strong "biological and behavioural personality", meaning that their "numbers" must be interpreted not only within the specific farm, but even within each cow. In fact, it has been evident for years how a diet high in starch content, even if fed to the whole herd, can impair rumen function of few cows of the herd, showing how only some cows will be affected in terms of rumen pH, because of both different feeding behaviour and rumen environment, when compared to other herd-mates. This software is continuously updated to help interpret the records also based on these scientific advances.

As nutrition is the largest expense of a dairy farm, we will spend few words on nutrition-related technologies. Activity sensors have been used by the dairy industry for many years, mainly to monitor general animal activity and relate it to oestrus detection. The availability of low cost tri-axial accelerometers and wireless telemetry has allowed accurate models of behaviour to be developed and, sometimes, combined with detection of rumination activity by acoustic sensors to monitor oestrus cycle. Three-D video imaging has allowed to body condition score cows removing the human factor bias. Some of these accelerometers offer information also on feeding behaviour (eating, ruminating, chewing, drinking). Wireless telemetry has been applied to develop boluses for monitoring rumen rumen function and possible metabolic disorder to detect sub-acute acidosis through measurements of pH, temperature and redox potential. The same companies are developing boluses that measure gases and ions formed in the rumen (CO_2, H_2, CH_4, K^+, NH_4^+) that can give a very accurate description of the rumen environment. However, these are being used and validated only in research at the moment and we are still learning on the best way to use these rumen parameters. Other nutrition-related technologies are body condition scoring (BCS) by image capture and individual feeding for calves, heifers and cows according to size, BCS, physiological stage, growth,

development and maturity. These last two technologies are obviously the ones that can have a more direct impact on a daily basis.

It is evident that there is no lack of technology and information that could be used. What may be lacking is the right “interpreter” of these numbers to reduce the information to simple answers and management inputs. However, what is more concerning we strongly believe is the attitude that many of us have with technologies, i.e. aiming for the hi-tech device that in the end is used only for the simplest task of the many. The objective of technology is to assist good husbandry, not replace it and the deliverable must be as simple as possible. A dairy farm should first make the possible to improve all basic management practices within all farm sub-units, it’s only then that precision farming and feeding can make use of technologies to improve animal efficiency and welfare and therefore farm profitability.



The “Super-Cow”: (1) ear tag, (2) halter, (3) neck collar with counterweight, (4) reticulorumen bolus (in reticulum), (5) rear leg pedometer, (6) upper tail ring, (7) tail head inject, and (8) vaginal bolus.

ANIMAL SENTIENCE, ANIMAL ETHICS AND THE VETERINARY PROFESSION

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Sentience in both human and non-human animals is the capacity to feel both pleasant and aversive sensations, which are conveyed to the brain via peripheral nerves and to experience pleasant and aversive brain generated emotions. In other words, it is the capacity to feel and to think. Sentient animals have interests that are linked to their nature. These interests are thoughtlessly and ignorantly violated in their use by humans. Animal sentience is about feelings that matter to the animals experiencing. This creates a moral imperative for humans to both respect and protect the welfare of these animals.

In recent times, the developing science of animal sentience has become pivotal in informing the advancement of animal welfare and the theories of animal ethics and animal rights. Awareness, feelings and cognition occur in different forms, degrees and combinations in different animals which have developed along different evolutionary paths. Notwithstanding this, veterinarians ought to be advocates for the welfare of farmed domesticated and wild animals and others and not be passive participants in their exploitation. This presentation will explore why sentience matters for practice and policy.

LAW, ANIMALS AND HUMAN RIGHTS

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What is the link between law, animal welfare and human rights? Is there a connection at all or are these mutually exclusive concepts? I argue that there is a link between these concepts. This is based on three principles. The first is the notion of power. Human rights and law are both embedded in systems of power and, in the modern constitutional age, these systems are being refined to ensure that those with power do not abuse those without. The second principle relates to the language and purport of the Constitution. For example, the Constitution refers to the rights of ‘everyone’ to life, dignity, health and bodily integrity. The word ‘everyone’ is not defined to mean every human being. Of course it would be fallacious to argue that the drafters of the Constitution intended to include non-human animals in the purview of the Bill of Rights. However, the Constitution is designed to be a transformative instrument to transition South Africa from apartheid to the era of democracy. This transformative aspiration is one that can and should be applied to all systems of oppression, especially in respect of the meat industry. The final principle relates to the link between the violation of animals and the violation of humans. If we prefer to take a human-centric approach to the protection of animals, we need to be insightful and honest about the way in which the meat industry harms human health, access to food and water, access to land and the continuum of poverty.

THE IMPORTANCE OF VETERINARIANS IN SUPPORTING ANIMAL WELFARE LEGISLATION

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Veterinarians play a critical role in both promoting the good care of animals and forwarding good animal welfare principles in terms of animal management. However the role of the Veterinarian also extends into the field of animal protection where their contribution and involvement can play an integral role in supporting the judicial process with the consequent effects of protecting communities as a whole.

This presentation acknowledges the value and role of veterinarians in general society but then goes on to explore this evolving role in more detail, especially in the ever changing local context. We review the South African dynamics affecting animal protection including economic challenges, environmental concerns and high levels of violence and the participative and committed approaches necessary to overcome these. .

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BETTER SCIENCE FROM FEWER ANIMALS – WHERE RESEARCH AND ANIMAL WELFARE MEET

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Widely accepted international principles that govern the humane care and use of non-human animals for scientific purposes (i.e. research, testing or education) are often expressed in terms of the ethical framework of the *Three Rs*. This represents the *Replacement* of animals by non-animal models whenever possible; the *Reduction* of the number of animals used to the minimum required to yield scientifically valid results; and the *Refinement* of experimental design and animal care standards, in order to limit the potential for pain, suffering, distress or lasting harm – thus improving animal wellbeing and honouring the dignity of animals.

South African ethical and regulatory standards for animal research are facing significant change, as reflected in the revision of National Standards and Guidelines for animal care and use, and regulatory requirements. Veterinary and para-veterinary professionals who work in this field – whether on clinical or oversight basis, or as member of Animal Ethics Committees – are facing the challenge of effectively implementing the Three Rs, while being often under-equipped to do so in the absence of formal training in this specialised field.

Education and training initiatives to empower veterinary and para-veterinary professionals in ethical decision-making nationally will be discussed in the context of Three Rs implementation.

THE DONKEY SKIN TRADE AND OTHER CHALLENGES FACING WORKING DONKEYS IN SOUTH AFRICA

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Donkeys are a critical and integral part of the lives of many of the most disadvantaged members of South African society, who rely on them essentially for transportation and draught power in agriculture.

Donkeys are remarkably tolerant and resilient animals that are able to survive in the most challenging environments and are widely used throughout sub-Saharan Africa. Working donkeys can be used by women and children without infringing on any cultural barriers, their size and temperament make them relatively safe and easy to handle and their remarkably tolerant constitution results in them surviving and working even when the most basic of care is lacking. The valuable role donkeys' play in sustaining disadvantaged communities is typically unrecognised and it is largely left to NGOs such as the NSPCA to provide primary care to working donkeys and education and training to their owners.

The Donkey Skin Trade is a new and serious challenge that has had significant impact on both donkeys and their owners. The presentation explores key areas of concern issues relating to working donkeys and their communities with the goal to create awareness and helpful dialogue and action.

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THE RISE AND FALL OF THE FRENCH BULLDOG

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The French bulldog breed has over the past year become the most popular dog breed globally, even surpassing the loyal Labrador according to the Kennel Club in the UK. This comes at a huge price not only for the unsuspecting owner but to the detriment of the French Bulldog breed too as unscrupulous breeders breed in numbers to satisfy the increasing demand without any regard for the health and quality of the puppies. This leads to an increased incidence of hereditary diseases (e.g. brachycephalic obstructive airway syndrome (BOAS), degenerative myelopathy, hemivertebrae, hip dysplasia, corneal ulceration). It is likely that more French bulldogs will be abandoned to rescue organisations due to health problems.

Currently only a handful of breeders do a very limited amount of health tests. There is no system in place to hold breeders accountable for the quality of the puppies they breed. It is envisaged that the establishment of a French bulldog Health Scheme will start to address the health issues in the breed. Mandatory testing for hereditary conditions and awareness campaigns may be utilised. The presentation will explore how South African veterinarians can be instrumental in ensuring the health of purebred dogs.

MONITORING PHYSIOLOGICAL INDICATORS OF STRESS DURING TRANS-RECTAL PALPATION OF THE REPRODUCTIVE TRACT IN MARES USED FOR TEACHING

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It is important to manage potential stress experienced by animals used in teaching veterinary students various clinical procedures so as to not compromise animal welfare standards. In this study the potential stress experienced by mares during transrectal palpation of the reproductive tract by veterinary students was assessed by means of heart rate variability (HRV) and endocrine stress-related indicators (salivary glucocorticoid- and faecal glucocorticoid metabolite concentrations). The technique evaluation and standardization confirmed that care should be taken when interpreting HRV results as correction factors can have an influence on the HRV indicators and heart rate measures. In addition, the repeatability and reliability of heart rate measures and HRV indicators may differ depending on the environment (unrestricted vs. restricted movement) being assessed. Although endocrine stress-related indicators did not indicate an overall stress response, the sympathy-adrenomedullary system (HRV) was able to identify short-term variations in autonomic cardiac control during palpation. Furthermore, the most significant shifts towards the sympathetic component were recorded during the first 5 minutes of palpation and 85 minutes after the start of palpation. The prominent vagal response in the initial stage of palpation may be attributed either to recognition (prediction of outcome) of the procedure or visceral pain. The age and experience of the habituated horses did not influence their stress response. In summary, the 20 minutes palpation period, restricted to one student, was tolerated well by the mares accustomed to the procedure, but the stress response after 55 minutes restricted movement was pronounced.

THE PERFORMING ANIMALS PROTECTION ACT AND HOW IT AFFECTS PRIVATE AND STATE VETERINARIANS: ARE WE READY FOR THIS ACT?

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ABSTRACT

The Performing Animals Protection Act (previously Act 24 1935) was recently amended, following a court challenge. In terms of the court order, the Department of Agriculture, Forestry's and Fisheries (DAFF) will now administer the Act. The Act states that "*Any person who intends to exhibit an animal; trains an animal to be exhibited; or uses an animal for safeguarding*", will now have to be inspected by trained state veterinarians and delegated animal scientists at least once a year. Inspections will be done in terms of a Veterinary Procedural Notice (VPN) developed by the Animal Welfare Working Group under the auspices of DAFF. As it is not expected that state veterinarians have a detailed knowledge of diseases and welfare of exotic animals, the inspectors will rely on the input of the private veterinarians who consult at these facilities. It has been observed, that many veterinarians, while very conversant with the diseases of these animals are not as conversant with current welfare norms and standards. In an effort to ensure that state veterinarians are fully conversant with not only general welfare requirements but also specific welfare requirements and to provide guidance for the inspections, eight hour training sessions were undertaken in all provinces to familiarise them with welfare issues pertaining to zoos and circuses as well as the VPN and how to do inspections. We believe that this training has created awareness of many of the issues associated not only with zoos circuses and guarding dogs but also issues of animal transport and slaughter. Welfare issues are no longer optional for veterinarians, but are essential to all the work that we undertake. If the profession does not take cognisance of this development, we believe that we will lose an opportunity to lead the way to science based progressive welfare.

INTRODUCTION

The Performing Animals Protection Act requires that all animals that are trained for exhibition or are exhibited (there are some exceptions in the act) and animals for safeguarding must have a licence.

These licences were previously issued by magistrates and pertained to magisterial districts. In 2013, after a challenge by the NSPCA, it was decided that the magistrates lacked the experience needed to administer this act and that it more properly belonged with the Department of Agriculture. After numerous extensions and delays, the implementation of the Act will commence in July 2017. In addition, an OIE inspection visit (2012) found that animal welfare concerns were a high priority for parts of South African society and the many NGOs involved. The current legislation is out-dated, not harmonised with OIE standards; there are no dedicated staff addressing animal welfare in the veterinary services and recommended that this be corrected.

In order to address both of these issues, DAFF formed the Animal Welfare Working Group (AWWG), which was formed from provincial representatives and a secretariat and chairman from DAFF. It was tasked not only to prepare for the implementation of PAPA Act but also to address animal welfare issues in general that pertained to the work of the department.

METHOD AND RESULTS

Initially, the AWWG met to consider the terms of reference and draw up a position statement. It was decided that a Veterinary Procedural Notice (VPN) such as is commonly used to guide the farm inspections would be used. The committee who took into account the multitude of animals that are covered by the Act drafted this. Private veterinarians are seen as playing a vital role in supplementing the knowledge of individual species and ensuring that the welfare of these animals is taken into account. Apart from animal health checks, many aspects covered in the VPN are required to be approved or endorsed by a facility veterinarian, such as a health and welfare plan for all the species/animals, the area allocated to each species, including shelter, feed and water points, examination area, storage and disposal of waste and mortalities, animal training (equipment and

methods), as well as associated records and registers. Disaster management plans should also be considered. Biannual (twice a year) veterinary visits will therefore be required. In addition, one of the members was designated to draw up training and once this was approved, training was taken to all 9 provinces. It was felt that it was important to provide context to the bill by using the training to raise general animal welfare issues. At least 30% of the training centred around the general animal welfare issues that all state vets encounter. In some instances, the Nature Conservation units were invited to participate in the training and this proved to be fruitful in raising awareness of certain constraints and issues around wildlife that were pertinent. Where this happened, the VPN was amended to include the new information. The response to the training was on the whole very positive. Only state veterinarians and designated animal scientists who have participated in the training are to be authorised to inspect premises. There are still distressing inconsistencies in the old Act, such as which performing or working animals need inspections and which are exempted. It is also more correct these days to refer to working animals as opposed to performing animals. These inconsistencies are however outside of the scope of the current amendments and will hopefully be addressed in future with an envisaged comprehensive Animal Welfare Act, which will probably take several years to pass all the consultative, legal and parliamentary processes.

CONCLUSION

There has been significant progress in introducing animal welfare into Veterinary Services. With increased awareness, state veterinarians are empowered to take better decisions. It is envisaged that additional training and course should be made available for all veterinarians, as the animal welfare is a field that is advancing at a rapid rate and is integral in all veterinary work.

PERPETRATORS OR PROTECTORS? WHY THE VETERINARY PROFESSION CANNOT IGNORE THE RIGHTS OF NON-HUMAN ANIMALS

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There is a disjuncture between society's perception of the caring role that it presumes veterinarians play and the reality on the ground of a profession that is deeply conflicted and in some cases the perpetrator of problematic and unethical behaviour. The paper will provide an overview of current landscape in relation to animal protection, welfare and rights and will examine the veterinary profession and veterinary associations in South Africa in relation to the political and ideological lens they apply to the relationship between humans and non-human animals. It will explore the role of veterinarians in the social construction and exploitation of non-human animals and key gaps in veterinary practices, curricula and policy. It will examine the "wildlife" and vivisection industries as examples of important sites of struggle for why we should be expanding and deepening conversations about human relationships with non-human animals and why there is a need to reshape public discourse, advocacy policy and legislation around controversial practices in South Africa, and what this recasted responsibility could look like.

TAIL DOCKING IN DOGS: THE EVIDENCE AND THE ETHICS TEN YEARS ON

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ABSTRACT

Non-therapeutic tail amputation, also referred to as prophylactic tail docking and tail maiming, is the amputation of a dog's tail at varying lengths to suit the recommendations of a breed standard. On 1 June 2008, the South African Veterinary Council (SAVC) decided that it would no longer condone the routine amputation of puppies' tails and pronounced the procedure unethical for veterinarians to perform (<www.savc.co.za>). Since then the procedure has been performed mostly by dog breeders although many breeders have chosen to abandon tail docking and most of the breed standards have been amended to accept dogs with intact tails. In South Africa currently veterinarians are allowed to amputate dogs' tails only for valid therapeutic reasons. As nearly a decade has passed since the SAVC's somewhat controversial decision, it is perhaps a good time to reflect and objectively consider the evidence base that is available on the topic, and possibly to finally come to a united ethical view on the procedure of routine tail docking for puppies. This article presents some of the literature and current information available on tail docking in dogs.

REASONS FOR TAIL DOCKING

Dogs have had their tails amputated for hundreds of years. Historically, the Romans did so because there was a belief that dogs who had the tips of their tails and/or tongues cut off, would not get rabies (Podberseck et al., 2000). Dogs owned by a poor person not permitted to hunt game, were docked as it was believed that the loss of the tail would make them slower and less successful at hunting (Fleming, 1895).

Nowadays, according to the Council of Docked Breeds in the UK (<http://www.cdb.org>), the reasons for tail docking in dogs, are to prevent tail damage especially when dogs are moving (hunting) in dense vegetation, for reasons of hygiene in long-haired dogs and to maintain breed standards (if previously docked breeds were no longer docked, they could end up with poor quality tails, and breeding for better tails would reduce the gene pool and promote inbreeding). It is claimed that when carried out correctly, either by rubber banding or surgical amputation, tail docking does not cause any pain or discomfort.

PUPPIES CAN FEEL PAIN

Neonatal puppies (puppies up to 2 weeks of age), even at a few days of age, have a fully developed nervous system and a well-developed sense of pain. In fact, they are considered to be more sensitive to pain due to incomplete myelination of nerves (Lefebre et al., 2007) than juvenile and adult animals (Matthews, 2008), (Matthews & Dyson, 2005), in line with similar findings in humans. The ascending pain inhibition pathways are only fully developed at 10 days of age (Hewson, 2008).

The pain associated with the actual procedure is relevant (Weary et al., 2006), as it involves cutting through or constricting nerves, blood vessels, ligaments, tendons, skin, cartilage and even bone in some instances. The potential complications afterwards such as bleeding and infection can also lead to pain and even the death of the puppy (Oghenemega and Oguntoye, 2016). There can also be complications later in life such as amputation neuroma formation (a very painful condition) (Gross and Carr, 1990) and damage to the anal sphincters resulting in faecal incontinence (Wansborough, 1996).

Breeders who perform tail docking maintain that they do not observe any signs of pain or suffering when the procedure is performed because they see the puppies starting to feed shortly after the procedure. In a study of the behaviour of puppies during tail docking (Noonan et al., 1996), it is pointed out that feeding directly after a traumatic experience may be a survival strategy – it is in the puppy's best interest to feed directly after a painful event in order to prevent weakness and susceptibility to disease. Furthermore, the act of suckling stimulates the release of endogenous

endorphins, which have an analgesic effect. Hence it is possible that the pups feed directly after the procedure in order to reduce pain and to seek comfort. Neonate lambs whose tails are amputated show both physiological and behavioural signs of pain associated with the procedure (Graham et al., 1997).

Studies in human infants and neonatal babies have shown that babies, who have been circumcised or had venipuncture without analgesia, subsequently were hypersensitive to painful stimuli. Similar findings were made when exposing rat pups to the injection of irritant substances without analgesia. Tail docking is far more invasive than either of these procedures, so if the research in rats is paralleled by research in infants, it is not unreasonable to extrapolate these findings to canine puppies (Hewson, 2008).

Certain analgesic drugs (pain killers) may not be effective in neonates due to underdevelopment of neurotransmitter receptors, hence pain management in neonatal puppies requires special consideration (Matthews, 2008) and pain-eliciting procedures should be avoided.

RUBBER BANDING

When rubber banding is used as the method of amputation, it causes ischaemia, which eventually results in necrosis. If the necrotic tissue becomes infected, it results in gangrene, which is potentially life-threatening.

It is widely accepted that ischaemia is accompanied by pain. In human medicine, rubber band tail docking can be compared to “compartment syndrome”, where the blood flow to a limb is compromised and it causes so much pain that emergency hospital admission is required (Lefebre et al., 2007). Studies performed in lambs where tail docking has traditionally been performed, found that both surgical docking as well as rubber banding resulted in behavioural and physiological evidence of pain (Kent & Molony, 1993). Frostbite, also caused by ischaemia, is listed as causing “moderate to severe, and severe” pain in animals (Matthews & Dyson, 2005).

FUNCTION OF THE TAIL

Dogs use their tail functionally mostly for communication and for balance.

Body language i.e. visual communication is the most important way in which dogs communicate (Overall, 2013; Landsberg, 2013). Various parts of the body are used, including the tail, ears, facial expression, body posture and eye contact. The height of the tail and its movement indicate what the dog is thinking and what its intentions are. A friendly approach is signified by wild wagging, above or below or the topline, an aggressive approach is evident when the tail is held high and just the tip is wagging. A fearful dog will hold its tail between its legs. One study even found that the dominant direction of the wag of a tail can indicate a dog’s motivational state (Quaranta et al., 2007) and that dogs can recognise this in other dogs (Sinischalchi et al., 2013). Leaver & Reimchen (2008) compared the effect of a short tail and long tail on inter-dog communication and found that a longer tail is more effective in conveying social messages.

These attributes are difficult if not impossible to observe in a dog with a maimed tail. This may lead to misunderstandings as the absence of the tail means that dogs and people are unable to read and interpret dogs’ signals correctly where there is only a short stump. This could lead to aggressive and potentially dangerous interactions. Over time, dogs with maimed tails may learn that people or other dogs are not trustworthy and they become more aggressive as a result. Not only does this have a direct negative influence on the dog’s quality of life, but it also poses a health risk to people and animals the dog may come in contact with.

The tail is anatomically part of the spinal column. It contains nervous tissue (the spinal cord and nerves), bone (tail vertebrae), cartilage, muscles, tendons and blood vessels. The tail helps stabilise the animal during movement as it moves in the opposite direction during loss of balance or a misstep and thus prevents falling. The absence of a tail therefore may lead to subsequent injury. Tails are also used as rudders when dogs are swimming.

PREVALENCE OF TAIL INJURIES

According to a Swedish study (Sejffert, 1992), the incidence of tail injuries in German shorthaired pointers increased significantly after the introduction of a tail-docking ban. However, the study has been criticised for various flaws including the lack of statistical comparisons (Diesel et al., 2010). A study in the UK showed that the risk for tail injuries in dogs with intact tails was only 0,23% i.e. a very low risk (281 injuries out of 138 212 dogs), and most of these injuries were as a result of household injuries, as opposed to hunting injuries (Diesel et al, 2010). This meant that 500 dogs would have to be docked to prevent one tail injury. Houlton (2008) studied injuries in game shooting dogs and found a 3,1% prevalence of tail injuries over two hunting seasons. This study also found a highly significant association between tail injuries and undocked dogs in two of the seven breeds studied (Springer spaniel and Cocker spaniels). In a study performed in New Zealand, 0,9% (n=619) of dogs presented to vets suffered from tail injuries. Of these, 13,8% required tail amputation (Wells, 2013).

LEGAL AND PROFESSIONAL POSITION

With regards to South African legislation, there is no law that specifically refers to tail docking but Section 2(1)a of the Animals Protection Act of 1962 (APA) refers to maiming:

> "Any person who cruelly overloads, overdrives, overrides, beats, kicks, goads, ill-treats, neglects, infuriates, terrifies, tortures or maims any animal….shall…be guilty of an offence".

The Concise Oxford English Dictionary (2008), defines "maim" as follows: "wound or injure (someone) so that part of the body is permanently damaged". The Free Dictionary (<www.freedictionary.com>) provides the following definition: "to injure, disable, or disfigure, usually by depriving of the use of a limb or other part of the body". Since the removal of the tail of a puppy results in permanent loss of (i.e. damage to) a body part, the procedure of removing a healthy tail can be considered to be a form of maiming, hence the term "maiming" is an accurate description of the procedure.

Any person, veterinarians included, who maims an animal, may be charged in terms of the APA. This is a criminal charge, heard in a criminal court. Veterinarians, if found guilty, may then additionally be charged with unprofessional conduct under the Veterinary and Para-Veterinary Professions Act of 1982.

At the time of writing, no successful prosecution for tail docking has been concluded. One of the reasons for this is the fact that veterinarians are either not willing to provide evidence in cases that go to court, or provide inappropriate evidence.

When formulating animal ethical positions, it is useful to take into account the recent findings of the South African Constitutional Court on the issue of animal sentience in its finding in the case of *NSPCA vs Department of Justice* on 8 December 2016 where it recognises that "…the rationale behind protecting animal welfare has shifted from merely safeguarding the moral status of humans to placing intrinsic value on animals as individuals" and quotes JA Cameron's minority judgment in another case, stating that "animals are sentient beings that are capable of suffering and experiencing pain".

CONCLUSION

Given the available evidence on tail docking in dogs, veterinarians should be able to make an informed ethical decision about the need for prophylactic tail docking in dogs.

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CAN HAPPINESS BE MEASURED?
THE BASICS OF SCIENTIFIC ANIMAL WELFARE ASSESSMENT FOR VETERINARIANS

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ABSTRACT

Ruth Harrison's book "Animal Machines" published in 1964, led to worldwide debate about the confinement of farm animals. The Brambell Committee (Brambell, 1965) established by the British government, reported that animal welfare embraced both physical and mental well-being, and called for research into veterinary medicine, stress physiology, animal science and, in particular, animal behaviour (Hemsworth et al., 2015). Thus the new discipline of animal welfare science emerged. Neuroscience, neurophysiology and immunology have all since contributed to the multidisciplinary nature of animal welfare science. Science helps us formulate well-considered ethical viewpoints on animal welfare matters. Ethics is informed by principles and beliefs, as well as science. Good science enables better ethics.

ARE THE FIVE FREEDOMS STILL RELEVANT?

Since the formulation of the Five Freedoms by John Webster (1994), they have served as a good general guide for the assessment of welfare of animals, especially when formulated together with their provisions (see Table 1). The Five Freedoms have been widely adopted and have facilitated many improvements in animal welfare because they are informative, easy to understand and practically useful.

Table 1: The Five Freedoms and their Provisions

	Freedoms	**Provisions**
1	Freedom from thirst, hunger and malnutrition	By providing ready access to fresh water and a diet to maintain full health and vigour
2	Freedom from discomfort and exposure	By providing an appropriate environment including shelter and a comfortable resting area
3	Freedom from pain, injury and disease	By prevention or rapid diagnosis and treatment
4	Freedom from fear and distress	By ensuring conditions and treatment which avoid mental suffering
5	Freedom to express normal behaviour	By providing sufficient space, proper facilities and company of the animal's own kind

There are however some challenges associated with the Five Freedoms (FF) which have become evident as the field of animal welfare science has made significant progress over the past few decades (Mellor, 2016a):

- The FF have come to represent absolute freedoms, contrary to the initial intent. It is misleading to refer to freedom from certain states, when we know that it is impossible to achieve complete freedom from negative states.
    - By referring to absolute freedoms, the impression may be created that fundamental freedoms are in fact attainable, and they could be construed as rights. This may result in expectations of idealistic states that are non-attainable.
    - Animals are genetically programmed to respond to negative states with specific behaviours, for example, hunger results in food acquisition and pain in escape or avoidance behaviours. Without any negative states at all, animals would not be able to engage in survival behaviour. High intensity negative states should clearly be avoided, but low-level negative states are needed for the performance of life-sustaining activities.
- The FF framework is limited in terms of the negative affects (feelings) that are referred to and does not distinguish between survival-critical and situation-related negative states (Table 2).

Survival-critical states refer to negative states generated by sensory inputs that register internal imbalances such as thirst, hunger and pain. Situation-related negative affects refer to sensory inputs from the external circumstances for example fear and frustration.

- Freedom 5 fails to recognise the importance of behaviours that are rewarding for example, affiliative social interactions and foraging behaviour, as opposed to any natural behaviour.
- The FF do not emphasise positive states to be attained, due to a widely-held assumption that once the negative internal states of thirst, hunger, distress, discomfort and pain are minimised, the net effect is improved welfare. This assumption is however challenged:
    - Managing negative internal states simply returns the animal to a neutral state, not necessarily a positive state.
    - Significantly negative survival-critical (internal) states inhibit the motivation of animals to engage in rewarding behaviour, for example significant pain inhibits mobility and the animal is less motivated to engage in rewarding foraging activities.
    - Therefore, in addition to minimising suffering, it is imperative that positive states (Table 2) are actively enhanced.
- The effect of improved environments in replacing negative survival-critical states with positive situation-related ones is not addressed in the FF. Stimulating, spacious and safe environments that provide the opportunity to engage in rewarding behaviours are more likely to enhance positive states and good welfare than barren, isolated environments which promote negative internal states.

Table 2: Negative and positive experiences, survival-critical and situation-related factors, the Five Domains (adapted form Mellor, 2016a)

		Negative experiences	**Positive experiences**
Physical / functional domain			
Survival-related factors (internal factors)	Domain 1: **Nutrition**	Restricted water & food Poor food quality	Enough water & food Balanced and varied diet
	Domain 2: **Environment**	Uncomfortable or unpleasant physical features of environment	Physical environment comfortable or pleasant
	Domain 3: **Health**	Disease, injury and / or functional impairment	Healthy, fit and / or uninjured
Situation-related factors (external factors)	Domain 4: **Behaviour**	Behavioural expression restricted	Able to express rewarding behaviours
Affective experience domain			
Survival-related factors	Domain 5: **Mental state**	Thirst Hunger Malnutrition malaise Chilling / overheating Hearing discomfort Breathlessness Pain Debility Weakness Nausea, sickness Dizziness	Drinking pleasures Taste pleasures Chewing pleasures Satiety Physical comforts Vigour of good health & fitness
Situation-related factors		Anger, frustration Boredom, helplessness Loneliness, depression Anxiety, fearfulness Panic, exhaustion	Reward Goal-directed engagement Calmness, in control Affectionate sociability Maternally rewarded Excited playfulness Sexually gratified

QUALITY OF LIFE

In order to apply the concepts described above, a distinction is made between husbandry systems that aim to keep animals alive (meeting basic needs), and those that enable animals to thrive (providing a range of opportunities for animals to experience positive affects). The concept of quality of life (QoL) acknowledges that animals have both positive and negative experiences and that these should be balanced. A net positive balance results in a good QoL. This has led to the animal welfare concepts of "a life not worth living", "a life worth living" and "a good life". In order to achieve "lives worth living", welfare codes should extend beyond standards that merely ensure minimal suffering (Mellor, 2016a).

THE FIVE DOMAINS

The Five Domains approach developed by Mellor and Reid (1994) addresses the shortcomings of the FF framework in that it distinguishes between physical / functional elements of well-being and the affective / emotional elements, as well as the survival-critical and situation-related elements (Table 2). Utilising the five domains (nutrition, environment, health, behaviour and mental state), a model was developed for grading animal welfare by assessing both welfare compromise (negative experiences) and welfare enhancement (positive experiences).

THE THREE ORIENTATIONS

Science-based thinking has led to the identification of three aspects that influence animal welfare, namely biological function, affective state (feelings) and natural living (Mellor, 2016a).

- Biological functioning as a framework for assessing animal welfare aligns with Broom's (2000) definition of animal welfare: "Animal welfare is the state of the animal as it attempts to cope with its environment." Non-coping is characterised by various behavioural (e.g. stereotypical and redirected behaviours) and physical / physiological parameters (e.g. activation of the physiological stress response) which are observable and measurable factors.
- An animal's capacity to experience different states means that predominantly negative experiences will result in a negative affective state and therefore poor welfare. Negative affective states are increasingly being studied from a neuroscience point of view, and it is becoming more evident that brain structure and chemistry is very similar between humans and other mammals. Affective states can also be characterised using physiological and behavioural measurements.
- In view of the fact that similar assessment parameters are used for these two animal welfare orientations, it makes sense that the biological functioning and affective states frameworks are increasingly integrated in the study of animal welfare.
- The third orientation, natural living, refers to the relevance of natural behaviours that animals find rewarding. Not all natural behaviour is necessarily beneficial in welfare terms, hence the need to define which behaviours are in fact desirable. Studies to date indicate that behaviours with elements of exploration, foraging / hunting, affiliative interactions, play, sexual behaviour, maternal and paternal care are rewarding.

ASSESSING/MEASURING WELFARE SCIENTIFICALLY

Animal welfare states are subjective as each animal experiences its own state subjectively. Conscious experiences of welfare are either positive or negative, and are the result of processing of sensory internal (survival-critical) or external (situation-related) inputs (Table 2) in the brain. The processing is affected by the species, the individual's own characteristics and past experience. An animal's welfare status at any one time may vary on a continuum from very bad to very good. Welfare states can be measured by using physical / physiological or behavioural parameters (Table 4).

Table 4: Behavioural and physical measures of welfare. HPA = hypothalamus-pituitary-adrenal axis. SAM = sympatho-adrenal medullary system.

Behavioural measures of welfare	**Physical measures of welfare**
Behaviour observation - time / activity budgets	Activation of HPA & SAM – physiological

Abnormal behaviour e.g. stereotypical behaviour Preference e.g. choice of different types of resources Work done to obtain a resource Work done to avoid something unpleasant Cognitive bias – optimism / pessimism Qualitative behavioural assessment (QBA)	measures Cortisol levels Glucose levels Respiration Pulse Blood pressure Heart rate variability Signs of disease / lesions / pathology Immune status Body condition Parasites Production Reproduction Mortality

There are three types of welfare measures:

- Animal-based for example body condition, emotional state
- Management-based for example feeding regimen, morbidity, mortality
- Resource-based for example amount and type of food, bedding
- Animal-based measures are considered superior in assessing the true welfare state as they cannot be manipulated by people

THE EFFECT OF HUMAN-ANIMAL INTERACTIONS

The relationship between humans and animals has a direct effect on welfare. Appropriate training and support of animal caregivers, and the potential for targeted cognitive-behavioural animal training to improve attitudes towards animals enhance good animal handling and consequently improve welfare. This would affect both the survival-critical and situation-related states; hence an assessment of welfare must include an evaluation of the human-animal relationship.

ENOUGH SCIENCE: WHAT ARE THE PRACTICAL WAYS IN WHICH VETS CAN ASSESS WELFARE?

Veterinarians may be requested to perform welfare assessments in different contexts, for example to assist animal welfare organisations when matters of animal cruelty are being investigated, to ensure compliance with overseas welfare standards for example for meat export or to assist retailers in the accreditation of welfare-friendly farms. While there is still a big scope for the development of welfare assessment protocols, some existing models for the practical assessment of welfare can be explored. Welfare assessments are holistic evaluations, and the models detailed here illustrate the need to consider several criteria together and not focus on just one or two issues.

Welfare Quality®

The Welfare Quality® system uses four main principles, which are sub-divided into a total of 12 criteria (Table 5): Each criterion contains certain measures and scores, which are detailed in the full protocol.

Table 5: Principles and criteria for the Welfare Quality® system

Principles	Criteria
A. Good feeding	1. Absence of prolonged hunger
	2. Absence of prolonged thirst
B. Good housing	3. Comfort around resting
	4. Thermal comfort
	5. Ease of movement
C. Good health	6. Absence of injuries
	7. Absence of disease
	8. Absence of pain induced by management procedures
D. Appropriate behaviour	9. Expression of social behaviours
	10. Expression of other behaviours
	11. Good human-animal relationship

12. Positive emotional state

This model was developed in the EU for the assessment of welfare of production animals, but the principles and criteria can be similarly applied to different species (see below – Shelter Quality protocol for dogs). The website <www.welfarequalitynetwork.net> contains useful resources including protocols for cattle, pigs and chickens. The full protocols are extremely comprehensive, although time-consuming to complete, and useful for official accreditation purposes.

Shelter Quality Protocol for shelter dogs
Based on the Welfare Quality® system, the Shelter Quality Protocol was developed in Italy and identifies criteria and welfare measures at shelter, pen and individual level. The principles, criteria and measures are listed in Table 6. The full protocol is available at <http://www.carodog.eu/wp-content/uploads/2014/02/Shelter-Quality-Protocol-2014.pdf>. The measurements for each criterion are described in detail in this resource, and includes recommendations such as enclosure sizes and .

Table 6: Criteria and measures of the Shelter Quality Protocol

Principles	Criteria	Measurements
Good feeding	Absence of prolonged hunger	Body condition
		Feeding
	Absence of prolonged thirst	Water supply
Good housing	Comfort around resting	Bedding sharp edges
		Cleanliness
	Thermal comfort	Shivering, huddling, panting
	Ease of movement	Space allowance
Good health	Absence of injuries	Skin condition / Lameness
	Absence of disease	Evidence of pain / Diarrhoea/ coughing
		Mortality / morbidity
	Absence of pain induced by management procedures	Surgeries & control of pain
Appropriate behaviour	Expression of social behaviours	Social housing
	Expression of other behaviours	Abnormal behaviour / barking
		Exercise
	Good human-animal relationship	Reaction to human
	Positive emotional state	Emotional state

The Hand
The Hand was developed by The Donkey Sanctuary in the UK and although it is designed specifically for donkeys, can just as easily be applied to other species with small adaptations. This model uses the human hand as a model whereby each anatomical part represents a relevant part of the welfare assessment. It is a structured approach that provides a standardised assessment of individual donkeys and groups of donkeys.

The hand is represented as follows:

- The thumb: Behaviour, demeanor
- The index finger: Body condition score
- The middle finger: Skin – wounds, hydration, harness
- The ring finger: Foot care – lameness, farriery
- The little finger: Other signs of injury and disease
- The palm: The whole life of the donkey – age, history, how is it worked / used, how many owners, end of life issues
- The knuckles: Human factors - explore working practices, attitudes, beliefs, traditions, cultures, community partnerships

The full protocol is available from the author. A South African donkey sanctuary, Eseltjiesrus <www.donkeysanctuary.co.za>, offers workshops to vets, donkey owners and welfare officials in the practical application of the welfare assessment.

CONCLUSION

Animal welfare assessments are holistic assessments of a variety of parameters. They should not focus only on alleviating negative states, but also determining and enhancing positive welfare. Providing positive experiences directly prevents suffering. Animal welfare science has practical applications in the everyday lives of veterinarians. A combination of the scientific knowledge and practical use of welfare assessment models, will enhance veterinarian's skills and their ability to make a difference for animals.

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In dogs with thunderstorm phobia profound responses may be diminished, but the dog may still pant, pace or stay close to the owner. Animals showing severe panic attacks may be difficult to control during a storm. Coexistence of other anxieties worsens the prognosis.

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COMPULSIVE DISORDERS IN DOGS

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ABSTRACT

Compulsive disorder (CD) describes a sequence of movements usually derived from normal maintenance behaviours that are performed out of context in a repetitive, exaggerated, ritualistic and sustained manner. They must be sufficiently pronounced to exceed what is necessary to meet its apparent goal or such that it interferes with normal functioning. The terms such as conflict-induced behaviour, frustration induced behaviour; displacement behaviour, redirected behaviour and vacuum activity are commonly used. Compulsive disorders initially originate from behavioural arousal, stress, conflict and frustration, which can lead to anxiety or displacement behaviours. Compulsive behaviour appears to develop when the animal discovers that multiple repetitions of a ritualised behaviour produces a reduction in arousal and frustration. Acral lick granuloma and tail chasing are common CD in small animals. Treatment is multi-modal and should consist of management, environmental changes, behaviour modification techniques and psychotrophic medications.

INTRODUCTION

Stereotypical behaviour describes repetitive, ritualised, out-of-context locomotor behaviour, such as pacing, circling, or shuffling. *Compulsive* behaviour was introduced to capture behaviours related to stereotype behaviour, but that are non-locomotor (vocalisation, licking, self-mutilation, staring, holding an object or part of body, staring into space). Today the term compulsive disorder is used as a general term for all the behaviours in this class[1,3,6,8].

Compulsive disorder (CD) describes a sequence of movements usually derived from normal maintenance behaviours that are performed out of context in a repetitive, exaggerated, ritualistic and sustained manner. They must be sufficiently pronounced to exceed what is necessary to meet its apparent goal or such that it interferes with normal functioning[2,3,6].

When discussing compulsive disorders terms such as conflict-induced behaviour, frustration induced behaviour, displacement behaviour, redirected behaviour and vacuum activity are commonly used. Below is a short definition of the terms:

- *Conflict induced behaviour*: The presence of two opposing, similarly strong motivations at the same time. This may lead to a displacement behaviour[1,3,8].
- *Frustration induced behaviour*: A situation where an animal is motivated to perform a behaviour but is prevented from doing so because of physical or psychological obstacles in the environment. The resultant behaviour could be a displacement behaviour or a redirected behaviour[1,3,8].
- *Displacement behaviour*: A normal behaviour shown at an inappropriate time and appearing out of context for the occasion. It may be observed in situations of arousal when there is no appropriate outlet for arousal[3].
- *Redirected behaviour*: The animal is motivated to perform an activity but is unable to gain access to the principle target. The behaviour is directed to an alternative target[3].
- *Vacuum activity*: An animal may be highly motivated to perform an instinctive behaviour but there is no available outlet. These activities have no apparent useful purpose[3].

DEVELOPMENT OF COMPULSIVE DISORDERS

Compulsive disorders initially originate from behavioural arousal, stress, conflict and frustration, which can lead to anxiety or displacement behaviours. Arousal and anticipation are physiological states that are vital to normal behaviour and are linked to anxiety and normal expectations of reward. When an

animal anticipates a positive outcome and the outcome is less rewarding or never happens, frustration is the result. When an animal anticipates threat or danger, there is a negative emotion, which is unpleasant and leads to anxiety. Frustration and fear are essentially the same emotional state in animals. Both anticipation of a threat or danger and anticipation of frustrated non-reward are therefore negative emotional experiences involving anxiety[1].

There appear to be at least two different mechanisms by which compulsive disorders arise. Locomotor CD (tail chasing, pacing) tend to develop in situations of stress, anxiety, conflict or frustration and tend to be displayed in situations of high arousal. By contrast, oral and self-directed CD (licking) may develop more acutely without any obvious conflict, and are most likely displayed in situations of minimal stimulation. They may even help to calm the pet[3].

Possible aetiologies for a compulsive disorder might include[3]:

- Insufficient stimulation
- Changes in routine
- Inconsistent or improper training
- Anxiety inducing situations
- Household changes, including change in family members or pets
- Situations of conflict or frustration

Compulsive behaviour appears to develop when the animal discovers that multiple repetitions of a ritualised behaviour produce a reduction in arousal and frustration. The behaviour provides a faster, more reliable, and more effective escape from the negative emotions. The experience of reduction of arousal rewards the behaviour, reinforcing it. Compulsive behaviours generalize to other contexts in which the animal experiences a high level of arousal. As the number of eliciting contexts increase, the threshold of arousal needed to elicit the compulsive behaviour decreases and the animal appears to lose the choice whether to perform the behaviour or not[4]. Any attention from owner inadvertently reinforces the behaviour.

There is a thought that compulsive behaviour may be self-reinforcing, caused by the release of endogenous opoids in the CNS, which may allow some animals to cope with conditions that don't meet their species-specific needs. This theory however lacks scientific support through research[2].

Compulsive behaviours can be classified in groups. They are[4]:

- Behaviours involving locomotion: spinning, tail chasing, pacing
- Oral behaviours: self-licking/chewing, flank sucking, pica, licking/chewing of objects
- Vocalisation: howling, barking
- Hallucinatory behaviours: staring at shadows, fly snapping, air licking
- Aggressive behaviours: aggression directed at inanimate objects or towards self

MALFUNCTIONAL VS. MALADAPTIVE

Compulsive behaviours are abnormal because they are displayed out of context and often are repetitive, exaggerated or sustained[3, 4]. With respect to captive animals however, controversy as to whether CD represent a normal response of a normal animal to an abnormal environment (maladaptive) or whether they are abnormal in the sense of lacking in function and/or being the expression of an underlying pathology (malfunctional)[9]. In maladaptive behaviours the animal attempts to find a surrogate for a missing normal behaviour, to escape from confinement or to otherwise alleviate a problem. They are abolished immediately by a specific change in husbandry. Malfunctional behaviours are a product of pathology, may occur with a range of other effects and may involve source behaviours that do not closely reflect the original cause of repetition[5, 7].

HISTORICAL FINDINGS

The age of onset of compulsive behaviours normally correlates with social maturity, median age of onset at 12 months. Male dogs over represented. There appears to be a genetic predisposition. Breed may affect the type of compulsive disorder. Examples of breed specific compulsive behaviours include:

- Bull terriers: tail chasing
- Doberman pinchers: flank sucking
- Border collies: chasing of shadows
- Large breed dogs: acral lick granuloma

Behaviour to gain attention can look similar to compulsive disorder. Dogs can simulate medical signs and display other behaviours that get a response from the owner. Contact by the owner reinforces the behaviour. Negative attention increases dog's anxiety and the need for further reassurance. This kind of attention seeking only occurs when owner is present but not directly attending to the dog. Compulsion arising from hyper-attachment (separation anxiety) causes behaviour when owner is unable to attend to the dog, including times when they are away, and is therefore different from attention seeking behaviour.

DIAGNOSIS

Compulsive behaviour is a diagnosis by exclusion. Attention seeking behaviours as well as neurological and medical disorders can produce similar signs and these must be excluded. Seizure foci differ from compulsive disorders in that seizures arise independent of any specific stimuli or events, do not occur with any degree of regularity or predictability, cannot be interrupted, may have a recognizable pre-and/or postictal phase and often improve with anticonvulsant therapy[3].

ASSESSMENT

The dog usually shows a normal level of awareness throughout the behaviour (vs epilepsy, cognitive impairment[3]. It may be difficult to interrupt behaviour and the dog may become aggressive if it is manually restrained. The dog shows normal behaviour between bouts. There is a sudden and abrupt transition to compulsive disorder, without indicator signs.

The severity of behaviour depends on several factors. These include:

- Ease of interrupting the behaviour
- The number of different contexts in which the behaviour occurs
- Different events/stimuli that trigger behaviour
- Time spent in compulsive behaviour, and the degree that it substitutes for normal behaviour
- Progression of the behaviour

Dogs that show compulsive behaviours in multiple contexts, with the behaviour substituting for a number of normal behaviours and being hard to interrupt, are seriously affected.

ACRAL LICK GRANULOMA AND TAIL CHASING

Acral lick granuloma (ALG) is a distinct clinical entity in which dogs lick one or more of their limbs, causing significant damage. There are raised, ulcerative, firm plaques usually located on the limbs, most often the carpus and metacarpus[2]. Large breed dogs are most commonly affected. Lack of stimulation is frequently cited as the cause, but the licking may also be a displacement behaviour arising out of situations of conflict, frustration or anxiety. The behaviour occurs both when the owner is present and may occur at an even higher rate when the owner is absent. Underlying anatomical

abnormalities (arthritis, fracture, neural entrapment) or infectious or inflammatory causes may contribute this behaviour[3].

Tail chasing or spinning describes the behaviour where an animal spins in tight circles apparently trying to catch its tail. Some animals make contact with their tail and injure it, while others just go through the chase sequence. The tail chasing may occur in times of stress, frustration and conflict[2].

TREATMENT

Since compulsive disorders are both debilitating and progressive, it must be treated aggressively from the start. A multi-modal approach should be used. Medical treatment must be provided for self-inflicted trauma (ALG and tail chasing).

Early experience prevents all kinds of anxiety disorders. Puppies that are used to living in sociable and complex domestic environments are attracted to novelty and they are used to periods of solitude. This reduces the risk of behavioural problems. The dog has learnt to cope with a normal range of stimulation, arousal, frustration and behavioural conflicts.

Management

For ALG management would include the prevention of licking to allow for healing, but this is not a long-term solution as it does not address the underlying behavioural pathology. For the same reason, there is no place for tail amputation in tail chasing.

Environmental changes

Routines that create profound sense of order must be put in place (feeding, training, playing). Routines reduce unpredictability and anxiety. The environment must be made more engaging so that time and energy budget is used up constructively. Food dispensing toys encourage interaction with the toy and are preferred. The animal must be provided with a quiet and calm refuge or resting place. The dog's level of mental and physical activity should be increased. Creating a predictable environment for the pet can reduce stress. This can be achieved by including daily scheduled exercise, social interactions and play[2].

Behaviour modification

The owners must make a detailed list of all stimuli that trigger the behaviour (doorbell, telephone, visitors, owner departure, and excitement). Where possible, avoid or reduce exposure to these triggers. The aim would be to associate the event/cue with a rewarded performance of another behaviour (response substitution) or counter condition the emotional response to it. The pet can be taught to be calm, settled and relaxed on cue in a specific location[2]. The trained calm behaviour can be associated with each of the stimuli and contexts that provoke the behaviour.

The client must be discouraged from reassuring the patient during stressful events as this can lead to attention seeking behaviour. There is no place for positive punishment and threats (scolding, smacking). Owners of a tail chasing dog must be cautioned against physical intervention during an event, as it may trigger redirected aggression.

Psychoactive medication

Drugs are indicated for cases where the condition is severe, longstanding or where the behaviour is difficult to interrupt. It should also be considered when progression is rapid and the situation continues to worsen. They must be used in combination with behavioural therapy and environmental management. It is a way to reduce the inflexibility and rate of expression of compulsive behaviours so that environmental change and behavioural modification can be more effective.

Clomipramine and fluoxetine are commonly used[1]. Tricyclic's, other than clomipramine, or merely anxiolytic drugs are unlikely to have an effect because they have much weaker effect on serotonin re-

uptake and because compulsive behaviour, once well established, can be performed even when the animal is not in a high state of anxiety[4].

The thought is to start with an SSRI. If there is failure to respond, switch to another SSRI or change to clomipramine[1]. Dose rates are generally a little higher for compulsive disorders, but it is better to switch to another drug if there is poor response[1].

Drugs used for ALG include clomipramine and selective serotonin reuptake inhibitors. It may take months of therapy before the lesions resolve. Doxepin and amitriptyline (both TCAs) might be useful as they have behavioural and antihistaminic effects, but they generally do not have sufficient effect on the serotonin re-uptake to be as effective as clomipramine[3].

Drug dose rates are[3]:

- Clomipramine: 2 – 3 mg/kg bid
- Fluoxetine: 1- 2 mg/kg sid
- Doxepin: 0.5 – 5mg/kg bid
- Amitriptyline: 2 – 4 mg/kg bid
- Gabapentin: 2- 5 mg/kg bid

PROGNOSIS

The owner must be informed that it may take weeks to see improvement. Intermittent relapses are common. Control, rather than cure, is a realistic expectation. There is a poor prognosis for dogs that continue to live in stressful or deprived environments.

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OVERVIEW OF HUMAN DIRECTED CANINE AGGRESSION

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ABSTRACT

Canine human directed aggression is a common reason for owners to request a behaviour consultation. Human directed aggression is not always motivated by the desire to control but is often associated with underlying anxiety, conflict, and poor communication or the lack of clear rules and structure between the owner and the pet during social interactions. The client should be asked to describe the body posture of the dog before, during and after an episode. This will help to determine the underlying reason for the aggression. Causes for aggression include fear, possessive aggression, redirected aggression and impulse control aggression (previously termed dominant aggression). The treatment is aimed at controlling the problem, not achieving a 'cure'. Treatment consists of management, structuring the owner pet relationship, behaviour modification and psychotropic drugs. When dealing with human directed aggression a risk assessment should be included. Aspects evaluated include predictability, potential to cause damage, the characteristics of the family and the complexity of the whole situation.

INTRODUCTION

Canine human directed aggression is a common reason for owners to request a behaviour consultation. It is also one of the most dangerous behaviour problems as it could lead to serious injuries to the owner. During the consultation a risk assessment is usually given, which may influence the decisions taken. From the outset the client should be informed that there is no cure for aggression, it can only be managed.

The underlying motivation for the aggression must be determined. The behaviourist needs to get a good description of the dog's behaviour before, during and after the aggressive event. This can prove quite challenging as owners are often unable to give a good description of what actually happened.

CAUSES FOR HUMAN-DIRECTED AGGRESSION

Human directed aggression is not always motivated by the desire to control but is often associated with underlying anxiety, conflict, and poor communication or the lack of clear rules and structure between the owner and the pet during social interactions[4]. Aggression usually occurs in situations involving access to preferred resources, interactions such as petting, moving, handling and reprimanding or attempting to take objects away from the pet. In some cases the dog reacts impulsively and unpredictably, with injurious and dangerous consequences. Some dogs may have poor social communication skills and are unable to read the signals from their owners which then leads to an improper response. This behaviour is usually aggravated by reprimands and physical punishment[4].

On questioning, the owners will indicate that early signs of aggression have been present for some time, but that this was not really seen as a problem. These behaviours include staring, growling, baring of the teeth or snapping. The aggression usually occurs towards people with whom the dog lives. The aggression might not be displayed towards all family members. The persons' ability to control the dog and the frequency of interactions play a role. Observant clients may be able to indicate which situations trigger the aggression while others will say that they cannot predict the aggression. These clients are usually not able to see the warning signs, including calming signals.

Although this behaviour could be part of normal canine social behaviour its expression is influenced by the environment, learning and genetics. Underlying medical conditions, early experiences, inconsistent or lack of clear rules and routines within the household and within interactions with family members may influence the manifestation of aggression.

The client should be asked to describe the body posture of the dog before, during and after an episode. Unfortunately most clients are not able to do this. Having some pictures on body language available for the client to choose from might be helpful. The client has to describe each aggressive event including where it happened, what time of the day it was, who was present, what actually happened and what their response was.

A dog that is highly aroused is at higher risk for aggression, as its emotional state can interfere with the ability to respond consciously and rationally to stimuli. An aroused dog may respond impulsively, reflexively and defensively to a novel or startling stimulus[5].

Fear aggression is the most common type of aggression in dogs. Fear aggression occurs when the dog perceives a situation as threatening. Behavioural signs include fearful body postures and signs of sympathetic stimulation including tachypnoea and tachycardia. The body language of fear is often seen in possessive or so-called 'dominance' aggression. However, fear is often overlooked as a motivator (or maintaining factor) in aggression. It is therefore of utmost importance to get a proper description of the posture and signalling used by the dog. The dog may show aggression when avoidant or appeasing behaviour has failed.

Early traumatic experiences or lack of appropriate interactions may contribute to the expression of fearful responses. In these dogs aggression is accompanied by fearful or submissive behaviours. The intensity of aggression may increase over time as the dog learns that aggression provides an escape from fearful stimuli. The aggression is often worse if the dog is cornered or if it cannot escape. Movement, rapid approach, or reaching for the dog may elicit an aggressive response. The aggressive response is often followed by withdrawal and distance increasing responses[4].

Possessive aggression refers to dogs that aggressively guard objects (food bowl, chews, stolen or found items). Some degree of guarding is usually within the range of normal behaviour. Owner responses are often punitive and may increase the aggressive behaviour. In some dogs there are elements of play behaviour and attention seeking. The aggression may be accompanied by fearful or submissive body postures. As the dog learns that the aggressive response is successful the body posture changes and the intensity of the aggression might increase. The aggressive response might be followed by withdrawal and distance increasing responses[4].

In *redirected aggression* the dog becomes aggressively aroused by a stimulus or target other than the person that it attacks. Initially, the aggression is directed towards the stimuli that caused the aggressive arousal, but rapidly this switches to the person exerting control or attempting to punish the dog[2].

Impulse control aggression is defined as an abnormal, inappropriate out-of-context aggression consistently exhibited by dogs toward people under any circumstance (involving passive or active control of the dog's behaviour or the dog's access to the behaviour) [6]. The aggression intensifies should the owner try to correct or interrupt the dogs' behaviour. In the past this type of aggression has been labelled 'dominance aggression', 'impulsive aggression' and 'conflict aggression'.

These dogs struggle with people over control of all aspects of the social environment. These dogs provoke people, as this is the only way they can get information from and about the social environment and interactions. They become impulsive and aggressive when they cannot get clear information or when the information received confirms a threat (in the dogs perception) [6]. These dogs appear unpredictable as the pattern of the dog's reactivity depends on that dog's relationship with the individual person and his threshold for reactivity at that time.

It appears that two subpopulations exist. For the one the dogs demand vast amounts of attention and will pick and choose when they interact with the owner or ignore commands that are given. In the other, the dogs will act more independently and withdrawn. They interact with the owners randomly but might prefer to stay by themselves[3].

Dogs with impulse control aggression may show aggression in various situations and may stiffen, stare, become aroused, block and/or grab people. Examples of such situations include being pushed on or handled around the head, muzzle or feet or manipulation of the body; prolonged petting; being

corrected (verbally or with a leash or collar); being bumped into or disturbed when resting; reached over or stepped over; staring or when they are physically moved out of the way[1,4].

Clients may mention that their dog may push on people, put their paws on peoples' heads, shoulders or backs; block access to doorways; may stare at people; 'talk back'; hug people and give them kisses by licking them over the entire head or face[1].

This type of aggression is dangerous as it may not occur consistently (at each interaction with the owner) and the level of aggression may not be in proportion to the owners' action. These dogs worsen quickly and can become extremely dangerous if punished, threatened, shocked or otherwise roughly treated. These dogs can inflict serious injury. Owners are often in denial about the severity of the aggression, as the dog appears normal and loving most of the time.

TREATMENT

The treatment is aimed at controlling the problem, not achieving a 'cure'. The most important part of treatment is to identify and avoid situations that trigger the aggressive response. The client must be taught to recognise the body language of fear and anxiety as well as warning signals, including calming signals that often precede aggression. This can help the owner prevent and avoid an aggressive encounter.

Dogs should never be punished for displaying warning signals such as growling. The warning signal gives the owner time to respond to prevent aggression. Dogs that are punished for displaying warning signals will stop signalling and resort to biting without warning. As a whole there is no role for punishment as this increases fear and anxiety and increases the level of arousal. Punishment increases fear and anxiety and increases the general level of arousal. An aroused dog is at higher risk for aggression as its emotional state can interfere with the ability to respond 'consciously' or 'rationally' to stimuli. This increases the risk of family members.

Management of aggression includes locking the dog away, fitting a head collar for increased control and wearing a basket muzzle. In some cases euthanasia is the only safe option. Rehoming is not a good option as this may put the new owners at risk.

For dogs with fear aggression all interactions should be based on a command/response relationship and non-confrontational methods must be used to teach the dog to view household members as leaders and in control[4]. The pet-owner relationship needs to be restructured. Rules for interaction need to be created so that the owner knows when and how to interact with the dog. Initially, the owner initiates all attention, with the pet getting attention when it is quiet and calm. The owner can call the pet, give it attention and end the session.

Behaviour modification for impulse control aggression involves teaching the dog to defer to people[6]. This modification will decrease reactivity and provide instructions for the dog about expectations for behaviour and the rules for interaction. The dog is basically asked to sit calmly and look at the human whenever anything is desired or needed. The dog learns to be calm, it learns that it can ask questions of people and that they can get guidance about what is expected. A qualified dog trainer should be able to help train these behaviours. Physical contact with the dog must be limited and control the affection by only giving after the dog has followed a prior command.

Basic obedience is always beneficial as it creates a way for the owner to communicate with the dog and gives the owner verbal control over the dog. This can be used to end situations where aggression has occurred in the past (use the 'off' command to get the pet off the furniture rather than physically getting the pet off the furniture).

Counterconditioning and desensitisation can begin once the trigger(s) that lead to aggression have been identified. A stimulus gradient must be established from the stimuli least likely to cause a fearful response. The best reinforcer must also be established – what will the dog work for? Favoured rewards are used for training only. The dog is gradually exposed to a greatly reduced stimulus. The non-fearful behaviour is rewarded. The level of the stimulus is manipulated and/or increased gradually. Proceeding rapidly can intensify rather than diminish the fearful response.

Psychotropic treatment

For impulse control aggression the medication of choice is the selective serotonin reuptake inhibitor (SSRI) fluoxetine, which was developed for the treatment of impulsive conditions[6]. Other tricyclic antidepressants (TCA) or SSRIs or non-selective serotonin reuptake inhibitors may be beneficial, alone or in combination. Gabapentin can be useful to decrease the general arousal level[6]. A combination of drugs might give a better outcome.

For fear aggression medications include SSRIs with or without TCAs. If the dog is hypervigilant, gabapentin alone or in combination may help. During times of panic benzodiazepines (BZD) such as alprazolam can be used. For dogs that are globally hyper-reactive and affected by fear aggression, clonidine plus a SSRI and/or TCA might be helpful.

Drug dosages[6]:

Alprazolam: 0.01 to 0.1 mg/kg as needed, not more than 4 mg per dog
Amitriptyline: 1 – 2 mg/kg bid
Clomipramine: 1 -3 mg/kg bid, increase dose gradually
Clonidine: 0.01 – 0.05 mg/kg as needed bid, not more than 0.9 mg total for medium dog
Fluoxetine: 0.5 – 1 mg/kg sid-bid
Gabapentin: 2 – 5 mg/kg bid, up to 10 – 20mg/kg bid.

RISK ASSESSMENT[5]

It is important to assess the risk of injury that the dog poses to its environment. To do this a full history must be obtained from all family members and others involved with the pet. The predictability, potential to cause damage, the characteristics of the family and the complexity of the whole situation must be evaluated.

Predictability: All the situations or stimuli that trigger aggression must be determined. Once the triggers for aggression are identified, it must be evaluated whether the pet's response to the triggers is consistent. A dog that does not consistently show aggression to a stimulus is more dangerous as people tend to lower their guard in these cases. The type of stimulus is also important. The danger increases when a dog reacts to a 'benign' stimulus (such as stroking). The absence of warning signals or a short time interval between warning and aggression (latency) also increases the risk of injury. A person cannot react if there is no warning signal or if there is no time to react.

The potential to cause damage: The physical aspect of the dog is obviously important when doing a risk assessment. A large, strong and young dog can inflict serious damage. The level of bite inhibition is also important. A dog that has bitten a few people but they have only resulted in slight bruises is probably safer than the dog that has only ever bitten once before but caused severe injuries. The intensity of focus and arousal level during aggressive situations is important. If the focus is high, it will be more difficult to interrupt a developing aggressive situation and injury is more likely. The target of the aggression is also important. Babies and young children are more easily injured with less effort than adults. Predatory aggression is the most dangerous as the intent is to kill. Territorial aggression is more dangerous than fear aggression as a territorial dog will go after the target, while a fear aggressive dog may avoid interaction.

Characteristics of the family: Some owners are in denial about their pet's behaviour. Other owners may not have grasped the danger that is present. The amount of activity and the complexity of schedules in some households makes safe control and management of the pet difficult. It is more difficult in a home with young children to provide safe supervision or confinement of the pet. Homes with young children or cognitively impaired adults have family members whom are more likely to put themselves at risk without realising it. The experience of the family with dogs also plays a role. The more experience the more they know about what to expect from them and how to appropriately interact with them. They are more likely to be aware of danger signals and dangerous situations.

The complexity of the behavioural situation: The danger increases when there are many types of aggression present or if the dog reacts aggressively to a variety of stimuli. The presence of other concurrent behaviour problems also increases the risk that aggression might occur. The occurrence of other behaviour problems increases the likelihood of confrontations with the pet. Example the owner might smack a dog for digging which could lead to aggression.

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IMPROVING THE PETS' EXPERIENCE AT THE VET

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ABSTRACT

A visit to the vet can be quite a fearful experience for the pet. However, there are several aspects that can be changed to improve the emotional state of the animal while at the hospital. Small changes can be made to the physical aspects of the hospital including the waiting area, the scale, the consulting room and the kennel area. The way the staff approach and interact with the pet is often unintentionally threatening to the pet. Every staff member must be able to recognise the signs of fear and anxiety, and act appropriately when these signs are seen. There are several low stress handling techniques that would make the handling and restraint less stressful for the animal. Veterinarians should try to employ these techniques, as it would improve the experience for the pet and decrease the change of getting hurt by a fearful animal.

INTRODUCTION

For many dogs and cats a visit to the vet is the worst thing that could happen to them. Basically a life threatening experience. There are several aspects that we as vets can change, that would improve the pets experience during the visit. This does not only decreases the anxiety in the animal, but also makes working with the animal a lot easier, resulting in a better clinical exam, being able to pick up behaviour changes while at the vet and enabling us to make a better diagnosis. A pet that is easy to handle makes the experience more pleasant for the owner too. An owner of a cooperative pet is more likely to bring the animal in at an earlier stage of disease, which will increase a positive clinical outcome.

For a puppy or kitten the first visit will expose it to a lot of new sensory stimuli that might be overwhelming. This includes the noises in a busy practice; the global odour consisting of animal, people and disinfectant smells, meeting unfamiliar animals and people, and slippery floors that interfere with balance and traction. Add to this an over protective client that is clutching at the patient the animal might take this as a signal to react.

It is thus no wonder that most animals never show their true behaviours at most veterinary visits[1]. The main focus must be on decreasing arousal and increasing affiliative behaviours. Patients that are shaking, trembling, drooling, hiding, staying flat on the floor, scanning the environment, urinating, defecating, vomiting or trying to leave are clearly unhappy[1].

The areas where changes can be made include the physical and structural changes (i.e. improving the environment), the way we greet the pets and the way in which we handle the pet. It is also imperative that all the staff in the veterinary hospital is able to recognise the signs of fear and anxiety. This indicates an uncomfortable pet and could lead to fear aggression should the threat to the pet (from the pets' perspective) continue. An important aspect is to get pets used to the veterinary environments at a young age by including a visit to the vet during early development (puppy school) where the pet has a pleasant experience.

THE PHYSICAL ENVIRONMENT

The environment the pet enters sets the tone for the appointment. It is imperative to start with a pet-friendly environment that will put even less-socialised pets at ease[3]. Certain aspects to practice design may decrease stress.

Entrance and reception

The doorway area should be spacious enough so that no animal is constrained to come within its personal approach distance of another animal. Large doors are optimal, but windows, glass doors and large panels of glass in doors also help. Staff can be taught to help ensure safe and non-stressful

movement through doors. If everyone can see who is coming or going then avoidance of crowding is possible.

If feasible, it would be best to have clients coming into the practice through the front door, and leaving through a side or back door. These practices make interactions between clients and patients safer, prevents congestion, keeps noise and confusion levels lower and respects patient privacy.

Dogs that slip on the floor on entry or cannot get grip as they move across the room experience anticipatory anxiety throughout the consultation as they know they have to leave the same way[1]. Rubberised flooring is better.

A reception desk with lower areas will prevent the receptionist having to lean out and over the desk to look at the patient, which could be threatening to the patient. Treats should be at hand, so that the receptionist can give treat to the pet. This builds a positive relationship with the patients and clients.

Waiting room

Many pets are anxious when they go to the vet. They are often fearful of unfamiliar people and/or animals. If they step into the hospital and the first thing they see are unfamiliar people and animals, they could immediately become fearful[3].

Waiting areas are best if they are spacious and flexible. Crowded multi-species waiting rooms that lack dividers can lead to anxiety for both the feline and canine patients. Once aroused, these animals often are fearful or aggressive for the rest of their visit[1]. It would be best to have separate cat and dog areas. Towels can be draped over cat cages.

Providing waiting areas outside the clinic might be advantageous as this prevents crowding and many pets and clients would be calmer than they would be waiting inside the hospital. Some exams and procedures could even be done outside where the patients can focus on other activities and not feel so entrapped[1]. Distressed patients might be calmer waiting in the car than in a busy and noisy waiting room.

Giving the clients some control over where they sit and whom their pets might meet immediately lessens their anxiety. Dogs should be able to sit without being molested or vocally threatened by other dogs. Working by appointment will decrease waiting time and will ensure that there are not a lot of people and pets waiting.

The scale

Weighing the patient can be an anxiety-producing event. Scales are often placed in corners away from heavy traffic. Patients can feel trapped in a corner on a device that makes them feel insecure and without control. The scale should be preferable walk through, and covered with a non-slip mat. It would be even better if it was flush with the floor.

It is less stressful for the pet to be weighed immediately after registration than to get up to be weighed after sitting in the waiting room[1].

Dogs can be taught that being on the scale is a pleasant experience by luring the dog onto the scale and using and positive reinforcement without using any force. This exercise should be included in all puppy schools and socialisation programmes.

Examination room and work areas

The examination room should be large enough to fit a family (two adults and two kids) as well as the pet comfortably. If the humans are crowded and socially uncomfortable, the dog or cat will be worried[1]. The pet should also not feel that it is trapped.

It is important to allow the pet to investigate the room. Dogs especially will feel calmer after being allowed to sniff and examine the room.

Smaller dogs and cats are often examined on the examination table. Use a rubber mat to supply secure footing and prevent slipping. It will also decrease glare from the table and will reduce noise. A

large, unruly and inappropriately behaving dog on a table can be very dangerous especially as his face is on the same level as the humans around him.

Cats should be carefully coaxed out of their boxes. Alternatively the box should be dismantled. The cat can be left to sit in the bottom part of the box during the examination, as this will provide some safety for the cat[2].

Planning for the design of the exam room and the procedures to be done should include the constraint of minimizing the numbers of entries and exits once the patient is in the room. Movement in and out of the room can be stressful for the animal. Ensure that all the equipment needed is in the room as this keeps movement in and out of the room at a minimum. Try and keep the equipment on one side of the room as this decreases having to step over or around the dog all the time.

Kennels

Both cats and dogs should be kept in such a way that they cannot see other animals. The hospital should be kept as quiet as possible. Many high-quality cages are made out of stainless steel and are loud, reflective and smooth. Rubberised mats will make all the difference in how much control the patient has over movement, will dampen sounds and be less reflective. Cages should not face each other. Placing a blanket over the kennel can provide some privacy. Cat cages should provide a hiding spot. Boxes can be placed inside the kennel. A pad on top of the box will provide an elevated spot for cats[2].

A favourite toy, bedding or an old, worn piece of clothing can be left with the pet that needs to be hospitalised. This will give it some form of familiarity and comfort. A radio might be helpful for some dogs.

Hospitalised pets should be examined and treated in a separate consulting room, out of sight of the other animals.

A visit from the owner can be extremely helpful, especially for those pets that do not want to eat in the owners' absence. However, it can also be disruptive for the other animals' in the ward.

Keeping the senses in mind

High noise levels can be a concern for pets. By having phones behind a closed door, a major source of disruptive noise is removed. Metal surfaces are noisy, and should be padded/rubberised where possible. The staff should be encouraged to keep noise levels down. This includes conversations, cleaning regimen and noise during feeding (clanging of metal bowls). Dogs barking in kennels can be stressful for other dogs and the staff alike. Consider putting those patients in a separate area (in a collapsible crate in another room) or place a blanket over the cage to muffle the sound.

Natural light is the best and a large window in each room is preferable. Animals do not like fluorescent light, and if practical, softer lighting should be considered.

Many patients will cringe at the smell of disinfectants, especially those from a rescue organisation. The reduction of certain odours, especially from anal gland excretions is important – for clients as well as pets. Anal sack content signals a warning to other dogs, so smelling this when walking into the consulting room might alarm the dog.

The use of synthetic pheromones is an option. It might not be as beneficial in the consulting room where there is short contact time and the smell will be overwhelmed by disinfectants as well as the visual and auditory signals[1]. However, dogs and cats may well benefit from its use in the kennel, as there will be prolonged exposure.

GREETING AND HANDLING OF THE PATIENT

The way we handle our patients plays an important part in how they experience the visit to the vet. A few small changes in our behaviour towards the pet can make a huge difference and could be the difference between a cooperative dog and a fearful aggressive dog. This applies from the first greeting with the pet, to the examination and how we move the pet around the facility. When possible, low stress restraint techniques should be used.

The support staff should approach no closer than the patients inter-personal approach space and talk to the client. The staff must allow the dog or cat to approach them and sniff them. No one should stare at the pet or make sudden moves. Their arms should be held loose and relaxed and gently and slightly extend an open hand. If the patient backs away the staff member should not reach for the pet. The staff member can back up and continue to speak to the client calmly. The client should not discipline a patient for being worried or concerned. The pet should be moved to a comfortable environment quickly. Treats can be spread around where the client will sit while waiting for the pet to find and learn that the vet clinic is not such a scary place.

It is important that the treating staff takes the time to settle a nervous dog. In a busy practice this might be difficult to do. It is important for all the staff to recognise the signs of fear and anxiety. These might be subtle but should not be ignored. By taking a few minutes to settle an anxious dog might save a lot of time in the long run as the dog is likely be more cooperative. It makes the visit more pleasant for the pet and may prevent injury to the veterinarian.

Practical aspects of how to approach, greet and handle our patients in a low stress way will be illustrated during the presentation and will make up the main part of the presentation.

PREVIOUS EXPERIENCES

Some patients have learnt that visiting the vet is painful. Learned pain can lead to fear and anxiety. Dogs should be desensitised and counter conditioned to coming to the vet. Visits when it is quiet and without having anything done will provide a good experience.

Dogs and cats are very good at reading the body language and auditory and olfactory cues of other dogs and cats. If the client has a particularly calm dog or cat in the household or one who actually likes to visit the vet, this one should accompany the nervous pet. The anxious pet may be too nervous to accept food treats, but the confident pet will not be, and the patient can learn from observing that pet get praise and treats.

PREVENTION IS BETTER THAN CURE

Vaccinated puppies should be encouraged to visit the practice, be handled and put on the scale and examination table using positive reinforcement. The owners can start at home by handling the feet, mouth and ears from a young age. Using treats while doing a procedure will distract the dog from what you are doing and make it a pleasant experience. This is not always doable, but treats should be used where the situation allows.

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OVERVIEW OF INTER-DOG AGGRESSION

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ABSTRACT

Inter-dog aggression between household dogs is one of the most severe behaviour conditions. Severe injury is most common. In inter-dog aggression there is a consistent voluntary aggression towards another dog that is out of context given the social signals, threat circumstances or the response from the receiver of the aggression. At least one of the four skill sets that normal dogs should possess is impaired in the aggressors in inter-dog aggression. Dogs who exhibit out-of-context or inappropriate responses to other dogs actually suffer from anxiety disorders. The aggressor tries to control the victim through the use of subtle threats and challenges, which include displacement of the victim, control of the victim, and the threat of the victim. In the development of this condition there will be more posturing and vocalisation, but as the condition progresses these clear signs diminish. Snarls become silent and attacks may occur with little or no warning. Management is the key part of treating this condition. The success of behaviour modification will depend on the depth of the pathology of the aggressor and how early in the course of the violence the problem was recognised. Medication might be indicated, and might be needed for the aggressor and the victim.

INTRODUCTION

In general, aggression refers to threatening or harmful behaviour directed toward another individual or group[3]. Aggression can include a variety of behaviours. It can be a subtle change in body language or a full blown aggressive attack. There are different types of aggression and the classification of aggression can be controversial[3]. The main motivations for aggression are[1]:

- Competition to gain an immediate share of resources or to gain the status that will guarantee permanent control over them
- Self-defence or defence of the group
- Defence of resources that are already held by the individual or group (including territory)

These motivations exist in every dog but whether they come to the fore as aggression will depend on the availability of resources, the dogs' personality, the dogs' ability to communicate, its experience and the behaviour of social partners (including people)[1].

In *normal* aggressive behaviour the dog displays aggression in circumstances that warrant aggression and the dog is able to inhibit the aggression and modify the response based upon the relative threat. Aggression is *abnormal* when the dog perceives a threat where it does not exist and when the dog has trouble to modify its response to the threat as the threat changes.

In *offensive* aggression there is an unprovoked attempt to gain some resource at the expense of another while in *defensive* aggression the victim aggresses toward another that it perceives as an instigator or threat[2].

It would be beneficial to see the aggressive display (either directly or a video) but this is not always practical and safe. One may therefore have to rely on the behaviour history to make the diagnosis. There are a number of criteria that are used to differentiate the type of aggression. These include[3]:

- Type of vocalisations
- Postures and behaviours
- Context or situation in which the aggression occurs
- Stimulus or the trigger for the aggression
- Target of the aggression
- Temperament of the pet
- Signalment
- Health of the pet
- Location where the aggressive behaviour is likely to occur

Although many types of aggression are described, aggression is often multifactorial or has multiple causes. Dogs that are highly aroused are at higher risk to display aggression as their emotional state can interfere with the ability to respond rationally to stimuli. The environment, circumstances and the presence of people may influence the manifestation of aggression. Other influences include medical conditions, pain, learned behaviour and owner reactions to the aggression. Owners may inadvertently reward the aggressive behaviour.

DESCRIPTION OF INTER-DOG AGGRESSION

It is normal that dogs can have some disagreements. However, in inter-dog aggression there is a consistent voluntary aggression towards another dog that is out of context given the social signals, threat circumstances or the response from the receiver of the aggression[4]. Inter-dog aggression is associated with social changes in relationships and interactions that occur as animals move through social maturity. However, either hierarchy or social maturity does not define it. This pathology depends on and is defined by the contextual response of the dogs involved[4]. With the development of maturity changes in social relationships, behaviours and perceived changes in the relative social hierarchy occur. The individual who is concerned about these maturation related changes can be the aggressor (offensive aggression) or the victim (defensive aggression)[4].

The pathology for inter-dog aggression is unknown, but at least one of the four skill sets that normal dogs should possess is impaired in the aggressors in inter-dog aggression:

- Reading signals correctly
- Correctly processing and understanding the information in those signals
- Making a plan to act appropriately on the signals
- Successfully and clearly signal and act on that plan

Dogs who exhibit out-of-context or inappropriate responses to other dogs actually suffer from anxiety disorders. They cannot adequately assess the risk associated with the other dog and so provoke the situation in an attempt to get more information or to pre-empt any challenges. However, every time they try to get information, they cannot use that information so they become more anxious and aggressive[4]. The dog who is victimised learns that the dog threatening or attacking him is unreliable. With repeated exposure to aggressive interactions, both dogs in the interaction become more anxious and reactive and often more aggressive[4].

Inter-dog aggression usually manifests itself between known dogs, especially dogs that live together. The affected dogs are sometimes seen as chronic bullies. Bullying may not escalate, but inter-dog aggression almost always does[4].

COMMON NON-SPECIFIC SIGNS

There are usually subtle signs where one dog tries to control the other dog. Owners, however, often miss these signs. They only notice that there is a problem between dogs when there is an outright fight. The aggressor tries to control the victim through the use of subtle threats and challenges, which include displacement of the victim, control of the victim, and the threat of the victim[4]. Challenges include:

- Blocking access to resources (food, bed, furniture)
- Lying or pushing themselves on the other dog
- Stealing the other dog's food, chews or toys
- Shoving past the other dog to get into or out of a door or car first
- Using halls, doorways and steps as situations to control the other dog and its access to other areas or escape from them
- Staring or vocalising
- Frank aggression

These behaviours can occur alone or in combination and may be self-limiting but can also escalate within a few minutes to an outright fight with grabbing and biting. On their own, these behaviours are designed to obtain information from the other dog, but the information received has virtually no effect on the course of the behaviours of the aggressors. In the development of this condition there will be more posturing and vocalisation, but as the condition progresses these clear signs diminish. Snarls become silent and attacks may occur with little or no warning. Early in the development of the

pathology the aggressor more likely grabs the shoulders, dorsal neck and legs. Later on the aggressor grabs the sides, under belly and the ventral neck and shakes and twists the skin and/or the dog. Later the attacks share many commonalities with predatory attacks[4].

DIFFERENTIAL DIAGNOSIS

There are a few conditions that need to be ruled out when dealing with inter-dog aggression. These include:

- Endocrine conditions (especially thyroid) as well as seizure disorders which may become apparent as dogs move through social maturity.
- Pain related conditions as aggression can be induced when one dogs bumps into another or when they have close contact.
- Any infectious or toxic agent that can affect behaviour. The recipient of the aggressive behaviour from an ill housemate knows that the pattern is not associated with their own behaviour but with something else. They will either avoid or try and help and care for the dog who is behaving inappropriately.
- Geriatric dogs may become intolerant of other dogs due to a decline in the senses (can't hear or see them) and because they are painful, arthritic and slower and less able to react. However, aging dogs will not seek out other dogs to victimise, and aggressive responses are most likely normal contextual responses.

AETIOLOGY, EPIDEMIOLOGY AND RISK GROUPS

Inter-dog aggression generally appears at social maturity (18- 24 months of age). A common scenario is that dogs that grew up together peacefully suddenly start fighting at the age of around two years. Inter-dog aggression occurs more between dogs of the same sex, and more so between young female dogs than young male dogs.

The aggression appears to be about social relationships between the dogs and how the aggressor perceives its relative social status or role and its control over that status /role.

TREATMENT

Management is the key part of treating this condition. The most important aspect is to relieve everyone's uncertainty and keep everyone safe. Clients must avoid aggressive events at all costs. Clients must be told in no uncertain terms that they may never have peace and that 'love' is not enough to change the situation. These dogs can never be left alone unsupervised. The aggressor can be locked in another room or area in such a way that the door cannot be accidently opened. They must not have visual contact so that there can be no threats. The victim is always given free range.

Using a head collar, the dogs may be able to be walked together, but often separate walks are necessary. If these dogs need to go in the same car together, the aggressor should be locked in a crate or behind a metal grate, in such a way that it is not able to see and stare at the other dog.

In households where the dogs sleep with the owners the sleeping arrangements will have to be adapted. Both dogs cannot sleep with them. The victim should be the dog that sleeps with the clients. Should the clients wish to have the aggressor sleep with them, they should watch for increased threats or aggression the following day. If this is noted the aggressor should not sleep with the clients.

Clients should not try to separate the dogs should there be a fight. This commonly results in the client being bitten. Blankets, brooms or cardboard can be used to separate the dogs. However, if they are truly separating the dogs a fight should not occur. A sealed can of soda water can be kept at hand. Should there be a fight, it can be shaken and sprayed on the dogs. Fighting dogs will often separate. However, this technique will only work once or twice before the dog learns to fight through it. Water (hosepipe under pressure or bucket) can be used. Air horns (pet corrector) can help, but the dogs will go back to fighting if they are not separated. Do not grab the dogs by the back legs and lift them to pull them apart, this often results in the dog just biting harder. Trying to pull dogs apart will often cause the dog to bite harder and tear injuries can cause serious damage. It can also lead to redirected aggression.

It might be that desensitisation and counter conditioning is too risky. However, both dogs will still benefit from behaviour modification to help them become calmer and less reactive.

The success of behaviour modification will depend on the depth of the pathology of the aggressor and how early in the course of the violence the problem was recognised. Once serious injury has occurred, the victim might become anxious and fearful. In mild cases with relative early intervention, behaviour modification can teach each dog to relax, not react and to take cues about the appropriateness of their behaviour from the owner. Here dogs are taught that inappropriate and reactive behaviours will not be tolerated or rewarded, but calm, non-threatening, delayed behaviours will be rewarded. Dogs are taught to look at the owner for signals that will keep them safe.

Medication might be indicated, and often the aggressor and the victim need medication. It should however be stressed that medication is not a quick fix and should always be accompanied by management of the aggression and behaviour modification.

The following drug choices can be considered:

- If the aggressor is explosive the selective serotonin reuptake inhibitor (SSRI) fluoxetine might be the best first choice medication.
- The victim needs to be treated with tricyclic antidepressants (TCA) or SSRIs
- If the victim becomes hypervigilant, gabapentin might help
- If the victim has become withdrawn, a benzodiazepine such as alprazolam may help. It can also stimulate appetite so that they are happier to work for treats.
- If a fight occurs, both dogs should be given a BZD (alprazolam) as soon as possible in an attempt to interrupt memory from being made.

Other causes for aggression between familiar dogs

Redirected aggression: Aggression is directed towards a third party when the aggressor is thwarted in or interrupted from exhibiting aggressive behaviours to the primary target. The aggressor actively pursues the third party, especially if they were associated directly with the interruption of the aggressors' behaviour.

Food-related aggression: This involves aggression that is exhibited in the presence of, and only in the presence of, food in the absence of starvation. There is usually lots of warning and signalling. Food guarding without ingestion is common.

Resource aggression: Conflict occurs when a resource is available, with dogs showing no animosity towards each other at any other time. Competition for resources is influenced by the availability of a resource, the value that each individual places on it and on any existing status relationship between the dogs[1].

Status related aggression: the condition in which a member of the social group consistently controls resources or the behaviour of others in the group is referred to as dominance. Aggression may be exhibited when an individual dog perceives that it is being challenged or is losing control of a resource or situation to a subordinate[3].

Fear aggression: this aggression is triggered by a stimulus that appears threatening to the dog. It usually occurs when the dog is unable to avoid the stimulus that it is fearful of[3].

CONTRIBUTING FACTORS

Lack of socialisation: During early socialisation the dog learns how to recognise and interact with its own species and other species. Dogs, who lack socialisation may not be able to read, interpret and act appropriately to signalling by another dog. This can be a contributor to aggression.

Inability to signal: there are several situations in which the dog cannot signal appropriately to the other dog its intentions. This can occur when the dogs are in close proximity, in a confined space (corridor, entrance hall, door way) or prevented by the owner (on a tight lead, held). The victim is not able to show appropriate body language. The aggressor sees this as a threat and responds with aggression.

This can also happen where the anatomy of the dog is such that it hampers the expression of normal body language (droopy lips, lots of facial hair, short tail).

COMMENTS

Inter-dog aggression between household dogs is one of the most severe behaviour conditions. These dogs can and do kill each other. Severe injury is most common. Clients need to be educated that their dogs at home are not a pack. The concept of pack implies that all dogs were born into the family group and grows up knowing all the other dogs, who are in some way related to them. In most households unknown dogs of all ages are brought together. These dogs are not a pack. The assumption that everyone should get along as a family group is dangerous[4].

This is not an easy condition to manage. It is difficult to keep dogs separated at all times and needs a lot of planning and effort on the owner's side. Quality of life for at least one of the dogs may be reduced. Many owners believe and long for peace in their home. This may sabotage their efforts to keep the dogs separated. In many households there is at least one more severe fight after diagnosis before they are convinced of the risks and the benefit of intervention and separation[4].

If the clients decide to find a home for one of the dogs, they should rehome the 'normal' victimised dog. No other dogs should be introduced into the household while the aggressor is alive.

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NOISE PHOBIA WITH EMPHASIS ON THUNDERSTORM PHOBIA IN DOGS

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ABSTRACT

Sonophobia (noise) is the commonest phobia in dogs; many of these develop from loud noises that have created a severe sound sensitivity and generalisation. Fear is part of normal behaviour, is essential for survival and is a highly adaptive response. Phobia, however, is always maladaptive and can seriously affect the individual by limiting its ability to engage in normal responses. Thunderstorm phobia is a sudden and profound, non-graded, severe response to thunderstorms or any aspect of them. How a dog reacts to certain stimuli depends on genetic influences as well as through learning and experiences. They can respond in an active way or have a passive response. Behaviours include pacing, panting, hiding, shaking, elimination and vocalisation. Treatment consists of immediate treatment when the threat of noise is unavoidable as well as long-term resolution of the problem. Treatment should have a multimodal approach and includes environmental management, behavioural therapy, pheromone treatment, pharmacological therapy and complimentary medicine. Although many cases will improve, some pets may still show some response to noise after treatment and some animals may need lifelong treatment.

INTRODUCTION

Noise phobias are of the most common recognised disorders associated with panic or phobic responses in dogs[10]. Fireworks and thunderstorms appear to be the most frequent triggers[4]. It can have a significant effect on the quality of life of the dog. These conditions are likely to worsen if they are not treated appropriately.

A fear of noises is a feeling of apprehension associated with a noise. This behaviour may be normal or abnormal depending on the context. A noise phobia is a profound, persistent, and excessive fear of noises and includes a sudden, non-graded, extreme response to noise. The response is considered as abnormal in relation to the context[6].

The way a dog reacts to a stimulus depends on two aspects. Firstly there is the genetic influence on behaviour. This is the species and breed specific response, which is established over generations (nature). Secondly, there is the individual aspect, which is established through learning and experience (nurture). The acceptance of a stimulus as 'normal' arises from early experiences. Inadequate exposure to noises during early development may play a role in the development of noise phobias. It can develop due to a learned aversion as result of a particularly traumatic or aversive noise-associated event[2, 6].

It has been estimated that about 20% of pet dogs suffer from noise phobia. A phobia can develop at any stage but many are apparent within the first year of life. Fearfulness can exist as a temperament trait in animals and describes the tendency for an animal to respond in a fearful way to its environment[7]. Herding breeds are over represented with thunderstorm phobia[1,9].

The distinction between 'reactivity' and 'phobia' is vague. Well-controlled dogs can look reactive. It is not known if all reactive dogs will become phobic, given time and exposure. Dogs who respond with concern to noise but lack the complete phobic criteria should be labelled as reactive and closely monitored[9].

As the symptoms of noise phobias are often not a cause for concern for the owners, owners tend to treat the pet themselves or to tolerate sound phobias, particularly when they are seasonal in nature[4]. This leads to diagnosis and treatment when the case has developed into severe behavioural changes, worsening the prognosis.

DEFINITIONS

Fear is the *apprehension* of a stimulus, object, individual or event. It is part of normal behaviour, is essential for survival and is a highly adaptive response[4, 8]. The animal's behaviour is directional and related to the location of the stimulus. The animal will try to reduce the sensation of fear by repelling, escaping or evading the source. Should this not be possible, then the animal will freeze and wait for an opportunity to engage in another response, or engage in a displacement activity. The fear is terminated when the stimulus is removed. The level of fear is graded according to the degree of the threat[2].

Anxiety is the *apprehensive anticipation* of a threat. It serves to broaden the animal's attention so that it is prepared to react to any threat. It occurs mostly in unfamiliar situations or where the animal has had aversive experiences before[2].

Phobia is an *irrational fear* that is out of proportion to the actual level of the threat. A phobia may develop with any stimulus that can evoke normal fear. Sonophobia (noise) is the commonest phobia in dogs; many of these develop from loud noises that have created a severe sound sensitivity and generalisation[2]. Phobia is normally an 'all or nothing', profound, emotional response, which is always maladaptive and can seriously affect the individual by limiting its ability to engage in normal responses[4]. Phobic fear persists long after the actual threat has gone away[2, 8].

Thunderstorm phobia is a sudden and profound, non-graded, severe response to thunderstorms or any aspect of them. Various weather elements become predictive cues and the animal shows behavioural signs earlier in the storm sequence (generalisation). Animals can become sensitised to changes in barometric pressure, electrostatic field or other factors associated with a storm such as lightning, darkening of the sky, wind and thunder[5, 6, 7]. The response manifests as intense avoidance, escape or anxiety and associated with the sympathetic branch of the autonomic nervous system[9].

Dogs will find a way to avoid having the same experience again. They start to associate events that predict the one of which it is frightened. These cues are likely to generalise, and the dog starts to mount an escape response when it hears similar noises. This process is called *generalisation*[2].

A dog that is able to predict a phobic event will be able to find an escape route or find somewhere to hide before the phobic event takes place. This is part of the coping strategy. Should a dog not be able to predict the phobic event, its ability to cope is undermined. Dogs that are confined cannot escape, may become anxious about being confined, and will attempt to break out. In some situations the owner may become vital in dogs' coping behaviour. These dogs seek comfort from the owner, and this behaviour must not be confused with separation anxiety or an over attachment problem[2].

TAKING OF THE HISTORY

The noises or types of noises that trigger the behaviour must be identified. The owner must describe the actual behaviour of the pet, and indicate whether there have been any changes in the behaviour. The first incident can shed light on the origin of the development of fear as well as any associations the animal has made between certain sounds and situations.

The onset, duration, intensity and progression of the behaviour must be established. The owner should be questioned as to how they respond to the behaviour, what they have done to treat the pet and whether this has been helpful. To differentiate noise phobia from separation anxiety, the behaviourist must establish whether the behaviour occurs in the presence and/or absence of the owner.

BEHAVIOURAL SIGNS

Animals react in different ways. They can respond in an active way (increased motor activity and increased heart rate) or have a passive response (the animal freezes or appears quiet). Fear is usually indicated by flight and escape responses, expressive facial and body signals, and physiological responses (increased heart and respiratory rate, muscle tremors).

The pet may show some or several of the following behaviours: restlessness, shaking or trembling; increased vigilance; increased startle response; bolting/hiding; panting; drooling; tucked tail/ ears backwards/crouched posture; destructiveness; defecation and urination; vocalisation; withdrawal; self-mutilation; inappetence; vomiting; expression of anal sacks; owner or comfort seeking; yawning; increased blink frequency[7]. Profound fearful responses may include immobility or freezing[7, 8].

Fear and anxiety in response to storms can manifest in a number of ways. The behavioural responses are similar to those of other noise phobias and include pacing, panting, hyper-vigilance, whining, trembling/shaking, hiding, salivation, mydriasis, vocalisation, following the owner, destruction, attempts to escape, freezing, and elimination. House soiling while the owner is away might be the only complaint since the owner is not there to witness the other signs[1,3,6,9]. The intensity of the response may vary with the storm itself. The dog may act differently when the owner is present compared to absence of the owner[6]. When there are other dogs in the household, dogs appear less reactive and have a more rapid recovery[1].

DIFFERENTIAL DIAGNOSIS

1. Any conditions with non-specific signs of fear and/or anxiety
2. Separation anxiety
3. Claustrophobia (barrier frustration)
4. Play and exploration may lead to destructive behaviour
5. Medical conditions causing inappropriate urination/defecation
6. Auditory sensory changes
7. Cognitive dysfunction

DIAGNOSIS

The diagnosis is made on basis of the history, presenting signs and to rule out other behavioural problems that may show a resemblance in behaviour.

A high percentage of dogs with noise phobia also suffer from some degree of separation anxiety[1,10]. It is therefore important that a pet diagnosed with noise phobia must also be screened for separation anxiety (and treated for it). Reaction to noises should occur whether the client is home or not.

TREATMENT

Owners are usually most concerned about the more active responses (destruction, vocalisation, pacing, or escape behaviour) whereas passive behaviours (catatonic state, withdrawal, hiding) are often overlooked[6].

Safety considerations for the owner as well as the pet are important. The dog may panic, and attempts from the owner to restrain the dog may lead to aggression. The dog may be a safety risk to itself in attempt to escape, breaking through a window and injuring itself[6].

The treatment consists of immediate treatment when the threat of noise is unavoidable as well as long term resolution of the problem. Exposure to the phobic stimuli must be avoided or limited as much as possible. Unfortunately, exposure to thunderstorms cannot be avoided.

The most important aspect of *environmental management* is that the dog must have a reliable hiding or escape area that is available at all times. It will be beneficial if the hideout is positioned in such a way that it decreases the intensity of the phobic event. In this area the sound should be muffled by blankets or pillows and darkening the area is helpful. Blocking windows can reduce the effect of lightning for thunderstorm phobias and the playing of background music or supplying white noise can block out thunder to some extend. Offering fun toys and playing with the dog may distract the dog[6]. A noise-phobic pet should not be left alone during an anticipated noise exposure.

Behavioural therapy consists of desensitisation and counter-conditioning and is necessary if there is to be any long-term resolution of the phobic condition[4]. There is no place for punishment, as this will aggravate the condition. The setting of routines will create a predictable environment for the dog and decrease general anxiety.

Desensitisation is the process used to decrease the dog's emotional reaction to the phobia-inducing stimulus. It involves repeated neutral presentation of the stimulus alone until the stimulus ceases to produce a significant emotional response. The aim is to play sounds at a level that is below the threshold for inducing a fear reaction. The dog may be aware of the sounds, but not flee from them. Once the dog does not show any reaction to the noises, the volume is increased to a slightly higher level. Should the dog show a reaction, the increments are stopped and exposure is continued at that level until the dog shows no fear of the stimulus. Through a process of repeated exposure and gradual increase in sound volume the dog ceases to react to the noise at any level. At this stage the process of counter-conditioning can begin[2, 4].

Counter-conditioning involves the association of the desensitised stimulus with something positive and enjoyable. The same stimulus is paired with events that produce a positive, emotional response and conflicts with fear. The ultimate result is that when the fear-provoking stimulus is experienced, an emotional swing towards a relaxed and happy emotional state takes place[2, 4].

This treatment takes weeks if not months to complete successfully. The client must be aware that the animal's response to a recording does not predict its response to the live event. However, recording-based programmes undoubtedly offer benefit in many cases[7]. During training the pet should not be exposed to the fear-evoking stimulus.

Clients that reassure the dog during a phobic event are inadvertently rewarding the behaviour. Clients need to calm the dog without trying to reassure it. The owners should show indifference or even a positive response to the noises themselves.

Canine pheromone products have been shown to reduce the signs of noise phobia and often bring significant relief[7]. It is also very beneficial during behaviour modification therapy.

Pharmacological therapy can be used either as short-term strategy to enable the patient to cope with an inevitable event or circumstance, or as long-term treatment, which assists in the application of behavioural modification techniques over a period of weeks and months.

In the short-term, the aim is to limit the negative consequences of the phobic event and the memory blocking properties of the benzodiazepines make them suited for this purpose. The long-term treatment aims to improve the response to behavioural therapy. In general, dogs that show inhibited behavioural responses will benefit from therapy with a mono-amine oxidase B inhibitor, while those that panic are more likely to require medication with one of the SSRIs[4].

Drugs that are used for fearful and phobic behaviours are[5, 6, 9,11]

- Benzodiazepines: episodic, acute, short-acting, anxiolytic medication for short-term treatment
    - Alprazolam:
        - Canine: 0.01-0.1 mg/kg bid routinely or every 4 to 6 hours as needed, starting dose 0.02 – 0.04 mg/kg, max 4 mg/dog/day
    - Clonazepam:
        - Canine: 0.125 – 1.0 mg/kg bid-tid
    - Diazepam:
        - Canine: 0.5-2.2 mg/kg at least 1 hour before anticipated storm, repeat every 4 – 6 hours as needed
- Clonidone: can be considered if the dog does not react to benzodiazepines or only partially responds. Clonidone helps to decrease the peripheral sympathetic response and may aid in learning new ways
        - Canine: 0.01 – 0.05mg/kg as needed every 12 hours, maximum 0.9 mg total for medium dog range
- Serotonergic medications: chronic, continuous, long-acting, anxiolytic medications
    - Fluoxetine:
        - Canine: 0.5 - 1.0 mg/kg sid-bid
    - Clomipramine:
        - Canine: 1-3mg/kg bid
    - Selegiline:
        - Canine: 0.5 – 1.0 mg/kg sid in the morning
    - Amitriptyline:
        - Canine: 1-2 mg/kg bid
    - Sertraline:
        - Canine: 1 mg/kg OID to start, can increase to 2 mg/kg sid if monitoring for serotonin based side effects is excellent and ongoing

Phenothiazines have been associated with potentiation of the fear response and are not recommended for general treatment[7].

Massage therapy (Tellington TTouch), an enveloping body suit to reduce anxiety (Anxiety wrap), and a static-minimising cape (Storm Defender) for use in animals are *complimentary treatments* that have anecdotally been helpful[7].

PROGNOSIS

Fears learned as a result of a traumatic association are generally more easily managed than those associated with problems in development. An improvement in over 90% of animals with noise fears is possible, but complete resolution is infrequent[7]. The earlier the case is treated the better the outcome[5]. Some cases need lifelong treatment. The pet may relapse after exposure to a traumatic noise-associated event.

In dogs with thunderstorm phobia profound responses may be diminished, but the dog may still pant, pace or stay close to the owner. Animals showing severe panic attacks may be difficult to control during a storm. Coexistence of other anxieties worsens the prognosis.

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VNASA ABSTRACTS

DELVING DEEPER INTO BITE WOUNDS

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ABSTRACT

Bite wounds present an emergency situation in the veterinary patient. These patients require intensive management of both the wound and the patient attached to the wound. Bite wounds can lead to systemic shock and death rapidly if not monitored carefully. The old use of adherent dressings in non-selective debridement of necrotic wounds is not acceptable anymore. The use of moisture retentive dressings, which promote a warm, moist wound environment, are currently the best treatment for wounds. Staged selective surgical debridement is then done on a daily basis to remove necrotic tissue. Wounds should only be sutured once a healthy bed of granulation tissue has formed. However this needs to be taken with a degree of common sense. Each wound should be treated on its own merits. There is no checklist to manage every type of wound. Thoracic and abdominal wounds present the possibility of severe underlying life threatening conditions that need to be quantified as soon as possible. Bite wounds to the limbs should be radiographed to check for underlying fractures.

DEALING WITH BITE WOUNDS

Bite wounds are a devastating injury that is often under estimated in veterinary medicine. I am sure many of us can attest to the pain caused by a work related bite injury to ourselves. One can only imagine the pain that the animal is suffering that comes in with as Dr Millward once put it with the uniquely South African ‘combine harvester’ bite wounds.

The biggest mistake made with bite wounds is trying to fix them to soon and not taking into account the crushing trauma caused by the bite of a dog. By trying to fix them to soon we forget that the tissue has undergone penetrating trauma from the teeth as well as crushing trauma form the jaws and shearing injury from movement while biting, which will only be evident in 3-5 days time. This crushing and shearing trauma is the real damage done from bite wounds. It damages vascular supply to the tissue releases cytokines and inflammatory mediators en mass and completely destroys tissue under the skin. This is why we forget about it because we often don’t see it. This has coined the adage of the iceberg effect of bite wounds. We only see the small home in the skin from the canine, we don’t see the fractures, avulsed intestine, shredded subcutaneous tissue and muscle and the severe inflammatory reaction caused by this.

This has to always be taken in reason and common sense. For example Mrs Jones’ poodle was bitten in the park by Mrs Smith’s Yorkshire terrier and has a tiny laceration on the neck. To try and make Mrs Jones come back daily for bandages and convince her 2 weeks later to try suture the wound will not go down to well. There will be unnecessary financial and inconvenience attached to this management strategy. There always has to be reason applied to the management of all wounds not just bite wounds. The mantra is if in doubt rather leave the wound open for a few days and see what happens. You can always suture at a later stage, you can never go back and un-suture a wound once it starts with wound dehiscence. Each wound is assessed on its own merit in deciding a management strategy no single calcification holds water for all wound types.

Cats with bite wounds present a different problem all together. These should generally be left until there is good granulation tissue and then closed. Cats develop poor granulation tissue when compared to dogs and have a higher incidence of wound breakdown. They have a high incidence of non-healing pocket wounds, which will only heal under no tension and generally, require an axial pattern flap to close. For the purpose of this talk we will discuss getting the wound to the point it can be sutured not how to closed the wound.

Initial wound management on presentation is very important as to how the outcome of the wound will be. Initial wound management is aimed at reducing the microbial burden of the wound, preventing further contamination and managing the patient's pain. One thing that cannot be forgotten is the patient attached to the wound. These patients can be in a severe state of shock, large bite wounds can lead to massive losses of serum protein, these patients can be in marked hypovolemic shock form SIRS and loss of blood. The life of the patient is more important that choosing the right dressing for the wound. Stabilise the patient first, prevent contamination then address the wound environment.

Wound irrigation is one of the simplest and most effective methods of decreasing microbial burdens of wounds. Wound irrigation physically removes the debris and contamination. The most important this to remember is large volume of fluid and then some more! There was no difference in infections when large volumes of tap water were used when compared to large volumes of sterile isotonic fluid. This demonstrates just that volume as apposed to type of fluid are important, it is best to use a sterile isotonic fluid to flush wounds in the clinical setting. A Lot has been said about the pressure used to irrigate the wound. There is no general consensus on what is best. The easiest is to create a flushing device made from a 2ml syringe, a drip bag, a 3-way stopcock and a drip line. This can be used to flush wounds each day for the same patient, all that is needed each day is a new Jelco^{tm} or other over the needle catheter to flush with.

Antimicrobial therapy is controversial. The general consensus is topical agents are useful in the acute stage when there is limited vascular supply to the wound area. Once granulation tissue has formed then there is no longer any need for any type of antibiotic. The current recommendation is that systemic antibiotics should be used for chronic wounds based on culture and topical antimicrobial solutions used for acute wounds.

The protective layer or wound covering placed on the wound has developed dramatically in the last few years. Our understanding of the wound environment has changed leading to these changes in how we manage wounds. Mechanical debridement or the classical wet to dry dressing, (a swab soaked in ringers removed daily once dry) is no longer indicated in wound care. The wet to dry dressing causes non-selective debridement of the wound. They dry out the wound surface inhibiting wound healing and leave lint and fibres behind in the wound.

The standard for debridement is now considered selective staged debridement. This is prepared under aseptic conditions starting superficially moving deeper removing all devitalised tissue leaving questionable tissue. The questionable tissue is then assessed the next day and removed if deemed devitalised. This should be done on a daily basis. Non-surgical debridement with various enzymes or chemical agents can be used. Enzymatic debridement may dehydrate normal tissue and in non-selective. Hyperosmotic agents such as Honey have shown great promise in topical treatment of wounds and are fast becoming the latest in treatment for wounds. Honey has both a hyperosmotic effect and a strong antimicrobial effect. Studies are showing a significant positive effect in wounds treated with honey with lowered incidence of resistant bacterial infections.

The currently trend for wound healing is to provide a moist warm wound environment. This facilitates debridement, enhances granulation tissue formation, and speeds up epithelisation. This is brought about by maintaining the autolytic enzymes at the wound site instead of constant removal, as was the case with wet to dry dressings. The mainstay of this therapy is to apply hydrophilic dressings to the wound. These dressing retain moisture from the wound thus keeping the environment moist at all times. These dressings are made from seaweed (alginate) polyurethane foam or maltodextran flakes. The most commonly used dressing is the alginate dressings made from seaweed. These dressings are potent hydrophilic agents and are often combined with silver sulfadiazine, honey or topical antibiotics to treat specific wounds. Bioscaffolds and synthetic matrix dressings provide a matrix for extracellular matrix formation. These stimulate angiogenesis and seem to be more useful in management of chronic non-healing wounds.

Negative pressure wound therapy is a new treatment option that has come about form the human literature to treat diabetic ulcers. This entails placement of foam dressing over the wound this is then covered by an occlusive dressing and connected to a drainage pipe. This is then connected to a continuous suction. This has shown positive results in some studies but not conclusive proof to be better than any other therapy. The apparatus can be cumbersome and difficult to maintain in animals.

All of these dressings can be used in the inflammatory phase of bit wounds to aid in debridement and promote granulation tissue. Severe bite wounds should only be closed once a healthy bed of granulation tissue has appeared. Once there is granulation tissue the decision to closed the wound or let it heal by second intention is made. This is made depending on the size, position and availability of skin for closure.

As mentioned before the wound is connected to a patient and a few important things need to be mentioned in regards to the patient. Abdominal or thoracic bite wounds pose a serious risk in the big dog biting small dog situation. In these cases thoracic radiographs and abdominal ultrasound are indicated. In abdominal bite wounds and obvious hernias to the abdominal wall emergency celiotomy is advised as soon as the patient is stable. These patients have a high risk of damage to a hollow viscus. If not managed rapidly septic peritonitis will develop with a guarded prognosis. Thoracic bite wounds rarely require surgery, they are often managed with thoracic drains till air stops developing.

Any bite wounds to a extremity should have radiographs taken of the leg. The force of a dog bite wound is easily capable of causing fractured bones. These need to be managed as an open fracture and are one of the few cases for intra-venous antibiotics and immediate stabilisation of the fracture once the patient is stable.

Bite wounds present a challenge to the veterinarian and their team. The most important steps are, stabilise the patient first without further contamination of the wound, monitor the patient for deterioration (plasma albumin, glucose, electrolytes) flush the wound, initiate debridement and check for underlying factors that could be life threatening.

FURTHER READING

1. Tobias and Johnston Veterinary small animal surgery volume two

2. BSAVA handbook on wound management

3. Fossum third edition small animal surgery

TO SUTURE OR NOT TO SUTURE

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ABSTRACT

There are vast types of suture materials available in veterinary medicine and the choice of which one to select can be a daunting task. The cost of having every type of suture material and needle is non-viable and unnecessary. By carefully selecting the best possible suture material, needle for your practice one can practice high quality veterinary medicine at a nominal cost. Certain suture materials are superior to others but there is no perfect suture material. Some materials cannot be used in certain situation and this needs to be common knowledge for the theatre nurse. The hope of this paper is to provide you with a reference to base your decision of which suture material to choose where.

INTRODUCTION

There are a vast number of suture materials available to the veterinarian. It can be difficult to decide on which material to buy to keep a reasonable range of stock for different conditions while being financially viable. There is no one-suture material that meets all the requirements of the supposed perfect suture material.

The ideal suture material is easy to handle, causes minimal tissue reaction, inhibits bacterial growth, has excellent knotting ability, resists shrinking in tissue, non-capillary action, non-allergenic, non-carcinogenic, non-ferromagnetic and absorbs with minimal tissue reaction.

When selecting a suture material the smallest size material that will support the tissue while it heals should be selected. This will minimize the trauma associated with passing the suture material through the tissue. And reduce the amount of foreign material in the wound.

In general suture materials are classified in relation to how they behave in tissue, are they completely absorbed in time or are the not absorbed in tissue. They are further broken down into their structure, are the monofilament or multifilament. These classifications have major implications in how each of these suture materials behave and where they can and cant be used. In understanding suture material one has to have an excellent understanding on the specific healing rates of specific tissues. This allows the surgeon to select the best suture material for that specific tissue.

Absorbable suture materials will gradually and consistently lose their tensile strength over a time period and eventually disappear from tissue completely. The process for their dissolution is phagocytosis by macrophages or hydrolysis, this depends on the type of suture material and how it has been manufactured. Some of these materials like triclosan-coated (TC)-monofilament polydioxanone have been coated with an antibiotic solution that will be released as the material is hydrolysed. This limits the attachment of bacteria to the suture material rather than treating wound infections. Polyglycolide and polydioxanone are made from lactic acid polymers, which will release lactic acid as they hydrolyse. This will inhibit bacterial adhesion to the suture material.

Non-absorbable suture materials are not absorbed or broken down by the immune system. Table 1 shows a breakdown of the commonly used suture materials in veterinary practice. Braided nylon sutures (Vetafil) should never be used. They predispose to fistula formation when used in tissue and they cause a marked tissue reaction when used in skin and have dramatic tissue drag. They may contribute to wound infection by wicking bacteria from the skin deeper into the wound.

Recently the advent of barbed suture material, which is so called knotless suture material, has been developed. This has tiny barbs on the length of the suture that will engage tissue and prevent pull out of the suture and maintain the tension without having to tie a knot. The author has no experience with this material and it is not available in South Africa to my knowledge.

Table 1.

Suture material	Common name	Suture type	Rate of loss of tensile strength	Complete absorption	Knot security	Tissue reaction
Chromic surgical gut	Catgut, surgigut	Absorbable multifilament	33% at 7 days 67% at 26 days	60 days	++	+++
Polygalactin	Vicryl, Viamac	Absorbable multifilament	35% at 14 days 60% at 21 days	60 days	++	+
Polygalactin 910	Vicryl rapid	Absorbable monofilament	50% at 7-10 days	35 days	++	+
Polyglycolic acid	Dexon	Absorbable multifilament	35% at 14 days 65% at 21 days	60-90 days	++	+
Polydioxanone	PDS	Absorbable monofilament	14% at 14 days 31% at 42 days	180 days	++	+
Polyglyconate	Maxon	Absorbable monofilament	30% at 14 days 45% at 21 days	180 days	++	+
Poliglecaprone	Monocryl	Absorbable monofilament	50% at 7 days 80% at 14 days	90 days	++	+
Glycomer 631	V-Loc 90, Biosyn	Absorbable multifilament	50% at 14 days	90 days	+	+
Polyester	Ethibond, Dacron	Non-absorbable multifilament	N/A	N/A	-	+++
Polyamide	Nylon, Ethilon, Monosof	Non-absorbable multifilament	30% at 2 years	N/A	+	-
Polypropylene	Prolene, surgilene	Non-absorbable monofilament	N/A	N/A	++	-

Adapted from Fossum et al

The needle of the suture material represents another decision that needs to be made. The only needles that should be selected in this day and age are swaged on needles. All suture materials are manufactured in this form. This decreases tissue drag and trauma caused by the needle and suture material complex moving through the tissue.

There is quite a range of needle shapes and designs within this category and once again one needs to chose a selection for the work done in ones practice that best suits you. Reverse cutting needles have their cutting surfaces on the convex surface. These are the most versatile needled and are ideal for tough tissue like skin, tendons or oral mucosa. Round-bodied needles pierce and spread the tissues with minimal cutting. They are used in easily penetrated tissues like the peritoneum and abdominal viscera. This type of needle is also used in intestinal anastomosis to prevent leakage. Blunt, round body needles are used to dissect through friable tissues like liver and kidney.Spatulated needles can have up to four sides and are used for ophthalmic surgery. The reverse cutting is the needle readily available and has the most uses. It may be a good idea to have a few round-bodied PDS packs for intestinal anastomosis.

A word of mention to certain absorbable sutures in certain environments, this can dramatically change the absorption rate of these materials these will be mentioned below.

Skin closure is generally performed with a monofilament non-absorbable material. These have little tissue reaction and don't wick bacteria into deeper tissues. Multifilament material should not be use

din the skin as discussed above. Absorbable monofilament suture material can be used but will have to be removed, as it requires contact with body fluids for hydrolysis. Subcutaneous tissue should be closed with polydiaxonone or polyglyconate.

Linea alba closure requires an extended period of support from the suture material. This is true for all muscle and tendon closure. Although non-absorbable monofilament materials such as nylon are the normal certain absorbable materials can be used. Most surgeons are moving away from nylon and placing polydiaxonone in the lineaalba to decrease the amount of foreign material present in the long term. The knot of nylon has been shown to act as a nidus for bacterial infection in a small percentage of dogs.

The urinary bladder presents a hostile environment that in cases of certain infections can rapidly hydrolyse suture material in a few hours. It has been shown that Polydioxanone, polyglyconate, and glycomer 631 are all acceptable in the case of sterile urine when compared to the bladder healing time. However in the case of infection especially with a proteus species no suture material will stay long enough for healing of the bladder. Generally it is recommended not to allow the suture material to be exposed to the urine. Using a submucosal closure pattern performs this. Poliglecaprone 25 should not be used in the bladder as it will not hold tensile strength for long enough for the bladder to heal. Catgut is unacceptable for repair of bladders.

Parenchymal organs such as the liver and spleen require a low reactive suture material like polydiaxonone or polyglyconate. These can be used as crushing sutures if need be.

The gastrointestinal tract requires an absorbable monofilament with minimal tissue reaction or drag. Polydiaxonone is the suture of choice for these organs.

The oral cavity heals rapidly and thus requires a suture material that should be present for a short period of time to aid in healing but not to long to prevent irritation of the mouth. Polyglycolic acid is the material of choice in human dentistry. Polygalactin 910 is rapidly absorbed and is soft so will prevent discomfort. This is a good option for oral mucosa. Polydioxanone caused little tissue reaction but will remain for a long time so is less desirable. Catgut is absorbed rapidly in the oral cavity but leads to a marked tissue reaction, it can be used if there is no alternative.

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BITE WOUNDS WRAPPING IT UP! BANDAGES, DRESSINGS & DRAINS

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ABSTRACT
Bite wounds in dogs and cats are seen very commonly in the veterinary practice and the veterinary nurse plays a vital role in the continuous treatment and care of these patients.
This presentation will focus on the following topics:

- Types of wounds
- Stages of wound healing
- Initial assessment of the patient on presentation
- Nursing a patient with severe bite wounds
- Wound dressings
- Bandages
- Management of active and passive drains

The aim of my presentation is to provide guidelines with regards to the management of bites wounds to assist the veterinary nurse when having to initiate an individual treatment plan for a patient depending on the severity and extent of the wounds.

HAPPY SNAPS: RADIOLOGY IN YOUNG ANIMALS

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Abstract:
Juvenile and sub-adult patients for an intergral part of ones practice with (hopefully) a steady stream of new puppies and kittens coming through the doors. Whilst one hopes that many of these visits are “routine” and for preventative or prophylactic health care, anyone who has ever owned a puppy or kitted (or anyone who has a baby or toddler) will know that things are never that simple! Accidents happen and curious minds prevail as learning about life ensues. This together with the unwanted occurrence of infectious conditions which abound mean that one will need to deal with these critters in a much more intensive manner at some stage.

Radiology still in this modern day, forms an crucial part of the “in-house” armery of diagnostic tests available to the veterinary practice. Although the science behind radiography has advanced dramatically in the last 2 decades, allowing for increased throughput and ultimately a better image. The principles of radiography and radiology remain largely unchanged with one of the crucial aspects been that an image of diagnostic quality must be obtained in order to allow for a proper diagnosis.

Even though puppies and kittens are just small dogs and cats, due to physiological and anatomical differences that are present between juvenile and adult patients, certain factors should be borne in mind when radiographing a juvenile patient and interpreting the images.

The aim of any diagnostic procedure or test, including radiographic examinations, is to maximise the benefit and minimise the risks. As such a procedure should be tailored to each and every patient. And this is where a nurse can form a vital link in problem-orientated patient care. After discussion with a clinician, and undestanding the desired outcome for a radiographic procedure, together with his/her clinical experience, they can often single handedly result in an optimal imaging procedure.

The various organ systems will be assessed in this presentation looking at juvenile conditions and idiosyncracies whilst giving some useful tips and tricks in order to maximise benfit and minimise risk to the juvenile patient in the radiology suite.

FEEDING FOR RECOVERY & LONG TERM HEALTH

Guy Fyvie BVSc
Hill's Pet Nutrition

Nutrition is a critical part of helping our patients recover as quickly and completely as possible. Not only is it important to help overcome the condition, but lack of nutrition in hospital is one of the main causes of infection, slow recovery and mortality.
We will cover not only the nutrients of importance, but also the methods and procedures needed to ensure success.

HEADING HOME, THE BEST WAY TO RECOVERY

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Abstract
Client non-compliance is one of the most frustrating aspects of any veterinary practice.The veterinary nurse plays a vital role in the link between the veterinarian and the owner/client. Bygetting the veterinary nurse to talking to the owners,educating them about their pets disease process and understanding their personal circumstances and adapting treatments/instructions to better suit them, compliance will be improved immensely.

Introduction
Veterinarian's work very had at making the correct diagnosis for each patient and then deciding on the most effective treatment. Veterinary nurses spend a lot of time caring for these patients while in hospital and it is one of the highlights of the job when these patient's recover and are eventually able to be discharged. But the therapeutic outcome may not be optimal if the owner does not follow the discharge instructions as prescribed[1]. This could mean, "heading home is NOT the best way to recover!" Client non-compliance is one of the most frustrating aspects of any veterinary practice. Non-compliance is defined as the extent to which a course of recommended treatment is not carried out. In a human study it was found that approximately 125,000 people with treatable diseases die each year because they did not take their medication properly[2]! In veterinary medicine, poor compliance can result in an inadequate response to treatment, resulting in the patient being brought back to the clinic for more unnecessary tests and medication. The non response may also cause the owner to start to doubts the veterinarian's knowledge and skills which could lead to mistrust[1]. If antibacterial drugs are given incorrectly, there is the risk of recurrent infections and the possibility of developing bacterial resistance, which could have public health implications. Certain drugs that are stopped abruptly may lead to withdrawal signs like seizures, collapse etc[1]. This can be very dangerous or even fatal for the patient.

How can client compliance be improved so "heading home IS the best way to recover?
The veterinary nurse plays a vital role in the link between the veterinarian and the owner/client. The nurse is usually responsible for communicating with the owner and helping them understand the condition/diagnosis, the treatment and how/why/when it needs to be given, as well as the home management of the patient[3]. They are

When discussing diagnosis, treatment, prognosis and other discharge instructions etc. with clients, choose a quiet environment to avoid distractions[4]. Do not bring the patient into the room while you are talking to the client as their attention will become diverted and they will not listen to your instructions. Introduce yourself by name and ensure your full attention is on them[4]. This time that is spent with the client is one of the most important factors that affect compliance. If they feel like you have time for them and their pet they become more connected to you and are more likely to follow your directions more rigorously[5].

During the discussion, explain the diagnosis and prognosis. It is important to educate the clients in such a way that they have a very clear sense of the problem and that they understand why you believe the treatment the vet is recommending is clearly the best way forward to resolve their pet's condition[6]. Explain why the medication is being dispensed, the expected outcomes expected, and the potential consequences of poor compliance. Ensure that the client understands the route of administration of the medication, how much to give, and how often to give it. Demonstrate how to

administer the medication and provide any tips on medicating. If appropriate, have the client demonstrate how to give the medication with a placebo.Repetition is key! The average person needs to hear something at least five times to remember it. Repeat the most important instructions related to administration of the medication. Where possible supply the client with printed or pictorial instructions. Keep in mind that the person that you are explaining all the information to may not be the only, or the primary, caregiver for the pet, so if you don't send materials home, the person who actually cares for the pet may not receive the information they need to do so properly[7].

The client should be given tools and knowledge to allow them to trouble shoot problems that may arise[3]. This can be achieved by understanding the client's capabilities and home environment, then predicting problems they may face, and devising solutions for them as far as possible. For example, if an owner works a full day it will be impossible for them to give their pet medication three times a day, so the nurse can discuss this with the vet and an alternative medication that can be given once or twice a day dispensed. If the client feels that their needs as well as their pets needs are being taken into account they are more likely to be compliant.

It well known that good nutrition has a positive impact on both health and disease in all animals[8]. Specific diets are often incorporated into treatment plans to help treat or decrease the risk of that disease processes[9]. For this reason nutrition should always be discussed with the client at discharge. Advise the client on the specific diet that you or the vet would want/recommend the patient to go home with. Explain the potential advantages of using the specific diet and include recommendations on the amount and frequency that the food should be fed[8]. Discuss with the client any issues that may limit adherence to dietary recommendations and realize that recommendations may need to be modified to suit the client's time, lifestyle, and financial limitations[10]. Care should be taken to avoid overloading a client with too many options or choices, as this will just confuse them. Most importantly avoid using guilt or pressure to gain client acceptance[6].

Finally, always find the time to call the client the day following a discharge to determine if they are coping with the treatment and answer any more questions they may have. Contacting the owner for progress reports will improve compliance, patient outcome, and client satisfaction.

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PERILS FOR PUPPIES & KITTENS - TALKING TOXICITIES

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Puppies and kittens undergo significant developmental changes during the first few months of life that affect toxin absorption, distribution, metabolism and excretion. Together with their inquisitive nature, this makes them a unique population at increased risk for intoxication with both unusual toxins as well as arb clinical presentations of these toxins. Quick, efficient and successful decontamination remains one of the basic principles of the treatment of toxicity in this patient population as in adults. Rodenticides, nonsteroidal anti-inflammatory drugs (NSAID), antidepressants, herbicides, mushrooms, silica gel, cleaning products, chocolate, amphetamines, birth control products, flea products, insoluble calcium oxalate plants and liquid potpourri are the most common toxicants for animals under a year of age. Unusual presentations, clinical signs and different recovery times should be expected to adult animals.

During the first three months of life puppies and kittens undergo significant developmental changes. These changes may affect all aspects of drug, and thus toxicant, absorption, distribution, metabolism and excretion. The young patient has many structural and physiological differences relative to adult animals that can increase the level of toxicant absorption or severity of toxicosis. Young animals have increased intestinal permeability, increased gastric pH, decreased plasma protein levels, decreased body fat, increased total body water, increased uptake of volatile gasses, increased transdermal absorption and an immature P-glycoprotein system. The increased permeability of the intestinal mucosa, a poorly developed blood brain barrier as well as renal and hepatic immaturity causes puppies and kittens to absorb possible toxicants that would normally pass through without causing harm. Therefore, toxicants may cross into the neurological system of which it may be protected from in adulthood or show delayed metabolism and excretion. The curious nature of puppies and kittens may cause increased exposure to possibly noxious substances. Consequently unique toxicities and even arb clinical signs for common toxicants may be found in these patients[9].

As with any other diagnostic process the toxicologic history is of utmost importance. Tests to confirm common toxicoses seen in everyday practice are often poorly accessible, with long turn-around times and may be costly. For these reasons, possible toxicosis is often excluded solely based on the history. Nursing staff may often play an integral role in obtaining a history for an emergent case. The history should focus on *what* toxicant the animal may have been exposed to, *when* the exposure occurred, *how much* the animal could've been exposed to as well as the possible *routes of exposure*. In puppies and kittens that are still suckling the milk of the dam should be considered as a possible source of intoxication. Collecting history about littermates or other animals in the environment may also provide invaluable information to help guide the diagnosis of toxicosis as well as the type of toxicosis.

Quick, efficient and successful decontamination is often the focus of most patients presenting with a toxicity. The aggressiveness of the decontamination procedures chosen is usually guided by the type and amount of toxicant the patient was exposed to. In some toxicoses the use of decontamination procedures such as the induction of vomiting may be contraindicated. The successful recovery of the toxicants are procedure and time dependent and the use of most gastrointestinal decontamination procedures may be of little value if attempted an hour post-exposure. In many cases the timeline of exposure may be unknown and decontamination should be guided by the severity and type of clinical signs the patient presents with.

Owners often seek telephonic advice regarding possible intoxications. Unfortunately, there are very few options and little information on the safety and efficacy for the induction of vomiting at home. Inducing vomiting at home should only be recommended if the animal can't be presented to a veterinarian within one hour of exposure and if it is indicated for the toxicant. A 1 – 2 ml/kg dose of 3% hydrogen peroxide (H_2O_2)can be used to induce vomiting at home but causes mild gastroduodenal ulceration[7]. Clinical outcome studies have not shown any benefit for the use of gastric lavage, single-dose activated charcoal or the administration of a cathartic in the management of poisoned patients. Multi-dose activated charcoal may be of benefit in patients exposed to certain toxins such as phenobarbitone, theophylline, amitriptyline, digoxin, phenylbutazone or piroxicam. In the young patient, multi-dose activated charcoal is more likely to be of benefit for a larger variety of toxicants due to delayed gastric emptying, irregular intestinal peristalsis, good enterohepatic recirculation, and poor P-glycoprotein function leaving the toxin more susceptible to binding with activated charcoal. The use of activated charcoal is contraindicated in patients with decreased airway protection or intestinal

obstruction. Whole bowel irrigation should be considered in patients exposed to sustained-release or enteric-coated drugs presenting two hours after ingestion. For some toxins, manual removal via surgery or haemodialysis may be beneficial. Many toxins have specific antidotes available but few are routinely available in most practices and may have to be sourced from a human hospital or pharmacy[9].

The Animal Poison Control Centre of the United States of America report that rodenticides, nonsteroidal anti-inflammatory drugs (NSAID), antidepressants, herbicides, mushrooms, silica gel, cleaning products, chocolate, amphetamines, birth control products, flea products, insoluble calcium oxalate plants and liquid potpourri are the most common toxicants for animals under a year of age[9]. These toxicants don't necessarily match that seen commonly in South Africa.

Most rat poisons seen in practice today cause bleeding in intoxicated patients. These poisons cause a coagulopathy by inhibiting the recycling of Vitamin K_1which interferes with the clotting cascade. Dogs are more commonly intoxicated than other species and may spontaneously bleed from any site. The site of bleeding, volume and rate of bleeding determines the clinical signs seen and can be extremely varied. Clinicopathologic changes are supportive of an acute bleed in most cases with prolonged clotting times. Initially the prothrombin time (PT) prolongs followed by a prolongation in partial thromboplastin time (PTT). Not all cases exposed to rodenticides may be develop toxicity and running a PT/PTT may help to differentiate exposure from toxicity in these cases. If a patient presents shortly after exposure it is recommended that the PT/PTT be checked 36 hours after ingestion. The costs of this approach may often be cheaper than to prophylactically treat a case with Vitamin K_1 for 3 – 4 weeks. When a patient presents in a coagulopathic state treatment is mainly supportive. The fluid choice is dependent on the clinical status of the animal but plasma or whole blood (if the patient is anaemic) may be used to replenish clotting factors. Vitamin K_1 should be administered as soon as possible and a reduction of PT/PTT clotting times supports a diagnosis of rodenticide poisoning. Vitamin K_1 (not vitamin K_3) should be administered at 1.25 – 2.5 mg/kg every twelve hours per os or sub-cutaneously. The oral route should always be chosen except in animals who received activated charcoal, that are vomiting, or have malabsorption issues. Administering the Vitamin K_1 along with canned food increases the bioavailability 4 – 5 times. Vitamin K_1 treatment should be continued for as long as is recommended for the specific toxin ingested. When the exact toxicant is unknown a protocol of treating for 3 – 4 weeks with follow-up PT 36 to 48 hours after cessation of therapy can be followed with good success[10].

NSAID toxicity is a common cause of toxicity in puppies and kittens due to the uninformed administration of these drugs by well-meaning owners. Young patients have extensive enterohepatic recirculation of these drugs which increases their toxicity. Cats are especially prone to toxicity with NSAID's due to a deficiency in enzymes needed for the breakdown of these medications. This allows for a longer half-life, increasing the toxicity of the drug in this species. NSAID's cause toxicity by reversible inhibition of the cyclooxygenase enzyme system which is important for the regulation of many normal processes such as gastric and renal blood flow. Clinical signs are mainly limited to the gastrointestinal system and include anorexia, vomiting, diarrhoea, malaena, polyuria, polydipsia and tachypnoea. In some patients the kidneys, liver, central nervous system or coagulation may also be affected. There is no antidote for NSAID toxicity and treatment is mainly supportive. If patients present within 2 hours of intoxication the induction of vomiting and the administration of activated charcoal and a cathartic (such as lactulose) may be beneficial to limit further absorption and disrupt enterohepatic recirculation of the drug. In patients with large intoxications, those at risk for renal damage, intravenous fluid therapy at twice maintenance rates for at least 48 hours are indicated. In patients with clinical signs of gastroduodenal ulceration the use of anti-ulcerative medications and prostaglandin analogues are indicated[10].

Tricyclic antidepressants, serotonin reuptake inhibitors or monoamine oxidase inhibitors are most commonly implicated in antidepressant toxicities. Puppies and kittens may accidentally consume these medications from the household environment when not stored out of reach. Most of these compounds cause central nervous system or cardiovascular abnormalities as soon as one hour from ingestion. The clinical signs can be acute in onset and the induction of vomiting should be done with caution. Activated charcoal and a cathartic should be administered to limit further absorption. Repeated activated charcoal doses should be administered to interrupt the enterohepatic recirculation seen with these drugs. Patients should be intensely monitored for deterioration and signs of acidosis. The treatment of neurological signs is mainly supportive. In cases of serotonin syndrome treatment with cyproheptadine at 1.1mg/kg per os may be indicated[10].

Acetylcholinesterase (AChE) inhibitor toxicity with compounds such as carbamates or organophosphates are commonly seen in South Africa as a malicious intoxication[1]. AChE inhibitors are also commonly found in household pesticides. Clinical signs include muscarinic, nicotinic and

central nervous system signs and can be remembered using the mnemonic DUMBELS for diarrhoea/depression, urination, miosis/muscle tremors, bronchospasm/bradycardia, emesis, lacrimation and salivation/seizures. Atropine at 0.1 – 0.5 mg/kg given at one quarter intravenously and the remainder given subcutaneously is the treatment of choice. Follow-up doses of atropine should be lower at 0.1mg/kg and guided by intensive monitoring of the heart rate and wet lung sounds. When organophosphate poisoning is suspected, or if the toxicant is unknown and the patient doesn't respond adequately to atropine, 2-pyridine aldoxime methyl chloride (2-PAM) can also be considered. However, this drug is not readily available in South Africa and is not indicated in the treatment of more commonly seen carbamate toxicities. There is little evidence for the use of other agents such as diphenhydramine in the management of this toxicity[10]. Intravenous fluid therapy, oxygen supplementation and diazepam for cases presenting with seizures may be utilised as needed.

Chocolate contains methylxanthine compounds such as theobromine and caffeine. The concentration of active compounds differ between types of chocolate with unsweetened baking chocolate, dark chocolate, milk chocolate and white chocolate containing decreasing amounts of methylxanthines. There are many free online, easily accessible chocolate toxicity calculators available to aid in the management of these toxicities. These calculators usually take the weight of the animal, the type of chocolate and estimated amount of chocolate consumed into account. Clinical signs include restlessness, weakness, hyperactivity, ataxia, tachycardia, diuresis, diarrhoea, muscle tremors and clonic seizures but depends on the amount and type of chocolate ingested. The gastric emptying of chocolate is often delayed and the induction of vomiting may still be effective for up to 6 hours after ingestion. Repeated activated charcoal doses can be considered in cases where large ingestions occurred. Intravenous fluid therapy is indicated to maintain renal perfusion and promote excretion of metabolites. Supportive treatment of tremors, central nervous and cardiovascular signs are indicated[10].

Cycads are common ornamental or garden plants in South Africa that may be fatal when consumed in even small amounts. All parts of the plant are poisonous and dogs often present with clinical signs suggestive of gastrointestinal or acute liver damage. Aggressive decontamination procedures are indicated due to the high mortality rate seen in these cases. Treatment is mainly supportive as indicated for severe acute liver injury including intravenous fluid therapy, hepatoprotectants and the supplementation of clotting factors through plasma transfusions in patients showing signs of coagulopathy. The prognosis for animals showing signs of toxicity is poor and animals a history of cycad ingestion may develop chronic liver failure at a later stage[5; 6].

Grapes, raisins, sultanas and currants may be toxic to some dogs. Toxic effects do not appear to be dose dependent and the mechanism of toxicity of unknown. Clinical signs mainly relate to acute kidney injury in cases where toxicity develops. Vomiting, diarrhoea, anorexia, lethargy and abdominal pain have been reported. Clinicopathologic findings usually mirror that of acute kidney injury. Treatment includes routine decontamination for up to 2 hours after ingestion as gastric emptying of fruits are usually slightly delayed. Intravenous fluid therapy and the monitoring of renal values are indicated for at least 72 hours after ingestion. Other treatments are mainly supportive as indicated for acute kidney injury[4; 10].

Xylitol is an artificial sweetener commonly found in many medications, dental care products and food items. Clinical signs relate to that of hypoglycaemia, hepatopathy or both. Hypoglycaemia is usually seen within one hour of ingestion but may be delayed for up to 48 hours. Lethargy, ataxia, collapse and seizures are commonly seen. Seizures may occur in the absence of hypoglycaemia. The induction of vomiting is advised but the use of activated charcoal is rarely recommended due to its poor ability to bind xylitol. Cases that ingest more than 0.1 - 0.5 g/kg of xylitol should be hospitalised for observation if no clinical signs are noted on presentation. Treatment is mainly supportive using a dextrose bolus followed by a constant rate infusion for 24 hours in cases with hypoglycaemia[3; 10].

Store-bought tick and flea products often contain pyrethrins and pyrethroids as active ingredients. Cats are more sensitive to the effects of this toxicant and often present after accidental use of a product labelled for dogs. Clinical signs include hypersalivation, irritability, vomiting, diarrhoea, hyperthermia, tremors, hyperaesthesia or seizures[2]. Decontamination is dependent on the route of exposure but cats with dermal exposure should be thoroughly bathed in a detergent liquid such as dishwashing liquid. Activated charcoal may be of little value in these cases but can be considered in cases with oral exposure as an once off oral dose shortly after ingestion. Neurological signs are treated symptomatically and methocarbamol anecdotally provides better control than diazepam in these cases. In cases with severe toxicosis intravenous lipid emulsion therapy may be considered but there are few reports available on the safety and efficacy of this treatment[8].

Paracetamol is widely available as an over-the-counter medication and exposure to dogs and cats are generally seen after uninformed administration by the owner. Cats are especially sensitive to the toxic

effects of paracetamol due to a diminished ability to metabolise the drug and an increased sensitivity of their red blood cells to the toxic effects of the drug. Cats often present with haemolytic anaemia or methheamoglobinaemia whereas liver damage are frequently seen in dogs. Treatment is aimed at preventing further absorption using routine decontamination procedures. Activated charcoal may be beneficial if administered within 4 – 6 hours of ingestion. Supportive therapy may include oxygen supplementation, intravenous fluid therapy, restoration of liver antioxidant (glutathione) stores using *N*-acetylcysteine and S-Adenosyl methionine (SAMe). Little information is available on the advantage of the use of methylene blue, ascorbic acid or cimetidine in the management of these cases[10].

Lilies are common household or garden plants. All parts of the plant are toxic and the ingestion of smalls amounts such as 2 - 3 leaves or eating part of a flower may be lethal to cats. The mechanism of toxicity is unknown but clinical signs are usually suggestive of acute kidney injury. Lethargy, vomiting, anorexia, polyuria followed by anuria, weakness and death have been reported. Treatment is aimed at decontamination, aggressive fluid diuresis using 2 to 3 times daily maintenance rates for 48 – 72 hours at least with close monitoring of urine output. Renal parameters should be monitored every 12 – 24 hours and dialysis may be considered in anuric cases if available[10; 11].

In conclusion, puppies and kittens present a unique population for clinical toxicology but the approach to treatment of the toxicity patient remains largely the same. The toxicological history is utmost importance when faced with toxicity in a young patient. Unusual presentations, clinical signs and different recovery times should be expected to adult animals.

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FLUID THERAPY: THE GOOD, THE BAD, THE UGLY.

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Abstract:
Fluid management with colloid and crystalloid solutions remains controversial peri-operatively. The selection and use of resuscitation fluids is based on physiological principles, but clinical practice is determined largely by clinician preference.

Fluid administration during anaesthesia is necessary to control vascular tone, maintain circulating volume, and improve cardiac output (CO) to provide oxygen to all tissues. Over-hydration and excessive fluid administration can be detrimental to patients just as dehydration and hypovolemia can lead to adverse consequences.

Potential adverse effects of overly aggressive fluid therapy include volume overload, pulmonary oedema, detrimental fluid shifts (eg, oedema of the brain, kidneys, and intestinal tract), electrolyte and acid base derangements, exacerbation of haemorrhage, and hemodilution coagulopathy.

Primary Treatment Goal

'Improve delivery of oxygen and other nutrients to metabolically active cells'

Clinical Fluid therapy:

Water is the most essential nutrient in the body. It functions as the transport medium of oxygen, solutes and hormones to the interstitium and delivers waste products to the liver, kidneys and lungs for excretion.

Monitoring:
Monitoring of fluid status is vital in the peri operative period and perfusion parameters and vital signs should be routinely and repeatedly evaluated

Five steps to intravascular resuscitation plan

Step 1 Determine whether there is an intravascular deficit affecting perfusion parameters by assessing the physical perfusion parameters
Step 2 Select appropriate fluids for the patient
Step 3 Determine resuscitation endpoints

- High normal – SIRS, metabolic disease
- Low normal – heart, lung, brain disease, coagulopathy, haemorrhage, oliguric renal failure

Step4 Determine resuscitation volume and route
Step 5 Ongoing monitoring and re-evaluation

One of the most common uses of fluid therapy is for patient support during the peri-anaesthetic period.
The paradigm of "crystalloid fluids at 10 mL/kg/hr, with higher volumes for "anaesthesia-induced hypotension" is not evidence-based. The primary risk of providing excessive IV fluids in healthy patients is the potential for vascular overload.

Current recommendations are 3 mL/kg/hr in cats and 5 mL/kg/hr in dogs.

Pre-operative volume loading of normovolemic patients is not recommended.
Those high fluid rates may actually lead to worsened outcomes, including increased body weight and lung water; decreased pulmonary function; coagulation deficits; reduced gut motility; reduced tissue oxygenation; increased infection rate; increased body weight; and positive fluid balance, with decreases in packed cell volume, total protein concentration, and body temperature.

Monitoring and Responding to Hypotension

During anaesthesia, blood pressure (BP) is the parameter often used to estimate tissue perfusion, although its usefulness as an indicator of blood flow is uncertain.Hypotension under anaesthesia is a

frequent occurrence, even in healthy anaesthetized veterinary patients. Assess excessive anaesthetic depth first because it is a common cause of hypotension.
If relative hypovolemia due to peripheral vasodilation is contributing to hypotension in the anaesthetized patient

- Decrease anaesthetic depth and/or inhalant concentration. ·
- Provide an IV bolus of an isotonic crystalloid such as LRS (3–10 mL/kg).
- Repeat once if needed. ·
- If response is inadequate, consider IV administration of a colloid such as hetastarch. Slowly administer 5–10 mL/kg for dogs and 1–5 mL/kg for cats, titrating to effect to minimize the risk of vascular overload (measure BP every 3–5 min).
- Colloids are more likely to increase BP than crystalloids
- If response to crystalloid and/or colloid boluses is inadequate and patient is not hypovolemic, techniques other than fluid therapy may be needed (e.g., vasopressors or, balanced anaesthetic techniques).

Post anaesthetic Fluid Therapy
Based on intra-anaesthetic complications and comorbid conditions. Patients that may benefit from fluid therapy after anaesthesia include geriatric patients and patients with either renal disease or ongoing fluid losses from gastrointestinal disease.
Monitor Response to Fluid Therapy
Individual patients' fluid therapy needs change often. Monitor for a resolution of the signs that indicated the patient was in need of fluids. Monitor for under-administration (e.g., persistent increased heart rate, poor pulse quality, hypotension, urine output), and overadministration (e.g., increased respiratory rate and effort, peripheral and/or pulmonary oedema, weight gain, pulmonary crackles [a late indicator]). Patients with a high risk of fluid overload include those with heart disease, renal disease, and patients receiving fluids via gravity flow.
Cats require very close monitoring. Their smaller blood volume, lower metabolic rate, and higher incidence of occult cardiac disease make them less tolerant of high fluid rates.
Treating hypovolemia
When intravascular volume expansion without whole blood is needed, use crystalloids, colloids, or both. IV isotonic crystalloid fluids are the initial fluid of choice.
How to administer crystalloids ·
Standard crystalloid shock doses are essentially one complete blood volume.
Shock rates are 80–90 mL/kg IV in dogs and 50–55 mL/kg IV in cats.
Begin by rapidly administering 25% of the calculated shock dose. Reassess the patient for the need to continue at each 25% dose increment.
Monitor signs as described in the patient assessment portion of this document. In general, if 50% of the calculated shock volume of isotonic crystalloid has not caused sufficient improvement, consider either switching to or adding a colloid.
Once shock is stabilized, replace initial calculated volume deficits over 6–8 hr depending on comorbidities such as renal function and cardiac disease.
Treating Hypovolaemic shock

Crystalloids

- Preferably an isotonic replacement solutions
- The standard dose is 25% of blood volume (BV) within 10 minutes. This equates to 20-22ml/kg in the dog given as fast as possible. For larger dogs, fluid pumps often do not go fast enough, therefore a pressure infusion bag should be used. The goal is to reassess after the first bolus and if the animal is still in shock, to give another 25% BV dose, as a bolus. If the animal has more severe shock, a larger initial dose should be given, up to a full blood volume if the patient appears to be dying.
- In cats, it is safer to give fluid boluses in the form of 60ml syringe doses. If you use a pump or open fluid bag, there is a risk of accidental fluid overload which may kill the cat. A dose of 60ml is about 10% BV. You can repeat the 60 ml boluses (by hand) up to a full BV (50-60ml/kg). Cats rarely need a full BV and are prone to fluid overload (i.e. lung oedema); therefore shock fluid therapy is done more cautiously in this species.
- If you are suspicious of severe electrolyte abnormalities in the patient due to the presenting problem, it is good practice to run electrolytes before bolusing large amounts of crystalloid. This may help to guide the fluid choice. It is also useful to run a PCV/TP before bolusing

fluids, as a baseline for future problems such as bleeding and hypoproteinaemia.

When to administer colloids ·
When it is difficult to administer sufficient volumes of fluids rapidly enough to resuscitate a patient and/or when achieving the greatest cardiovascular benefit with the least volume of infused fluids is desirable (e.g., large patient, emergency surgery, large fluid loss). ·
In patients with large volume losses where crystalloids are not effectively improving or maintaining blood volume restoration. · When increased tissue perfusion and O2 delivery is needed.
When decreased oncotic pressure is suspected or when the total protein is <35 g/L (or albumin is , < 15 g/L).
The typical hydroxyethyl starch dose for the dog is up to 20 mL/kg/24 hr (divide into 5 mL/kg boluses and reassess).
For the cat, the dose range is 10–20 mL/kg/24 hr (typically, 10 mL/kg in 2.5–3 mL/kg boluses).
Titrate the amount of colloid infused to effect. Simultaneously administering crystalloids and colloids · Use this technique when it is necessary to both increase intravascular volume (via colloids) and replenish interstitial deficits (via crystalloids). ·
Administer colloids at 5–10 mL/kg in the dog and 1–5 mL/kg in the cat. Administer the crystalloids at 40–45 mL/kg in the dog and 25–27 mL/kg in the cat, which is equivalent to approximately half the shock dose.
Titrate to effect and continually reassess clinical parameters to adjust rate and type of fluid administered (crystalloid and/or colloid).
Treating hypovolemia due to blood loss
The decision of when to use blood products instead of balanced electrolyte solutions is based on the severity of estimated blood loss. If blood products are not deemed necessary, note that patients with low vascular volume (due to either vasodilation or haemorrhage) will benefit more from the use of colloids than crystalloids.
Following 15 mL/kg of haemorrhage, even 75 mL/kg of crystalloid will not return blood volume to prehaemorrhage levels because crystalloids are highly redistributed. Large volumes may be needed to achieve blood volume restoration goals, and large volumes may be detrimental to patients with normal whole body fluid volume but decreased vascular volume resulting from acute blood loss.

Treatment endpoints

Aim Improving

- Mental state
- Mucous membrane colour
- Capillary refill time
- Pulse rate pulse quality
- Extremity TºC
- Heart rate (decreased)
- Blood pressure
- Urine production (>1-2 ml/kg/hr)

Hypervolemia
Hypervolemia can be due to heart failure, renal failure, and/or iatrogenic fluid overload. Hypertension is not an indicator of hypervolemia. Treatment is directed at correcting underlying disease (e.g., chronic renal disease, heart disease), decreasing or stopping fluid administration, and (possibly) use of diuretics. Consider using hypotonic 0.45% sodium chloride as maintenance fluid therapy in patients susceptible to volume overload (such as those with heart disease) due to the decreased Na^+ load.

The traditional “recipe-based” approach to peri-operative fluid therapy is outdated. Fluid therapy should be prescribed like any other drug, monitored and adapted to observed changes.

Suggested Reading:

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DiBartola SP, ed. Fluid, Electrolyte, and Acid-Base Disorders. 3rd ed. Saunders Elsevier, St. Louis, MO. 2006: Wellman ML, DiBartola SP, Kohn CW.

Chapter 1: Applied Physiology of Body Fluids in Dogs and Cats, pg 3 – 2

Chapter 14: Introduction to Fluid Therapy, pg 325 – 344.

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FLUID THERAPY: THE GOOD, THE BAD, THE UGLY.

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Abstract:

There is an increasing demand on veterinary nurses to take more and more responsibility for drugs and drug protocols. The ability to do accurate and quick drug calculations is an essential skill for nurses.

This lecture aims to explain calculations in a step by step approach for routine drugs used during anaesthesia, but also include fluid rates and constant rate infusions. There are quite a few calculators available online, but it is still important to understand how these calculations are done and to develop a “gut feel” if a dose or dose rate seem to be wrong.

Additional reading:

https://www.vetnurse.co.uk/nursing/w/vet-nurse-revision_1/calculation-of-drug-doses-key-notes.aspx
Dosage Calculations for Veterinary Nurses & Techniciansby Terry LakePublished March 15th 2004 by Butterworth-Heinemann
http://www.vasg.org/drug_delivery_calculators.htm

WHY IS PAEDIATRIC CARE IN ICU IMPORTANT?

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Abstract
Paediatric patients have a unique physiological system. This unique physiology results in considerable differences in hemodynamic parameters, drug dosages, laboratory data, and diagnostic imaging compared with those of adults.It is thus imperative that caregivers of these young patients be familiar with these differences to be able to treat and monitor these patients appropriately.

Introduction
In veterinary medicine the term neonate is usually used for puppies and kittens in the first 2 weeks of life and the term paediatric generally refers to animals between 2 weeks and 6 months of age[1,2]I will use the term paediatric to refer to all young patients in this talk. It is very important to appreciate that paediatric patients are not small adults! They may suffer from some of the same diseases processes as adults, but ill paediatric patients can quickly become critically ill.Their monitoring and care is markedly different from that of the adult patient due to their unique physiologic systems that results in considerable differences in hemodynamic parameters, drug dosages, laboratory data, and diagnostic imaging.It is thus imperative that clinicians and nurses be familiar with these differences to be able to treat and monitor these patients appropriately[1]. Caring for healthy orphaned kittens and puppies is beyond the scope of this lecture. Excellent guides are available for the care of orphaned kittens and puppies[3,4].

How they differ
Clinical parameters
Paediatrics have higher heart and respiratory rates than adults. The heart rate slowly decreases as the parasympathetic tone starts to increase at about 4 weeks of age. The heart rate is approximately 180-200bpm. Heart rate in paediatric patients does not respond to volume change like mature animals do. Limited information is gained from thoracic auscultation due to the small size of the thorax and the small tidal volume along with increased interstitial fluid in the normal paediatric lungs. Auscultation is best performed using a paediatric stethoscope[5]. Heart murmurs can often be heard in young patients. Majority of theses are innocent murmurs and resolve within the first 3 months of life. However other causes of heart murmurs such as congenital cardiac defects, anaemia, fever etc. could be present and should be ruled out[6]. A normal respiratory rate for neonates is around 10–18bpm at birth, increasing to 15–35bpm by 1 week of age. The rectal temperature is approximately 36°C-37°C at birth and then increases to 38°C by I month of age[5]. These young patients are essentially poikilothermic and lack a shiver reflex for the first 2 weeks, then gradually become homoeothermic, but are still susceptible to environmental conditions and may become hypothermic easily due to their large surface-to-mass ratio, so always examine then on a warm surface[7].

Imaging
Radiographs can be challenging to interpret. The resolution of the radiograph can be improved by decreasing the kilovoltage peak value (kVp) to half of the adult setting and using detailed screens[8]. Most studies in paediatrics are radiographed using the range of 40–60kVp.[5] Abdominal radiographs are usually not very helpful as there is loss of abdominal detail because of lack of fat and a small amount of abdominal effusion making abdominal organ abnormalities difficult to detect. The liver frequently looks like it extends beyond the ribcage due to the absence of costochondral mineralization and is often incorrectly interpreted as hepatomegaly. The lung parenchyma in young animals has a higher water content, which makes them appear more opaque on radiographs. The thymus can also add confusion as it can look like a mediastinal mass or lung consolidation. Lastly, the heart appears falsely enlarged as it takes up more space in the thorax[1,6].

Diagnostic tests
Due to the small size of paediatrics, only small volumes of blood can be drawn. The recommended volume is no more than 1% of the body weight in a 24-hour period. It is thus vital that each blood sample be used efficiently. The paediatric patients blood results differs from those of adult in the following ways: The haematocrit (Ht) is usually high at birth, but declines dramatically by 3 days of age, and continues to decrease till the nadir at 3 weeks in puppies and 4-6 weeks in kittens. Thereafter the Ht will start to increase again and be in the normal adult range by 6 months of age[6,9].

White blood cell parameters in these patients are similar to those of adult[9]. There are some dramatic changes in biochemical values that should be noted. All paediatrics will have a slightly increased bilirubin, elevated calcium and phosphorus (as a result of bone growth), and dramatically increased liver enzymes. The alkaline phosphatase (ALP) and g-glutamyltransferase (GGT) is 20 times greater that of the adult reference range in puppies, and the ALP is 3 times range in kittens. The creatinine, blood urea nitrogen, albumin and cholesterol are lower than reference value[6,10]. Low urine specific gravity (SG) (1.006–1.0017) is normal until approximately 9 to 10 weeks of age. Glucosuria and proteinuria can be a normal finding in neonates up to 8 weeks of age because tubular maturation occurs later than glomerular maturation[5]. Urine concentration is expected to compare with that of the adult dog by 6 to 8 weeks of age[9].

Pharmacology

Drug absorption, distribution, metabolism, and elimination differ substantially in paediatrics compared to adults[11]. Drug clearance reaches adult capacity at about 12 weeks of age. The blood-brain barrier (BBB) is more permeable, therefore drugs that would not normally cross the BBB in healthy adult animals may enter their central nervous system (CNS)[1]. Drug absorption in theses patientscan be either higher or lower. The area of absorption in their small intestine is much larger relative to their size, which results in rapid absorption of drugs with a high bioavailability, leading to high peak plasma concentrations of these drugs.Thevolume of distribution of lipid soluble drugs is decreased due to the decreased body fat[12]. Gastric emptying is reduced in very young animals but then increases to higher rates than that found in adults, which will also affect drug absorption[12]. Hepatic microsomal and P450enzyme activity does not develop fully until 4 to 5 months of age, so caution must be exercised when prescribing medication that requires hepatic metabolism or excretion[9]. Renally excreted or metabolised drugs should also be used cautiously because of the paediatric patient's limited capacity to eliminate drugs that depend on renal excretory mechanisms. The potential for renal toxicity with nonsteroidal anti-inflammatory drugs is also far greater than in the adult animal, and their use in paediatric medicine is generally not advised[13]. Subcutaneous (SC) injections are preferred over the intramuscular (IM) route (because of reduced muscle mass in paediatric patients), and due to their increased total body water and decreased fat, absorption is better[12].

Treatment principles

Physical examination

Sick paediatric patients should be examined as soon as possible, using the same systematic approach that is used on any ill patient, which includes a complete history, thorough clinical examination including the weight of the patient, followed by further diagnostic testing as needed. In the more seriously ill paediatric patient, urgent medical attention is needed (as these patients can decompensate quickly).A brief history should be obtained along with a quick primary survey. The primary survey is designed to rapidly identify abnormalities in the major body systems (airway/breathing, circulation, CNS dysfunction) that can be associated with life-threatening conditions. Immediate life-saving therapy should then be initiated as necessary[14]. Focusing on the four H's[11]:

1.Hypovolemia/hydration 2. Hypoglycaemia 3. Hypothermia 4.Hypoxemia.

1. Hypovolaemia/hydration

Adult animals are able to partially compensate for hypovolaemia by increasing their heart rate, concentrating their urine and decreasing urine production. As discussed above, paediatric patients have very poor compensatory mechanisms. The parameters that are used to determine a patient's hydration and volume status e.g. mucous membrane colour, moisture, skin turgor, urine SG, heart rate, pulse quality and blood pressure are more difficult to gauge in very young animals[10]. Mean arterial blood pressure is lower, at 1 month of age it is around 49mmHg and only increases to 94mmHg by approximately 9 months of age. The central venous pressures are higher, reading 8cmH_2O at 1 month and decreasing to 2cmH_2O by 9 months[8]. A urine SG reaching 1.020 is an indication of dehydration in paediatric patients[10].

Paediatric patients have higher fluid requirements than adults as they have an increased extracellular fluid volume, higher water content (80% VS 60%), they use more water due to their higher metabolic rate, and lose more water due to their greater surface area: body weight ratio, lack of body fat, decreased ability to concentrate urine, more permeable skin and increased respiratory rate that

results in increased insensible fluid losses[2]. Because of theses higher fluid requirements, anything that results in increased fluid loses, e.g. inadequate intake, vomiting and diarrhoea, will result in dehydration, which can rapidly lead to hypovolaemia and shock if not corrected promptly. Warm, isotonic crystalloid replacement fluids can be used safely in these patients. Lactated Ringer's is recommended by some because lactate has been shown to be a preferred metabolic fuel in the neonatal brain during hypoglycaemia[6] but not in patients younger than 6 weeks of age as they cannot metabolise the lactate effectively to bicarbonate[11]. Hypovolaemic patients can receive a fluid bolus at 3 to 4 mL/100g (puppies) and 2 to 3 mL/100g (kittens)[2], thereafter the patient should be reassessed to determine the response to the bolus and evaluate the need for further fluid resuscitation. Once hypovolaemia has been addressed, maintenance fluid rates with the estimated on going losses added to it should be given as a CRI. Maintenance fluid requirements range between 80-120mL/kg/day for puppies, whereas a slightly lower rate of 60–80mL/kg/day can be used for kittens. Once the patient is about 4 to 6 months of age these maintenance fluid rates can be decrease to those of adult animals[2]. Although dehydration is common, overhydration is also a serious concern, because the kidneys cannot concentrate or dilute urine to rid the body of excess water[1]. Fluid therapy should therefore be administered with care and continually monitored and adjusted to ensure adequate volume maintenance without overhydration or oncotic loading[9]. An accurate paediatric gram scale is essential for monitoring fluid loads, and weighing the patient at least every 12h, but preferably every 6 – 8h is recommended[10]. Haematocrit and total protein values can also be monitored, bearing in mind the differences in values as discussed[6]. To prevent volume overload paediatric drip sets should be used along with infusion pumps or syringe drivers. If these are not available then a buratrol can also be used. The preferred route to administer fluids to hypovolaemic patients is via the intravenous (IV) route, but due to their small size this is often difficult. The small gauge catheters that are often needed in theses patients frequently develop burrs as they pass through the skin. This can be prevent by elevating the skin and making a small skin puncture using a 20G needle, and then passing the catheter through the skin puncture[8]. If attempts at IV access are unsuccessful, the intraosseous (IO) route can be used. An 18-22-gauge spinal or hypodermic needle can be used to gain IO access at the following sites: the greater tubercle of the humerus, trochanteric fossa of the femur, tibial tuberosity, or wing of the ilium[2,15]. The needle is placed parallel to the long axis of the bone and then secured. As with IV catheter placement aseptic placement technique are essential to prevent sepsis and osteomyelitis at the area of IO catheter placement.[11] As soon as IV access can be established the IO catheter must be removed (preferably with in 2h)[8].

2. Hypoglycaemia

Paediatrics have an increased demand for glucose, but at the same time they have an increased loss of glucose in the urine, and they have inefficient hepatic gluconeogenesis, decreased glycogen stores, and an immature glucose feedback mechanism making them extremely susceptible to hypoglycaemia. Vomiting, diarrhoea, infections, and decreased intake all contribute to hypoglycemia in young animals[1]. If hypoglycaemia is suspected, a glucose bolus can be administered before blood glucose concentration is measured.Subsequent doses should only be repeated based on measured blood glucose concentration. Dilute a 50% dextrose solution 1:3 in sterile water to make a 12.5% dextrose solution, and bolus 1-3mL of this solution IV or IO[2]. Always follow a glucose bolus with a CRI of isotonic fluids supplemented with dextrose to make a 2.5% to 5% solution to prevent rebound hypoglycemia[1] . Do not over supplementation with dextrose as this can result in osmotic diuresis and worsening of dehydration[11].

3. Hypothermia

Hypothermic patients should be warmed slowly over 1–3h, as rapid warming will lead to peripheral vasodilation and shock[8]. Body temperature has a dramatic effect on gastrointestinal movement in young animals. If the temperature drops below 34.4°C, ileus develops, predisposing the patient to anorexia, vomiting with possible aspiration[9].

4. Hypoxaemia

The amount of work and pressure that is required by a paediatric patient to maintain tidal breathing is increased as because of the high compliance of their chest wall. Any respiratory disorder that shortens inspiratory duration has a greater potential to have a negative impact on gas exchange in these patients[9]. Initial therapy in all patients with respiratory distress should consist of oxygen supplementation via flow by, facemask, oxygen cage or if severe distress, endotracheal intubation. The fraction of inspired oxygen should never exceed 40%–60%, as oxygen toxicity can occur and is even more of a concern in paediatric patients where excess oxygen supplementation can cause

retrolental fibroplasia, which can lead to permanent blindness[1]. If higher levels of oxygen are necessary to relieve signs of respiratory distress, the use of positive-pressure ventilation will be needed, but this will be technically challenging in these small patients[11].

Conclusion

Knowledge of normal physiologic parameters, will aid appropriate monitoring and treatment of pediatric patients. Despite their tiny size, critically ill puppies and kittens can be treated successfully, allowing them to go on to have a full life.

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OPENING A CAN OF WORMS – THE 3 TERRORS

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Value your Values

Values are those things that mean the most to us as individuals. They are emotions, personal beliefs and behaviours that we hold as most dear to us. In my story, the most important values were friendship, making a go of it in South Africa and fulfilling my future as a veterinarian. Those were the three most valuable things in my life at that stage.

Your mind-set, performance, likes, dislikes and even goals are based on your values. Values dictate your choice in clothing, the TV programmes and movies you enjoy, and if, when and whom you will marry, and even whether you will have children. Your values will determine your response to any given situation. Values drive our decisions and judgements. Our perceptions of what is right and wrong, good and bad derive from whether something is aligned with our values or not. You will find that you like people who support or share your values and dislike people who don't. Every person has his or her own unique appreciation of what values are most appropriate for them and being different to your own does not necessarily make them good or bad. It simply makes us unique.

The truth is that most people have spent little, if any, time identifying their personal values. A further complication I find with my clients is that the values they list at first are sometimes not their true values at all. When this happens, we need to spend time together, carefully examining the meaning of each one, using the most appropriate terminology, matching it to their current situation – behaviour, activity and status quo are often quite revealing; for example, adventure addicts are far less likely to have certainty as one of their core values – as well as differentiating their true values from values they wish they had. For instance, if you are chronically obese, you cannot have fitness as a priority value; rather, this is a value you want, or an aspirational value. Our past activity has resulted in our current situation guided by our value systems. Actions that are not aligned with our values cause conflict, either on a conscious or subconscious level, and manifest as discomfort; this disconnect is also referred to as "cognitive dissonance".

Having set that foundation will give you a fantastic start towards achieving your objectives in life. We can now continue with confidence, knowing that your inner self (values) is in alignment with your life's vision. You should already be able to sense the resultant satisfaction, which will create a shift in your consciousness. You no longer have to wait for things to get better, they are better. You can create the better circumstances for yourself and nobody else. Mastering this skill will give you the ultimate control you wish to have over your life.

Opening a can of worms: The 3 Terrors

Infectious diseases are a very real threat for our cats and dogs in South Africa and it is a topic of discussion every single day in private practice. The diseases we have to protect our pets against are also a threat to us as humans either directly or indirectly (zoonoses) through our very own animals. The vaccination protocols that we have relied on for so many decades have been re-evaluated and reconsidered a number of times in recent years. What is the rationale behind the various recommendations and protocols? Which one do I apply in my practice? Do I subscribe to the annual revaccination protocol for adults animals or the 3-year protocols? Should we be even considering the 3-year protocol in South Africa?

Even the list of core vaccines has been revisited and been challenged to maybe include antigens that were traditionally considered non-core vaccinations like leptospirosis. How common is leptospirosis in South Africa? Are there particular parts of the country where the risk of leptospirosis is higher?

Which serovars of leptospirosis are present in South Africa? What protection do our vaccines in South Africa provide against the serovars that exist within our borders?

Questions on Feline Leukaemia have become more frequent from a diagnostic perspective as well as a vaccination standpoint. Do we vaccinate kittens, at what age and do we need to test them before we do so? What are the implications of having this disease in a house of a breeding facility?

Kennel cough is a social disease and a misnoma implying that only dogs going to kennels should be considered as potential recipients for this vaccine. Kennel Cough is a syndrome, not a diagnosis. We revisit the potential causes of this very common disease and relate it back to our vaccination practices. A number of the core vaccines have been diagnosed more frequently lately including canine parvovirosis, canine distemper and Rabies. How would you explain it to a pet owner who asks questions after having vaccinated their pet and is now having their animal treated for one of these conditions?

Deworming programmes are often part of a practice's service offering and sometimes there may be a reason to reconsider and update. What are the fundamental principles behind our deworming practices? What should we be communicating to the pet owners? Considerations surrounding the rotation of active ingredients and resistance development should be revisited once a in a while based on your experience as a practice and the cases seen in your facility. How do we approach the less common infestations with parasites such as *Spirocercalupi*, coccidiosis and Giardia?

External parasites like ticks, fleas, sarcoptic mange, demodectic mange, flies, etc. continue to plague us and applying products onto or within our pets may not be sufficient. Environmental control is sometimes required and may very well be a strategy that will make all the difference. What are some of the myths that create problems for our pets, clients and ourselves? Equipping ourselves against these myths, empowering our clients with the correct knowledge and applying the basics consistently has been shown to have a significant impact on the basic welfare of our pets, the success of our treatments and the risk associated with the environment within which we live.

LIFETIME NUTRITION - START AT THE VERY BEGINNING

Guy Fyvie BVSc
Hill's Pet Nutrition

How we feed growing puppies and kittens not only affects their current wellbeing, but has long term consequences on their health and happiness. Proper skeletal development, immune systems, brain function, skin etc. are all affected by the way we feed during the growth phase.
We will cover the nutrients of concern and how they interact, as well as protocols that should be followed to ensure puppies and kittens that grow up to be healthy adults.

IT WASN'T ME!
ACCEPTING ACCOUNTABILITY

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As we develop and grow in our Veterinary Practices, one of the key areas to focus on is our important part of a bigger team. Problems and difficulties crop up daily and it would seem easier to avoid and dodge the responsibilities placed before us.

To fully understand the importance of developing accountability, we need to explore the behaviours that either leave us empowered or the powerless victim. Whether we are a product of our environment or we take charge of the environment around us.

We will start at the very beginning of the ownership process moving to why being an accountable member of our team would have any benefit. This is a talk designed to successfully take nurses to the next level of their professional and personal development.

BREAKING THE CYCLE OF BURNOUT

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Burn Out is often misunderstood and perceived to be something it is not. Burn Out has many faces ranging from emotional exhaustion to depersonalization of others, even a loss of self respect and lowered self-esteem.

Professionals often engage in self-talk that compounds and accelerates situations that were previously achievable to 'out of control'. Exploring various contributors to Burn Out can help slow or eliminate the effects both emotionally and physically. With Burnout relating more to the loss of the desire to work and not necessarily to trauma, the differentiation needs to be made between Burn Out and Compassion Fatigue.

The causes, symptoms, red flags and myths of Compassion Fatigue will be discussed along with the ever presence of Depression and its contributions to Compassion Fatigue and Burn Out. The mystery of Work Life Balance in practices is revealed along with strategies to create a work environment and lifestyle that is healthy and thriving.

VETERINARY NOSOCOMIAL INFECTIONS

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Veterinary hospital acquired infections are a matter of fact, with an incidence of 4-9% (Mielke, 2010), compared to a human level of 5-10% (Burke, 2003). Hospitals are occupied by sick patients. Nosocomial infection outbreaks, with differing aetiologies have been documented, a significant percentage of which have been zoonotic infections (Milton et al 2015). A nosocomial (hospital acquired), infections are contracted in a hospital environment, arising between 48 hours following admission, up until 30 days following discharge. Common nosocomial infections in the veterinary hospital include urinary tract infections, surgical wound infections and infectious diarrhoea. Historically efforts focused on the control of infectious diseases such as canine parvovirus, more recently it is the control of infectious zoonotic diseases such as MRSA, *C. difficile* and MRSP that have taken precedent. It is accepted that such infections are endemic within veterinary hospitals. The longer a patient is hospitalised, the more invasive the procedure (e.g. i/v or urinary catheters), the greater the risk.

With the now necessary reduction in the use of prophylactic antibiosis for hospitalised surgical and medical cases, all practices must re-harness the old adage ‘Cleanliness is next to Godliness’, returning to the days of Florence Nightingale, when cleanliness and infection control was taken very seriously in all hospitals, knowing that a lack of infection control was a matter of life and death.

This presentation will tackle practical aspects of cleaning, disinfection, fomite identification and most importantly measuring the efficiency of biosecurity management using Adenine Triphosphate testing to verify cleanliness.

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PEDIATRIC VS. GERIATRIC ANAESTHESIA

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The frequency with which we are asked to anaesthetize the very young or very old is increasing in veterinary clinical practice. This is a result of increased interest in early spaying and neutering, veterinary care becoming more available, better health care and an increased population of aged pets.

Pediatric dogs and cats are considered to be less than 12 weeks of age. Defining the geriatric period is more difficult because of species, breed and individual variation in life expectancy. Geriatric patients are those considered to have attained 75% of their expected life span.

Pediatric patients gradually develop their physiological responses and organ function to resemble the adult animal. The majority of circulatory, ventilatory, thermoregulation, hepatic and renal functions are well developed but not yet to the capacity of an adult. Aging is an all-encompassing multifactorial process. In the geriatric patient this results in a decreased capacity for adaptation and produces a decrease in functional reserve of all the organ systems. Aging is not a disease in itself but may be accompanied by the development of many age-related diseases. The aging process varies from individual to individual and from one organ system to another within a given patient.

MINIMISING ANAESTHETIC RISKS

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The purpose of anaesthesia is to provide reversible unconsciousness, amnesia, analgesia and immobility for invasive procedures. The administration of all anaesthetic drugs, the recumbent and immobile state compromise patient homeostasis. Anaesthetic complications and crises are unpredictable and tend to be rapid as well as devastating in nature. The purpose of monitoring during the peri-anaesthetic period is to maximize safety.

The main areas of minimizing anaesthetic complications can be divided into 3 categories. They include patient evaluation, peri-operative checks and procedure factors. Patient evaluation includes physical examination and medical history, which determines which laboratory, and diagnostic procedures are needed. Peri-operative checks include all the equipment and drugs needed for the anaesthetic management for each case. Procedure factors include the influence the procedure performed will have on the homeostasis and clinical parameters of the patient.

THE TICKING TIME BOMB! TOOLS FOR TIME MANAGEMENT

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There is never enough time in the day to do the things we need to do and so often it leaves us feeling defeated and hopeless in our practices. Time management is a skill that if exercised, can empower and transform the productivity and work ethos of any team.

Understanding the difference between Urgent and Important tasks can streamline our action process. We will go through the Six Steps to Time Management and discuss how implementing these in our practices can benefit us, our team and our patients.

Procrastination is a huge obstacle to productivity in any setting. We will explore why we are drawn to procrastinating and how to avoid the lure of falling into the same time-sucking behaviours that eat away at our motivation and productivity. And just to make sure we leave skilled and motivated for any task ahead – we will learn how to confidently ‘Eat that Frog’!

CHEETAH MANAGEMENT – LESSONS LEARNT

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I started working at the Hoedspruit Cheetah Project in 1995. The reason for appointing a full time vet was because of a very high percentage of neonatal mortalities experienced in the cheetahs. Together with a committee of experts from the Onderstepoort Faculty of Veterinary Science, the problem was found to be a Vitamin E deficiency complicated by a Salmonella septicaemia. A supplement was developed and added to the meat that was fed and the problem disappeared literally overnight. The initial anaesthetics that I used were Saffan (intravenous) or a Ketamine/Xylazine combination. Zoletil soon came on the market and was a wonderful drug to use, especially in debilitated animals. I had a few problems however- losing one animal due to an allergic type reaction and having similar problems with three other animals, which we managed to pull through. Medetomidine/Ketamine combination became my anaesthetic of choice until the advent of Bamanil (Butorphanol : Azaperone : Medetomidine combination).

Antibiotics used commonly in cheetah include long acting Penicillin (Lentrax, Procapen L/A) Enrofloxacin (Baytril), Amoxycillin:Clavulanic Acid (Synulox) and more recently Cefovecin (Convenia) A vaccination program starting with an inactivated vaccine Felovax IV plus Calicivax as soon as possible after 9 weeks followed by a booster 3 weeks later and another booster of live vaccine Felocell 3 or 4, three weeks later. Rabies vaccine is also given twice in the same period. Mild cases of Herpes and Calici virus infections have been diagnosed periodically – even now that we vaccinate the cheetahs twice a year. Internal parasites do not appear to be too much of a problem but external parasites, especially fleas, can be. Ticks are not much of a problem, however. Frontline Plus, Bravecto and Comfortis are used to combat these potential problems. Fungal infections of the hair causing the hair to break off have also been diagnosed in the younger animals. The causative organism, *Trichophyton mentagrophytes* is easily controlled using a lime sulphur dip. True cases of ringworm were diagnosed at another rehabilitation centre, which responded well to six weeks of treatment with Griseofulvin. This centre also had two cheetahs die due to bovine tuberculosis (*Mycobacteria bovis*), which may have been introduced from infected carcasses.

More recently, mortalities were experienced due to a severe, acute nephrosis. Extensive research eventually found that this was caused by NSAIDs residues, in this case phenylbutazone in the meat that was fed.

In the last year, a syndrome appeared in younger cheetah that started biting the ends of their tails. Histopathology revealed dermatitis, peri-neuritis and a vasculitus but the aetiology is still unknown.

OVERVIEW OF DISEASES OF THE AFRICAN RHINOCEROS

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ABSTRACT

With increasing threats from poaching and habitat fragmentation, African rhinoceros are being more intensively managed. Therefore, wildlife veterinarians need to be aware of diseases, diagnostic and therapeutic approaches that may be required to address health issues in these species. This presentation will provide an overview of some of the infectious and non-infectious diseases that have been reported in black and white rhinoceros. The information serves as a foundation for dealing with morbidity and mortality in African rhinoceros.

BACTERIAL DISEASES

Salmonellosis

This is a clinically important disease of rhinos, especially in confinement. Clinical syndromes vary; non-specific signs, anorexia, and lethargy; enteric disease with diarrhoea or bloody feces, colic; septicaemia, which may be fatal. Animals may recover and eliminate infection or they may become a carrier with inapparent shedding or intermittent clinical signs. In a study of black rhinos in a zoo, four of six animals were intermittent shedders with 2.4% of faecal samples culture positive.[1] Diagnosis is based on culture or PCR and serotyping. Isolation of Salmonella in an ill animal does NOT necessarily identify this as a cause of the clinical signs. Rhinos receiving antibiotics or immunocompromised by other disease or stress may shed bacteria. Intermittent shedding requires that samples are submitted for multiple cultures. Serotyping is based on cell wall and flagellar antigens, as well as biochemical characteristics; this is important epidemiologically. Treatment is primarily supportive, including fluid therapy, nutritional support, and anti-inflammatories. Antibiotics may be used but should be based on sensitivities since therapy may lead to a carrier state or antibiotic resistance. Generally antibiotics are used in more serious cases, young, or compromised animals. Predisposing factors for Salmonellosis include stressful events, such as transport or introductions, changes in feed resulting in gut flora imbalance, and concurrent disease. Preventive measures should minimize stress, provide good hygiene, and isolation of any animals that are shedding bacteria.

Tuberculosis (Mycobacteriosis)

This is a sporadic disease with significant clinical and regulatory consequences. It can be caused by either *M. bovis* or *M. tuberculosis* in rhinos. TB has been documented in white, black, greater one-horned, and Sumatran rhinos in captivity.[2] Anecdotal evidence suggests that browsing rhinos may be more susceptible. TB typically affects adults. Clinical signs vary – dyspnoea, coughing, nasal discharge, weight loss, weakness, or lethargy. Often animals are asymptomatic until disease is advanced, which may take months to years. At least 21 cases have been documented in African rhinos in zoos.[2] There have been four cases of documented *M. bovis* infection in black rhinos in South Africa.[3] Ante-mortem diagnosis is very difficult. Intradermal tuberculin testing is unreliable in rhinos. Tracheo-bronchial and gastric lavage samples can be used for mycobacterial culture. Serology appears promising and there are some commercial kits available (Chembio VetTB DPP). Research on experimental cytokine assays is underway. However, most diagnoses are made post-mortem.

Paratuberculosis (*M. avium* subsp. *paratuberculosis)*

This has been diagnosed in a single captive black rhino.[4] The animal had a 4 month history of diarrhea and weight loss. *M. avium* subsp. *paratuberculosis* was isolated from a faecal culture. The rhino was treated with anti-mycobacterial drugs and clinically resolved.

Leptospirosis
his is a zoonotic disease with worldwide distribution. Leptospirosis is caused by one of more than 250 pathogenic serovars of Leptospira. Rodents are the maintenance hosts. Antibodies has been reported in wild black and white rhinos in range countries.[5,6] Urine-contaminated feed is the source of infection. Transmission occurs through contact of the bacteria with mucous membranes or damaged skin. Leptospires can colonize the liver, kidneys, lungs, genital tract, and CNS. Clinical signs are variable; acute, systemic febrile disease with renal and/or hepatic damage has bene reported in black rhinos; uveitis, haemolytic anaemia, muscle pain, and abortion/stillbirth may also occur. Leptospirosis can be confirmed by identifying bacteria in blood, urine or tissues using immunofluorescence, PCR, or culture; however, it is more commonly diagnosed by elevated antibody titres using a microagglutination test. A 4-fold increase in serum samples taken 7-10 days apart or a single titer>1:800-1600 with compatible clinical signs are common criteria. Prevention includes good rodent control and vaccination using a polyvalent inactivated vaccine.

Clostridial diseases
***C. septicum* -** Malignant oedema occurs when there is bacterial contamination of wounds with local toxins causing severe oedema and necrosis. This disease may be a concern for rhinos if there is fighting, transport, or other wounds.
C. tetani - There are rare cases of tetanus in rhinos associated with spore-contaminated wounds. Clinical signs are similar to those in domestic animals. Equine tetanus vaccine has been used in captive rhinos.
C. novyi - There has been a fatal outbreak in white and black rhinos in semi-intensive management. Presented with peracute signs; typically, animals would collapse and progress rapidly to death. On post-mortem, hemorrhagic enterocolitis was evident. A vaccine has been developed.
C. perfringens (Enterotoxaemia) - This has resulted in 9 black rhino mortalities in Kenya[7], as well as morbidity in captive black and white rhinos. Clinical signs are often peracute with severe abdominal pain, laboured breathing, and death within hours. Severe necrotizing haemorrhagic enteritis is found at necropsy. Overgrowth of intestinal flora can be precipitated by change in diet, stress, and antibiotic treatment.

Diagnosis of clostridial diseases usually requires anaerobic culture of gastric or small intestinal contents, or tissue (including wounds). There are PCR assays for toxin genes. A mouse bioassay can also be used to detect toxins. Histopathology provides supporting evidence. Treatment requires intensive supportive care and potential administration of antitoxin if diagnosed early. Prevention is the key and there are multivalent vaccines that have been used in rhinos.

Streptococcal and Staphylococcal Infections
Beta haemolytic *Streptococcus* has been isolated from skin lesions and wounds in rhinos; this may progress to septicaemia and death. It has also been linked to vegetative endocarditis, myocardial degeneration, meningoencephalitis, and idiopathic haemorrhagic vasculopathy syndrome in captive black rhinos. Methicillin-resistant *Staphylococcus aureus* (MRSA) has been isolated from chronic wounds on captive rhino feet (zoonotic threat).

VIRAL DISEASES

Encephalomyocarditis virus (EMCV)
Typically a peracute disease with signs consistent with cardiac failure. Non-suppurative myocarditis and pulmonary oedema found post-mortem. Survivors may have cardiac damage. Exposure is measured by antibody titres, with diagnosis confirmed using virus culture, PCR, and/or histopathology.

Arboviral diseases
These are caused by a diverse group of vector-borne viruses (usually mosquitoes and midges). Viruses that have been reported in viruses include Shuni virus (Orthobunyavirus), Sindbis and Middelburg viruses (Alphaviruses), and West Nile virus (Flavivirus). Shuni, Sindbis and Middelburg viruses have been identified in rhinos with neurological signs; weakness, progressive paralysis and death within 24 hours of recumbency. West Nile virus infection has been suspected in zoo rhinos, causing nonspecific signs such as lethargy. Rhinos in endemic areas commonly develop antibody titres (up to 1:640). Antibodies to other arboviruses have been found in rhinos, but not associated with disease; Wesselbron, African Horse Sickness, Bluetongue, and Rift Valley Fever (high

seroprevalence in some areas).[6,8] Diagnosis is usually by serological tests (ELISA, plaque reduction neutralization test, etc.) Other available tests include PCR and immunohistochemistry for some viruses. Management requires good vector control and consideration of vaccination in captive rhino (for example WNV).

FUNGAL DISEASE

Aspergillosis

Fungal pneumonia caused by *Aspergillus* sp. has been identified primarily in captive black rhinos.[9] These cases were usually associated with immunosuppressive diseases or therapy (ex. steroid administration). All rhinos had concurrent disease such as anaemia, mucocutaneous ulcers, or TB. Diagnosis is based on Aspergillosis serology, fungal culture, and/or histopathology. Treatment is usually unsuccessful (eg. itraconazole).

PROTOZOAL AND PARASITIC DISEASES

Tick-borne protozoal diseases

Babesia bicornis (piroplasm) has been reported to cause mortalities in black rhinos in Tanzania and RSA.[10] However, the organism has also been found in healthy animals. Disease may be precipitated by stress. *Theileria bicornis* (piroplasm) has also been found in black and white rhinos in Kenya and RSA, but has not been associated with disease. *Ehrlichia ruminantium* may infect black and white rhinos, based on the presence of antibodies to heartwater found in animals in Zimbabwe.

Neosporosis (*N. caninum*)

N. caninum is a coccidian parasite of domestic dogs found worldwide. Transmission is faecal-oral or transplacental. Abortion, acute death (myocarditis) have been seen in white rhinos, and may also cause neurological signs.[11,12] Diagnosis is based on serological tests (ELISA, IFAT, agglutination), immunohistochemistry, or PCR.

Trypanosomiasis (*T. vivax, T. congolense, T. simiae, T. godfreyi, T. evansi*)

Mortalities have been associated with translocation of black and white rhinos from fly-free to tsetse areas due to Trypanosomiasis.[13] Animals exhibit loss of condition, weakness, and death associated with anaemia, leukopenia, thrombocytopenia, and hypoproteinaemia. There has been a fatal outbreak of *T. evansi* in Sumatran rhinos in a Malaysian sanctuary.[14] Acquired immunity results in asymptomatic infection until an individual is subjected to stress. Diagnosis is based on blood smear, PCR, IHC, and ELISA. Management requires tsetse control and possibly strategic use of trypanocides (diminazene aceturate).

Filarial skin disease

Stephanofilaria dinniki is a subcutaneous parasite of black rhinos that usually results in self-limiting skin disease. Clinical manifestation may be associated with environmental conditions and stress. There has been an outbreak of skin lesions in black and white rhinos in Meru National Park.[15] Although they were unable to identify the parasites, lesions responded to treatment with ivermectin, amoxicillin and debridement.

TOXIC, METABOLIC/NUTRITIONAL, NONINFECTIOUS AND IDIOPATHIC DISEASES

Cyanobacteria intoxication

Blue-green algae (*Microcystis* spp., others) release biotoxins that are hepato- and neurotoxic. Ingestion while drinking results in death of rhinos and other species due to liver and respiratory failure.[16] On necropsy, the liver is enlarged and friable, with widespread areas of haemorrhage.

Iron Overload Disorder (IOD)

Previously known as iron storage disease, this is an important disease of captive black rhinos.[17,18] It has also been observed in recently captured black rhinos placed in bomas (Zimbabwe). Unknown etiology, but suspected to be related to captive diets. Usually associated with nonspecific clinical signs. Diagnosis is based on iron profiles (TIBC, ferritin), and histopathology. Treatment can include therapeutic phlebotomy and or chelation therapy.

Chronic renal disease

This is an emerging problem in rhinos (both black and white).[19] Based on necropsies of captive black rhinos performed between 2007 and 2012, 74% of animals had significant renal disease. Unfortunately, there are no obvious clinicopathological changes that provide a clear diagnosis. Progression usually to renal failure with development of anaemia, lethargy, decreased appetite, and loss of condition. Aetiology is unknown but may be related to diets high in protein (Lucerne, alfalfa) and chronic nonspecific inflammation. Diagnosis is usually made post-mortem. Treatment is supportive.

SUMMARY

Infectious, metabolic, nutritional, immune-mediated, toxic, traumatic, and idiopathic diseases may affect white and black rhinos. Differential diagnostic list in ill/dead rhinos should include common and uncommon diseases of domestic animals. Further studies are required to investigate epidemiology of diseases in these species.

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FIRST AID AND IMMOBILISATION DRUG EXPOSURE

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Game capture (physical or chemical immobilization) often involves the use of dangerous equipment, animals and drugs which if handled incorrectly could lead to human injury and potentially death. Precautions need to be taken to avoid this. Adequate knowledge of the subject, thorough planning before a capture operation and a well trained capture team will all aid in lowering the risk of injury and death. Accidents do however happen and having a thorough knowledge of first aid and drug intoxication could be life saving. The veterinarian is often the best-qualified person to handle a first-aid case but training other personnel in the capture team is invaluable. Minor injuries resulting from capture equipment and animals are common. Occasionally animals may lethally injure a person. Most of the lethal injuries result from penetrating wounds from horns or tusks. By far the most common potential lethal risk is the accidental exposure to capture drugs like the potent opioids. Humans are highly sensitive to these drugs and small volumes may be enough to cause death. A thorough understanding of the pharmacology of these drugs and basic life-support is of utmost importance when veterinarians decide to use and handle these drugs.

WHAT FUTURE FOR AFRICAN VULTURES?

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The Asian vulture crisis, caused losses of >98% of the population between 1992-2007 (Cuthbert *et al* 2009) due to Diclofenac. Press and conservationists have been concerned with 'sexy threatened species in Africa' (rhino, lion, elephant), forgetting 'natures clean up team' - the vultures (playing a vital role in clearing away carcasses, anthrax, rabies, tuberculosis and botulism). In 2015 six of Africa's 11 vulture spp – had an upgraded risk of extinction, (Birdlife International 2015). African vulture populations (outside protected areas) have declined by 98% in the last 30 years (Thiollay 2006). In Africa there is not a single attributable cause so control is challenging. A recent review of vulture losses (Ogada 2015) reported the use of vulture body parts in traditional medicine ('muti') accounting for 29% of losses. Accidental poisonings resulted in 94 vultures a year between 1970 – 2011, (3967), the greatest increase is seen in deliberate targeting by poachers (681 vultures per year 2012-2014 (2044 in total), with average fatalities 186 per incident, ranging 1-700). Carbamate pesticides are being misused by livestock owners to poison predators such as lions and hyenas (Ogada 2014), causing a reduction of crushed bone in vulture diets, leading to calcium deficiencies in developing chicks. Vulture deaths were caused by malicious or accidental poisoning 61%, traditional medicine 29%, food 1% (i.e. 91% avoidable), with 9% caused by trauma, electrical supply or infra-structure. Vulture restaurants have proven popular, but research has shown a vulture home range of 30,000 to 80,000 Km^2, travelling up to 250km a day (Kendall & Virana 2012), so poisoning still looms large. Vultures are vital to the eco-system consuming 70% of all meat in the Masai Mara. What can and should we be doing as a profession to slow or reverse this calamity.

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TRIAGE AND FIRST AID FOR INJURED FREE-LIVING AFRICAN VULTURES

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Three African vulture species are now defined as 'Endangered', whilst four are 'Critically Endangered'. Not only for welfare reasons, but also to maximise the recovery and release of vultures back to the wild, prompt and efficient care of wildlife casualty cases is vitally important. Managing wildlife casualty cases is akin to working in a war zone. All birds should undergo initial 'triage': cases are given a brief (so as not to cause excessive stress), clinical examination, with cases being divided into:

- Hopeless (obvious critical injuries) should euthanase as soon as possible
- Critical - needs urgent care to save
- Less urgent or critical needs.

When dealing with endangered species, the initial consideration is not whether the bird will ever be fit for release, and euthanasia if not, as unreleasable birds may serve a valuable contribution to species survival if they can become fit enough to breed in captivity, or even to act as a foster parent for orphan chicks.

Considering the causes of death in wild African vultures:

- 61% poisoning
- 9% electrocution / power line collisions
- 29% muti related
- 1% for food.

Many of the poisoning cases will never reach veterinary care, however prompt treatment in that situation is vitally important. Casualty cases may often present with neurological abnormalities, aetiologies will be varied:

- Toxins
- Heavy metal
- Hypocalcaemia
- Hyper or hypo glycaemia
- Septicaemia/viraemia
- Trauma or localised infection
- Control seizures / CNS signs.

Of these toxins are the most significant group:

- Organophosphate: e.g. acephate, carbosulfan, malathion, chlorpyrifos, dimethoate, diazinon.
- Organochlorine: (Highly residual in the environment), e.g. endosulfan, dieldrin, DDT.
- Carbamates: (V fast acting – 2 step), e.g. alidicarb, carbofuran, carbosulfan.

Toxicity

Clinical signs of OP or carbamate toxicity

Convulsions, hyper-excitability, incoordination of muscular action (ataxia), muscular weakness (myasthenia), difficult breathing (dyspnoea), rapid breathing (tachypnoea), vomiting, defecation, diarrhoea, spasmodic contraction of anal sphincter (tenesmus, lethargy, induced tranquillity, head and limbs arched back (opisthotonos), slight paralysis (paresis), blindness, contraction of pupils (miosis), dilation of pupils (mydriasis), drooping of eyelid (ptosis), protrusion of eyes (exopthalmia), excessive tear formation (lacrimation), excessive thirst (polydipsia), bleeding from nares (epistaxis), erection of contour feathers (piloerection).

Treating toxicities

If possible find the poison type. However speed is essential, typically ineffective if not commenced before 24-48hrs.

OP – treat with 2 PAM (pralidoxime chloride), at 100mg/kg once IV, repeated after 6 hours if necessary, then every 24hrs as required. If OP is not treated within the first 24 hrs, therapy may actually make signs worse, i.e. treatment is contraindicated.

Carbamate – treat with atropine 50mg/kg, if responds then deteriorates, repeat treatments as often as necessary, to maintain response.

If uncertain of poison type: Atropine at 25mg/kg plus 2 PAM at 50mg/kg, repeated as necessary.

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RHINO IMMOBILIZATION, ORPHAN HANDREARING, DEHORNING AND TREATING POACHING VICTIMS – RECENT DEVELOPMENTS

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With the current rhino poaching epidemic, a lot of work has been done on improving techniques involved in rhino anaesthesia, the hand rearing of orphaned rhino calves, and the treating of injured rhino poaching victims.

As far as rhino anaesthesia is concerned, the original drug, etorphine hydrochloride is still the drug of choice. It is, however, a severe cardiorespiratory depressant – the symptoms of which are alleviated using butorphanol together with oxygen supplementation. The hypertension associated with opioids is remedied to a large extent using azaperone in the cocktail. Hyaluronidase is added to decrease the induction period. Recent research has shown that a sternal recumbency anaesthetic is far superior and safer than an anaesthetic in lateral recumbency.

One of the rhino poaching consequences is that many rhino calves are orphaned. Here I usually use a butorphanol, azaperone and hyalase combination or miniscule amounts of etorphine, azaperone and hyalase in order to immobilize them. Dehydration, hypothermia and digestive issues are common place. The administration of rhino plasma to calves showing clinical signs associated with a low immunity can be a vital tool. Antibiotics and anti-inflammatories are also used when indicated.

The only effective and practical proactive measure at the moment as far as rhino poaching is concerned, is dehorning. The treatment of poaching survivors is now common place. It is virtually impossible to remove the bullet so the wound needs to be flushed out with antiseptics and the animal treated with antibiotics and anti-inflammatories. More recently, I have had to treat rhinos that have been poached using etorphine and darts – the survivors are then left walking around with huge holes extending into their paranasal sinuses. The holes are closed using a plastic type prosthesis and allowed to granulate underneath.

COMMON AND EMERGING INFECTIOUS DISEASES OF FARMED SABLE ANTELOPE

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The sable antelope is a commonly farmed species in South Africa mainly due to their attractiveness especially among trophy hunters. The high value of sable antelope has resulted in animals being kept in smaller camp systems and with this intensification comes an increased risk to diseases. Common infectious diseases include Haemonchosis, Theileriosis, Clostridial myositis, Teaniosis and Coccidiosis. Emerging diseases include Brucellosis, Q-fever, Pneumonia and Tuberculosis. Other documented diseases include Babesiosis, Dermatophilosus and Rift Valley Fever. Haemonchosis is by far the largest threat to the sable farming industry, as sable antelope appear to be highly susceptible to *Haemonchus contortus* infestation. Deworming is commonly performed on farms but misuse of anthelmintics has led to widespread resistance. Theileriosis may be more common in sable antelope in certain regions of South Africa due to the tick distribution. This area seems to be ever increasing. Holistic parasite management and better farming practices may aid in reducing clinical disease in both Haemonchosis and Theileriosis. The aim should be to breed immune and resilient sable antelope. In this paper we look at the diagnosis, treatment and prevention of the diseases of sable antelope.

REVIEW OF BASIC RUMINANT DIGESTION - APPLICATION IN GAME RANCHING

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ABSTRACT

General principals of ruminant digestion is reviewed with the focus on practical application in game farming. Wildlife veterinarians in the field are often confronted with issues of sub-optimal production, disease and even deaths in antelope species, which can be attributed (at least indirectly) to sub-optimal nutrition. One reason is the intensification of wildlife farming practices, which led to animals not having access to adequate natural vegetation in many instances. Another problem is where species are kept in habitats not suited to their needs. It is necessary for veterinarians dealing with these issues to have a basic understanding of ruminant digestion and nutritional needs in order to provide meaningful advice to farmers in this regard.

INTRODUCTION

No vertebrate animal is able to digest plant fibre, as they do not possess the necessary enzymes. There is a lack of appreciation for the wonder of ruminants: they evolved to become hosts to billions upon billions of bacteria (as well as protozoa and fungi) to live in a symbiotic relationship: the micro-organisms ferment plant material (including fibre) in the rumen and in the process the micro-organisms themselves become the main protein source and provide the main energy substrate to the ruminant.

The ruminant stomach takes up almost 75 percent of the space in the abdominal cavity. The rumen and reticulum comprise 84 percent of the volume of the total stomach, the omasum 12 percent, and the abomasum 4 percent.[5] One milliliter of rumen fluid contains 10-50 billion microbes and over 1 million protozoa.[6]

The rumen is adapted for the digestion of fibre. Ingested feedstuffs arrive in the reticulo-rumen and get exposed to the rumen micro-organisms. The rumen is never stationary, and constant contractions (one to two a minute on average) mix the content of the rumen, ensuring that the micro-organisms are exposed to all of the feedstuffs. Ruminating helps to physically break down the rumen content into smaller pieces, increasing the surface area to which the micro-organisms are exposed. Good quality of adequate length roughage is required to stimulate rumen wall contractions; this is called the roughage effect. As a rule of thumb, the width of the mouth of the ruminant can be used as in indication of minimum required fibre length for the relevant species.

RUMINANT DIGESTION

Carbohydrate digestion

Plant material consists of +-75% carbohydrates. The end products of microbial fermentation of carbohydrates in the rumen are:

- Volatile fatty acids (energy for the ruminant)
- Ammonia (protein for the ruminant as it is used to form more micro-organisms)
- Gasses (mainly carbon dioxide and methane).

- Non-structural carbohydrate (sugars and starch) fermentation:
  - Happens relatively fast (especially sugars)
  - Creates an acidic environment which is deleterious to micro-organisms responsible for fibre fermentation.[3]
  - Favours the yield of propionic acid (used for glucose synthesis).[5]
  - If intake is too high, will suppress roughage intake.[4]

- Structural carbohydrate (fibre) fermentation:
  - Happens a lot slower compared to fermentation of non-structural carbohydrates (starches and sugars).

- Responsible micro-organisms cannot tolerate an acidic environment, as caused by increased non-structural carbohydrate fermentation.[3]
- Favours the yield of acetic acid (used for sat synthesis)[5], which increases milk fat content

Protein digestion

- Degradable intake protein (DIP):
  Protein undergoing fermentation in the rumen is converted to ammonia, organic acids and amino acid. Approximately 40 to 75 percent of the natural protein in feed is broken down in the rumen.[1]
  Rumen microbes convert the ammonia and organic acids into amino acids that are assembled into microbial protein. Microbes are flushed through the rumen with the rest of the degraded rumen content and become the main protein source to the ruminant (bacteria are 60% protein).[2]

- Undegradable intake protein (UIP)
  Passes through the rumen undigested and becomes available as protein source directly to the ruminant.

Fat digestion

Most of the digestion and absorption of fat occurs in the small intestine. Rumen micro-organisms change unsaturated fatty acids to saturated acids through the addition of hydrogen molecules. Thus, ruminants absorb more saturated fat than simple-stomach animals. Feeding large quantities of unsaturated fatty acids can be toxic to rumen bacteria, depress fibre digestion, and lower rumen pH.[1]

COMMON NUTRITION-RELATED PROBLEMS ENCOUNTERED ON GAME FARMS

Intensive (small camp) systems

Farmers often focus on the concentrate portion of antelope nutrition. In zero grazing camps it is very common that animals are fed a combination of pellets and lucerne only. It is important to realise that lucerne (especially good quality lucerne) is very easily digestible and does not contain enough slowly digestible fibre to ensure optimal rumen health. Add a good quality roughage source e.g. teff or oat hay for grazers.
A second issue to keep in mind where lucerne is the only form of roughage is the calcium to phosphate ratio:
Lucerne contains 14 grams of calcium per kg and 2 grams of phosphate, thus a Ca: PO_4 of 7:1 which is much higher than recommended.
A total mixed ration is preferred in a zero grazing system, to prevent selective feeding.

Semi-extensive systems

Case study in Northern Cape (Kimberley area):
Sable and Roan antelope managed according to the regional recommended carrying capacity - no overgrazing, and thus adequate availability of natural grazing. Animals were in good condition in summer, but poor condition in winter. The animals showed pica in late summer/autumn.

The animals were supplemented in the dry season with a mixture of maize, lucerne and a commercial molasses based meal containing trace elements. Enough of each feedstuff was supplied in order to completely fulfill the total energy and protein needs of each animal. Yet the animals' condition in winter remained poor, even though they reproduced well.

The main problems with this system were identified as follows:
1. High grain intake suppressed roughage intake of natural grazing.[4]
2. Lower roughage intake = slower saliva flow = less buffering effect = low grade acidosis
3. High grain intake led to increased VFA production with lactic acid byproduct - also led to acidosis
4. Lower rumen pH detrimental to the rumen micro-organisms responsible for roughage fermentation - which led to slower digestion and even less total intake of feed

The effect was chronic, low-grade acidosis leading to inadequate digestion as well as reduced total feed intake, with the result of excessive weight loss in winter.

The main purpose of supplemental feeding on natural winter grazing is to provide optimal conditions for the micro-organisms to thrive, by adding what is lacking in the grazing - address each feed component:

- Roughage:
  A combination of easily digestible and slower digestible is needed, but in this case the slower digestible was supplied in abundance from the natural vegetation.
- Protein:
  This was the most lacking substance in the original supplement.
- Energy:
  Adequate easily available energy (e.g. sugars, starches or soluble fibre) should be available, but be careful not to over-supply, as was originally the case.
- Macro minerals: (Depending on the area) - phosphate deficiency was the main macro mineral deficiency in this case, leading to pica.
- Trace elements: Consider trace elements, especially those responsible for a healthy immune system (e.g. copper, zinc, selenium). Remember organic vs. inorganic is not the same! For example: inorganic selenium is a pro-oxidant and suppresses the absorption of copper, whereas organic selenium is an anti-oxidant and promotes the absorption of copper.[7]

SUMMARY

Think from big to small:

- Roughage
- Protein/Energy
- (fat)
- Macro minerals
- Trace elements

Remember antelope are ruminants. Always first consider whether roughage supply is adequate. On farms where there is adequate roughage, do not waste money on buying in roughage. Aim to determine that which is lacking on the specific farm, and strategically supplement accordingly.
Lucerne on its own does not provide adequate roughage for grazer antelope species.
Phosphate deficiencies should be considered for most parts of South Africa.

Veterinarians have an excellent foundation to develop knowledge and understanding of ruminant nutrition. It is worthwhile for the game veterinarian to invest time in developing his/her skills in this field. An important contribution can be made to the general health of antelope species if sound nutrition-related advice can be provided to the farmer.

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AVIAN EMERGENCIES, TRIAGE AND STABILISATION

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ABSTRACT

Many general practitioners have had unpleasant experiences with ill birds in that they are perceived to die as soon as the well meaning vet begins to examine them. Often the vet is thus cautious to treat ill avian patients. With a basic understanding of what constitutes an emergency in an avian patient as well as the initial steps to take in stabilising the bird, treating the ill bird may become a much more rewarding experience.

WHAT CONSTITUTES AN EMERGENCY

Unlike truly domesticated companion animals, birds are at best only a few generations away from their wild ancestors. Wild animals know that should they appear weak, they become a target for predators. An ill bird will thus mask its symptoms until late in the disease process. The signs of illness in avian patients are subtle until the bird is almost moribund[2].

Typical indicators of ill health include depression (not talking, less mobile than normal, fluffed up feathers, drooping eyelids, easier to handle etc); anorexia (distinguish between mouthing and crumbling food and actually ingesting it); changes in stool production or appearance (diarrhoea, polyuria, scant or dark stool, melaena, haematochezia etc); weight loss (evaluated by palpating the pectoral muscle mass over the sternum); dyspnoea (including tail bobbing, tachypnoea, loss of normal voice, open mouthed breathing and exercise intolerance) and poor peripheral perfusion (evaluated by examining the turgidity and refill time of the medial ulnar (basilic) vein)[2].
Sitting at the bottom of the cage (in perching species), respiratory difficulty, haemorrhage, regurgitation and anorexia are all considered emergencies[1].

Any bird showing obvious signs of ill health in the strange environment of the veterinary clinic (where the stress of the foreign environment should make the bird mask its illness as much as possible) is extremely compromised.

STABILISATION

The basics of emergency care include supplemental Oxygen (by mask or in an induction chamber), fluid therapy (preferably intravenous but intraosseous or subcutaneous administration can also be useful) and warmth.

We will typically place a compromised bird into an Oxygen chamber for 10-15 minutes before performing a clinical exam. Covering the oxygen chamber to provide a secluded environment is also beneficial to reduce stress[1]. During this period a detailed history should be taken and all equipment necessary to treat the patient should be prepared.

The initial examination is hands off. The birds posture, ability to ambulate, interest in the environment and respiratory status as well as any signs in the cage of vomition, diarrhoea, haemorrhage or sources of lead or zinc can be assessed[1].

During the clinical examination care should be taken not to place any pressure over the ribs or sternum. Having no diaphragm, birds ventilate the lungs and airsacs by expansion of the chest and it is easy for an inexperienced handler to accidentally compress the sternum to the point where hypoventilation occurs.

A dyspnoeic bird should also not be placed into dorsal recumbency. Many birds with respiratory tract disease will have fluid in their airsacs and dorsal recumbency could potentially allow fluid to drain from the airsacs into the lungs thus effectively drowning the bird.

A thorough hands-on clinical exam should be performed only once the bird has stabilised[1]. A suitably sized towel is used to gently enfold the bird for examination. One hand is used to surround the bird's neck and shoulders and the other hand is used to bundle the wings and feet into the towel. Towel handling is minimally traumatic for the bird and does not predispose them to hand-shyness. The towel also protects the feathers and is perceived as less traumatic by the owner. For biosecurity reasons a fresh towel is used for each patient.

The basic clinical examination includes cardiac and airsac auscultation, evaluation of body and feather condition, oral examination, evaluation of the refill time of the basilic vein (which can give an indication of blood pressure and hydration status) and abdominal palpation for ascites, eggs or palpable masses. There are numerous excellent articles available on the clinical examination of the bird.

FLUID THERAPY

Fluid therapy is frequently indicated in cases of shock or dehydration. A small (22-26g) gauge intravenous catheter is used to cannulate the basilic vein in most species. The catheter is secured in place by using cyanoacrylate glue on an elastoplast butterfly. The drip line is secured to a few wing feathers to prevent movement of the catheter. In larger and long legged species the medial metatarsal vein is used.

The jugular vein is easily accessible in many species but is not an effective site for cannulation due to the flexibility of the bird's neck. The jugular is however commonly used for blood collection and for emergency boluses of dextrose/colloids etc.

Intraosseous cannulation into the distal ulna or proximal tibia can be used in cases where intravenous access is difficult, as in very small or collapsed patients. Any drug that can be given intravenously can safely be administered into an intraosseous line. As the bone does not expand, fluids should not be introduced too quickly as the increased pressure in the medullary cavity will cause pain. The humerus and femur of many species are pneumatised and connect directly to the airsac system. For this reason these bones should be avoided as sites for intraosseous cannulation.

Lactated Ringers Solution is a good choice of fluid as the lactate present may help to correct metabolic acidosis (it is metabolised in the liver to liberate bicarbonate). The patient's fluid deficit should be corrected over 4-6 hours. Fluid deficit is calculated by: fluid volume (ml) = body weight (g) X %dehydration. Daily maintenance fluid requirements are approximated at 50-60ml/kg/day. This amount is added to the rehydration volume for initial stabilisation.

An avian patient is considered 10% dehydrated when the refill time of the Basilic vein is slowed, the mucous membranes are tacky and the skin is flushed. With more severe dehydration the eyes become sunken and severe depression develops[2].

Colloids such as Hetastarch/Voluven and plasma are highly effective in counteracting shock and acute blood loss. Synthetic colloids may be given at 10ml/kg as a slow bolus over 15 minutes or may be combined with crystalloids as a constant rate infusion. We use a mixture containing 8ml Voluven added to 48ml Lactated Ringers. 4ml of 50% Dextrose may be added. This solution can safely be used at twice maintenance (120ml/kg/day) for several days in cases of shock or severe compromise.

BLOOD TRANSFUSIONS

Total blood volume is estimated to be 10% of body mass. An otherwise healthy bird can lose approximately 30% of its blood volume without life threatening effects. Should the PCV drop below 15%, a blood transfusion should be considered. A volume of 1% of body mass may safely be collected from a donor bird (which should ideally be of the same species, or at least the same genus). Citrate Phosphate Dextrose at 10% of the volume to be collected is an effective anticoagulant. Cross matching is not routinely performed. Anaphylactic reactions are rare.

In an emergency situation, pigeon or chicken blood can be used as a “universal donor” but the erythrocytes transfused into a different species have a much shorter lifespan than those in a homologous transfusion.

HOUSING

Birds have a much higher core temperature than mammals (40-42°C) and due to their high metabolic rates and large relative surface area can become hypothermic very quickly. After warmed fluid administration has been begun we will place the patient in an incubator at approximately 29-32 degrees C.

Post surgical patients are also kept in incubators for recovery as hypothermia develops quickly during anaesthesia and surgical procedures (especially where alcohol is used to prep the bird and when the coelomic cavity is exposed). We never allow an avian patient to lie on a steel table but rather on a blanket with a heating pad placed beneath. Hair driers and even normal desk lamps can be used for supplemental heat in an emergency.

A dimly lit, quiet environment away from potential predator species such as dogs and cats is needed to minimise the stress associated with hospitalisation.

PAIN MANAGEMENT

Extremely important. A bird in pain will often present depressed and immobile. Increased aggression, guarding of the painful body part, tachycardia and tachypnoea are also common indicators of pain. Meloxicam at 0.2 to 0.5mg/kg once daily is an effective Non-Steroidal Anti Inflammatory with minimal side effects and Butorphanol at 0.5-1mg/kg q8hrs[1] is an effective opioid. Buprenorphine has not proven to provide analgesia in the bird[1].

MAINTENANCE OF CALORIC REQUIREMENTS

Hypoglycaemia is common, especially in smaller patients. Birds are only starved for a maximum of 4 hours prior to general anaesthesia and are encouraged to eat as soon as possible on recovery. Ill or compromised avian patients should also be offered tempting foods to encourage eating.

An ill bird will often refuse to descend from the cage perch to feed from a food bowl placed on the floor. Favourite food items should be offered and food and water bowls should be elevated to the level of the bird’s perch to encourage feeding behaviour.

Anorexic avian patients must be assist-fed sooner rather than later. Gavage feeding is indicated with purpose made steel crop needles (in hard billed species such as parrots) or with standard feeding tubes in species incapable of biting through the tube. Useful gavage feeding products include avian handrearing formulas and canine recovery diets, depending on species requirements.

In an adult Psittacine (parrot type bird) approximately 3-5% of body mass may be gavage fed 2-3X daily. Neonates have larger crops and can be fed up to 10% of body mass per feeding.

Crop feeding should be performed after any other planned procedures to minimise the chances of regurgitation and subsequent aspiration[1].

ANAESTHESIA

This may be required for the placement of fluid lines or for certain procedures. Isofluorane or Sevofluorane mask induction is considered the safest induction agent[1]. Intubation and positive pressure ventilation are recommended to prevent hypoventilation. Once again, many texts on avian anaesthesia are available.

CONCLUSION

Although birds are much more fragile than mammals, with return to first principles and a careful eye to notice the subtle signs of disease, treating the ill avian patient can be a rewarding, although challenging experience.

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HOW NOT TO KILL YOUR EXOTIC PATIENTS – COMMON PITFALLS AWAITING THE GENERAL PRACTITIONER

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ABSTRACT

The veterinarian may be faced with a wide assortment of species over the course of their career. Although it is often possible to extrapolate from common companion animal medicine to the more exotic species, one has to be careful to take into account the specific idiosyncrasies of many exotics. It is uncomfortably easy to make a fatal mistake, using drugs or methods commonly employed in dog and cat medicine. This article will aim to point out common pitfalls, enabling you to give a better (and less stress-filled) service to your clients.

RODENTS AND RABBITS

Beware of giving oral penicillin to a rabbit, guinea pig or hamster

Penicillins are often a mainstay in the busy practitioner's arsenal. In most mammals, these antibiotics are associated with very few side effects. However, in rabbits, hamsters and guinea pigs the oral use of these drugs (or any antibiotic with a mainly gram positive spectrum) can lead to a fatal dysbacteriosis within days. Due to their hindgut fermentation these animals have a predominantly gram positive intestinal flora. Drugs such as ampicillin, chlortetracycline and clindamycin will destroy susceptible gram positive organisms, permitting overgrowth of *Clostridium spiroforme, difficile,* or *perfringens* and elaboration of its iota toxin. The end result is a hemorrhagic typhlitis. The effect seems to be worst in animals with hindgut fermentation, and a well-developed cecum, such as rabbits and guinea pigs. It follows that these drugs should also be used with care in other less common small furry pets, such as chinchillas and gerbils (similar to hamsters). For this reason, enrofloxacin (5-10 mg/kg oid-bid) and trimethoprim sulphonamides (30 mg/kg bid) are often the preferred oral antibiotics for use in these species. Remember that these animals are fastidious groomers, and will also ingest topically applied creams and ointments, with a similar end result. As rats and mice have simple digestive systems, one can safely use almost any antibiotic in the latter two species, taking into account the increased doses needed due to their rapid metabolism.

Oral antibiotics associated with enterotoxaemia

Clindamycin	Chlortetracycline
Lincomycin	Ampicillin
Cephalosporins	Amoxicillin
Erythromycin	Amoxicillin-clavulanic acid

Beware of using Fipronil spray on rabbits

Fipronil has been widely and safely used on most exotic species, including snakes. However, Frontline® spray has been associated with several rabbit deaths internationally. The propionyl alcohol carrier is believed to be responsible for the toxic effect, as rabbits are particularly susceptible to this compound. However, Imidacloprid and carbamate can be safely used for ectoparasite control.

Weigh your small patients and work out a proper dose (or at least have a good idea of what they weigh)

It can be very tempting to estimate a 'drop' of medication for the smaller patient, however, this practice can lead to significant overdosing of tiny animals.

Typical weights

	Female	Male
Mouse	20g-60gr	20gr-40gr
Rat	225gr-325gr	250gr-500gr
Hamster (golden)	90gr-120gr	90gr-130gr
Hamster (Russian dwarf)	30gr-45gr	30gr-45gr
Guinea pig	700gr-900gr	900gr-1200gr
Chinchilla	400gr-600gr	400gr-500gr

Take a drug like ivermectin. The dose range stays fairly well conserved amongst species, at 0.2 – 0.6 mg/kg. An Ivomec 1% dose of 0.05ml (1 drop) would be appropriate for a 2 kg animal, but a 5x overdose for a young rat and a 50x overdose for a Russian dwarf hamster.

Know their nutritional and handling requirements
Guinea pigs lack the enzyme needed to convert glucose to ascorbic acid, and are thus incapable of synthesizing endogenous vitamin C. Signs of deficiency (Scurvy) include haemorrhage into the joints and gingiva, loosened teeth, rough hair coat, diarrhoea, delayed wound healing and increased susceptibility to bacterial infections. Guinea pigs require 15-25 mg of vitamin C per day (30mg/day if pregnant). Rabbit pellets are not fortified with vitamin C, and even commercial guinea pig pellets only retain their ascorbic acid levels for 3 months from milling. As such, the diet will need to be supplemented with foods rich in vitamin C (cabbage, kale, kiwi and oranges), or vitamin C should be added to the drinking water daily (200-400 mg/l)**.**

Rabbits on a predominantly pelleted diet develop dental and gastrointestinal problems. Hay plays an important role in wearing down the continuously growing molars, as well as in regulating gastrointestinal motility and function. A common guideline for pet rabbits is that they should not be getting more than a quarter of a cup of rabbit pellets per day – the rest should be good quality teff hay, with no more than a third of the roughage being Lucerne hay.

Always support a rabbit's hind legs when carrying it – they are prone to kicking out and damaging their backs, to the point of permanent hind limb paralysis. This can be attributed to the fact that their skeleton is thinner and lighter than a comparatively sized cats', for example, and the muscles well developed for running.

Why do my small patients die under anaesthesia?
The number one killer of small pets is hypothermia. The smaller the patient, the higher the body surface area relatively to core volume, and the faster heat is lost. Always keep these patients warm, ensure that they do not become hypoglycaemic, and remember – they are prey animals. Rabbits do not need to be starved before anaesthesia – in the normal course of events, they cannot vomit. Very short fasting times are recommended for small mammals, such as rats and mice, as these animals become hypoglycaemic very quickly. Stress and pain can kill – once recovered, these animals need to feel secure (give them somewhere to hide). Also ensure that they have painkillers on board (meloxicam 0.2-0.5mg/kg oid, opioid doses vary according to species). A comfortable patient should be up and eating within a few hours of the procedure.

Should the small patient not begin to eat quickly they should be syringe fed with an appropriate diet. Oxbow Critical Care for Herbivores is the gold standard for rabbits, guinea pigs and chinchillas. Besides the hypoglycaemia the hindgut fermenters can quickly develop ileus which leads to a number of complications.

REPTILES

Do not be tempted to give ivermectin to a tortoise
Ivermectin is a macrocytic lactone which acts on the gamma–aminobutyric acid (GABA) synapses in nematodes, stimulating an excess release of GABA, an inhibitory neurotransmitter. In mammals, ivermectin is generally not able to cross the blood-brain barrier. However, ivermectin can paralyse and kill chelonians, with the leopard tortoise (*Geochelone pardalis*) being particularly susceptible. Signs of intoxication include paresis, paralysis and eventually, respiratory failure. It is postulated that the drug is able to cross the blood brain barrier, or alternatively that GABA is a more important

peripheral neurotransmitter in this group of animals. Skinks and indigo snakes have also been reported to have adverse effects. With supportive care, intubation and ventilation, some animals will survive, but recovery will take more than a week.

Florfenicol has been anecdotally shown to cause fatal hepatopathy in tortoises although it is widely used in other reptiles.

Do not let your reptile patients get cold (and never put them in the fridge!)
Each individual reptile species has an optimal temperature zone at which its immune response will be maximized. Reptiles create a behavioural fever when ill, i.e. they seek out warm spaces in order to increase their body temperature, and hence their immune response. This optimal temperature zone varies between species – when dealing with the rarer species it is important to research relevant husbandry information. As a general rule of thumb tropical species such as the Burmese python need a background temperature of 28°C, with the hottest area in the cage going up to 34°C, while temperate species, such as the corn snake, need to be kept at cooler temperatures (background 24°C, hottest area 28°C). Cooling down nonvenomous reptiles in order to work with them is detrimental to the patient, and does not provide any analgesia.

AVIANS

Cortisone
The use of cortisone in birds is contra-indicated except in a few very specific situations including acute cranial trauma and insect stings etc. When used, the ultra-short-acting intravenous products should be used, and with extreme caution.

Cortisone, even ocular, nebulised and topical products, has been shown to cause severe immunosuppression and hepatopathy in birds.

CONCLUSION:
Although it may seem intimidating to deal with exotic and unusual species, there are numerous texts available including exotic formularies to guide the general practitioner in their care and treatment. Basic principles can be extrapolated from the more common species as long as the particular idiosyncrasies of the species are taken into account.

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RECENT ADVANCES IN AVIAN ORTHOPAEDIC SURGERY

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In treating birds with fractures, the clinician must be prepared to think laterally, considering each case individually, taking into account: compound / closed, proximity to a joint, species temperament (in respect of post-operative care) and life style (in respect of degree of perfection achievable and necessary for an acceptable life).

For many years now, the hybrid or tie in fixator has been the main stay technique in avian orthopaedics, within this and many other facilities.

This paper, will be focused on newer techniques in avian orthopaedics. Recent adjustments to the hybrid-fixator technique, broadening its traditional applications to improve the management of more complex fractures affecting the diaphyses. Elbow and inter-tarsal luxations, splay legs, distraction osteogenesis, stabilisation of cervical fractures, management of bilateral mandibular fractures, mandibular deficits and mandibular mal-alignment will all be discussed.

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BASIC REPTILE HUSBANDRY, MEDICINE AND SURGERY

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ABSTRACT

Reptiles are, on the evolutionary scale, very ancient creatures. They have survived almost unchanged for many millions of years and are without exception supremely adapted to their individual environmental niches. In order to keep and treat reptiles effectively one has to understand one fundamental concept: Reptiles are ectothermic, having no capacity to endogenously regulate their own body temperature. They use outside heat sources eg basking on warm rocks. They are extremely resilient within their Preferred Optimum Temperature Zone (POTZ) but have poor adaptability to varying environmental conditions. Each species has very specific environmental requirements and will only be healthy if kept within those parameters. Common diseases are discussed.

HUSBANDRY

This is the most important aspect of reptile care. Aspects particularly relevant to good reptile care include correct: temperature, humidity, ventilation, lighting, cage design, social grouping, and food.

Temperature

Beware of lights, hot rocks and heating pads that the reptile can come into direct contact with. An animal spending too long on a heating unit, especially if the ambient room temperature is too cold may cause severe thermal burns. The animal should also be provided with a thermal gradient within the cage in order to allow it to regulate its own body temperature within the POTZ.

Humidity and ventilation

Tropical species require extremely high humidity. Unfortunately humidity and ventilation work inversely as increasing the one causes a decrease in the other.

All species need good ventilation. Having air vents around the top of the cage helps very little when both the faeces and the unfortunate inhabitant are restricted to the floor. If ammonia can be smelled in the cage, it is already reaching damaging levels to the sensitive reptile lung (remember, many snakes have only 1).

Cross ventilation is important, with vents on opposite sides and at different heights in the cage, to allow for proper through flow of air.

Lighting

Apart from its use to enhance viewing pleasure, for many reptiles suitable lighting is as important as the correct diet. Herbivorous species and desert-adapted species have high ultraviolet light requirements. Ideally these animals should be housed with constant access to direct (unfiltered i.e. not through glass) sunlight. Animals housed indoors may have their U/V intake supplemented by the proper use of full spectrum (U/V emitting) fluorescent lights. Bear in mind that no light can possibly provide the same intensity of U/V as the sun itself and that most lights lose their U/V emitting capabilities within 6 months. Lights also need to be placed as close to the animal as possible (remember the inverse square law from radiography?). Lastly, you get what you pay for and good U/V lights are very expensive. Sunlight is free! Use it.

Cage design

More sedentary species may be comfortably housed in units of one square meter of floor space per m of body length. Active, nervous species such as American Garter Snakes, Basilisks etc may need much larger units.

Once again, housing suitable for one species may be completely inappropriate for another. Arboreal species prefer tall cages with branches to lie upon. Fossorial (digging) species may only be happy when allowed to burrow into the substrate.

Most reptiles need a hide box of some sort to use as a den. Hatchlings often feel safer if kept in containers with completely opaque sides.

Suitable cages need to be made of impervious materials so as to be easy to clean.

Particulate substrates should be avoided in preference to Astroturf, carpet tiles or paper. Gravel, corn cob and bark chips etc may be ingested leading to intestinal obstructions.

Social grouping
Dependant on species.

Food
Mice and rats are commonly used, as are rabbits, frogs, chickens, fish, crickets, mealworms and greenery.

Encourage owners to either breed their own feeder animals or ensure a reliable source. It is important to realise that what you put in is what you get out. Feeder animals should be fed the best diet possible so as to ensure top quality prey for the reptiles.

Never feed live rodents to snakes. A non-hungry snake will ignore live mice and they can inflict severe, even fatal injury to the snake.

Invertebrate prey should be gut loaded with a suitable formula. Gut loading means feeding the prey insect a diet that will increase its nutritional value (The predator digests the gut contents of the insect as well as its body). High protein and calcium mixtures are often used. Beware of pesticide residues on vegetable food.

BASIC CLINICAL EXAM AND PROCEDURES

Applied anatomy

- Reptiles do not possess a diaphragm, thus their lungs do not collapse on penetration of the coelomic cavity.
- Snakes easily asphyxiate from exudates blocking the long trachea in cases of severe pneumonia (no diaphragm and little ability to cough effectively to expel exudate). Regular suction may be necessary.
- Positive pressure ventilation is recommended for anesthetised reptiles, as many will become apnoeic when on a surgical plane of anaesthesia**.**
- Snakes have extremely elongated organs and most possess only one functional lung.
- Reptiles have a renal portal system whereby the venous blood drained from the hindquarters flows in large part directly through the kidneys. The practical implication being that any drug injected into the hindquarters could either be removed from systemic circulation (if renal metabolism) or could be severely nephrotoxic.
  Thus we always inject reptiles in the cranial third of the body.
- Reptile skin is closed with horizontal mattress sutures in order to evert the wound edges. This pattern allows for tissue on tissue contact. Non-everting sutures allow the skin to curl inwards with scale on scale contact. This will delay wound healing.
- Surgical incisions into the coelomic cavity of reptiles are always performed at a paramedian site. The large ventral abdominal vein lies just below the body wall and is easily traumatised with midline incisions.

Venipuncture

- Ventral tail vein in lizards, approach laterally in iguanids and ventrally in agamids;
- Ventral tail vein or heart in snakes;
- Jugular, dorsal tail vein or subcarapacial sinus in tortoises.

Removing brills
Retained eye caps (fused clear eyelid in snakes) may be gently loosened with fine forceps around the edges and then lifted off. Alternately masking tape may be applied sticky side down and slowly pulled away to loosen the cap. Prior soaking and lubrication aids greatly in removal. Ball Pythons (*Python regius)* have naturally wrinkled eye caps that may be mistaken for retained skin. Never forcibly remove a cap as severe damage to the cornea may occur.

Collecting faeces
Gentle massage and expression of the caudal gut will often deliver sufficient faeces for wet preparations, cultures and smears. If this fails a colonic wash with a few ml of saline may be tried. Lizards will often defaecate when placed in shallow warm water.

Probe sexing
A smooth, blunt ended lubricated probe may be passed into the inverted hemipene in a caudal direction from the lateral side of the cloaca. In male snakes the probe will pass a fair distance into one of the inverted hemipenes, thus travelling a fair distance into the tail region. In females the probe can will stop much sooner. Experience is needed to know the relative probing distances of the various species. Lizards are much more difficult to probe. Monitors may be sexed by the injection of sterile water into the tail base, which causes the hemipene to prolapse. This needs experience. A few species have ossified hemipenes that may be visible on radiograph.

Visual sexual dimorphism
- Many lizards – males have enlarged femoral pores and hemipenal bulges.
- Chameleons – in some species the males have spurs on the hind feet.
- Tortoises – Males tend to have more concave plastrons and longer tails than females.
- Some male snakes have longer tails and slimmer bodies than females. Hemipenal bulges may also be visible.

COMMONLY ENCOUNTERED SPECIES:

➢ *This sign indicates commonly encountered conditions*

Chelonians (Tortoises)
- Ivermectin is TOXIC to TORTOISES!
- Florfenicol has anecdotally been associated with hepatic failure.

Red-Eared Sliders
- Exotic Terrapins (North America)
- Aquatic
- ➢ Salmonella Risk, Hypovitaminosis A, Buoyancy Problems, Shell Rot.

Tortoises
- Numerous species in the pet trade
- Commonly kept as pets
- Diet NB! No Lettuce! No Cabbage! Need grasses, herbs, veggies and fruit. Diet varies from species to species.
- ➢ Shell Trauma (Motor Vehicle Accidents, Dog Bites), Runny Nose Syndrome (Herpes/Mycoplasma), Metabolic Bone Disease, Egg Binding, Vitamin defiencies (esp Vitamin A).

Saurians (Lizards)

Chameleons:
- Very specialised husbandry
- Will only drink dewdrops off leaves, need frequent mist spraying.
- Some have extremely high U/V light requirements.
- Eat live insects.

- Collapsed eye (dehydration), Tongue Paralysis, Fractures and collapse from Metabolic Bone Disease, Trauma, Parasitosis, Renal failure from chronic dehydration.

Monitors:

- Carnivorous
- Can inflict severe bites/scratches and tail whipping. Some reports of mild neurotoxicity of bites.
- Metabolic Bone disease, Obesity, Egg Binding, Parasitosis

Iguanas:

- Very large (up to 6 foot) tropical lizards.
- Vegetarian (proper diet is NB). No Lettuce! No Cabbage! Need large variety of greens with proper Ca:P ratio.
- Very high U/V light and heat requirements
- Enjoy swimming.
- Metabolic Bone Disease, Autotomy, Dysecdysis, Cloacal Prolapse, Parasitosis, Salmonella, Rostral Trauma.

Bearded Dragons

- Desert dwellers
- Omnivorous (insects and greenery)
- Very high U/V light and heat requirements.
- Insect prey should be gut loaded with high Ca.
- Metabolic Bone Disease, Obstipation (from too-large or indigestible prey). Prone to Gut Impaction resulting from Pica if Hypocalcaemic.
- Adenovirus, Yellow Fungus Disease.

Leopard Geckos:

- Small Size.
- Very attractive colour mutations.
- Insectivorous
- Lower U/V requirements.(crepuscular)
- Prone to dysecdysis esp on toes (Provide a humidity area), Metabolic Bone Disease, Internal Parasites.

Tokay Geckos:

- Capable of inflicting SEVERE BITES.
- Can run straight up glass panels (suckers on toes).

Squamata (Snakes)

Of the many species in the trade, there are 2 main groups that will commonly be encountered by the veterinarian.

Temperate Snakes (Colubrids: Rat, Corn, Kingsnakes)

- Excellent pets, docile, moderate size, simple husbandry.
- Mainly oviparous.
- Non-venomous, bites from large individuals may draw a little blood.
- Rodent eaters, young may take skinks
- Summer temp 24-29 degrees
- Winter hibernation NB for Breeding Success, Temp below 15 degrees otherwise snake will not hibernate but will not feed, becoming thin.
- Moderate humidity.

Tropical Snakes (Boids: Boa constrictor, Burmese python, Reticulated python)

- Generally very large.
- Boas ovoviviparous, Pythons oviparous (some will brood the eggs).
- Although non-venomous, can inflict severe wounds with their multiple recurved teeth.
- Most species are mammal eaters, some take chickens etc too.

- Beware of feeding avian prey due to the disease risk (Salmonella).
- Need very high temperatures, 27-35 degrees
- NO HIBERNATION!!!
- Need very high humidity, 70%
- Need good ventilation.

➢ Pneumonia, Dystocia, Rostral Trauma, Rat Bites, Parasitosis, Salmonella, Dysecdysis, Septicaemia, Mouth Rot.

Venomous Snakes
(Copperhead, Western Diamond Back Rattler, Exotic Cobras, White Lipped Tree Viper). Should not be handled unless by experts.

COMMONLY ENCOUNTERED CONDITIONS:

Parasites
Massive variety of internal and external, protozoan and multicellular parasites. Many of these parasites are commensal in the wild state but can nevertheless become responsible for clinical disease in poor, overcrowded or otherwise unsuitable captive conditions.

Ticks are common on tortoises and wild caught lizards.
Mites are common on snakes.
Both can be treated (Extra-label use) with Fipronil. The product should be applied with a cloth in a well-ventilated environment as reptile deaths have been associated with inhalation of the product.
Flagellates are motile protozoan parasites often observed on a fresh faecal wet prep. Although found in healthy animals too, these parasites often seem to multiply enormously in compromised animals and may be responsible for diarrhoea, weight loss, anorexia, dysecdysis or intestinal haemorrhage. Most are Metronidazole responsive. Doses range from 40 to 200mg/kg.
(Anecdotally, Metronidazole may have an appetite stimulating effect in anorexic snakes).
Helminths are generally treated when seen and Fenbendazole is effective for many.

Trauma: shell injuries, autotomy, rostral trauma, rat bites
Excellent regenerative capabilities. Rostral trauma is often seen in stressed; wild caught individuals kept in unsuitable housing. These animals spend hours rubbing along the cage edges looking for an escape route. Tortoises are often presented with horrific shell wounds from road traffic accidents or dog bites. Many lizards are capable of autotomy and these wounds generally heal well. Should the wound be sutured, the tail will not regrow.

Metabolic bone disease:
Extremely common in herbivorous reptiles that are fed incorrectly and are given inadequate exposure to sunlight. Incorrect Ca:P ratios in the diet may also lead to MBD.

Young animals may present with facial deformities, pathological fractures and ileus. Older animals may present with paresis, tremors or seizures. Radiographically the bone density is poor and ionised Ca levels may be low.

Treatment consists of ca supplementation, supportive care and correction of the underlying husbandry problems.

Dystocia and paraphimosis
Surgical correction often needed due to the delayed presentation of these animals. A snake with one hemipene amputated is still capable of breeding so long as the other organ is intact.

Dystocias are often caused by the lack of a suitable nesting site in the cage. Depending on the species, boxes half filled with damp vermiculite, large burrows or warm, dry hides may be appropriate.

Removal of bound eggs and/or foetuses is possible but many owners are now opting for elective ovariohysterectomy in pet lizards.

Pneumonia and septicaemia
Common in tropical species. Generally initiated by a drop in temperature or other husbandry issues. *Pseudomonas/Aeromonas* and *Proteus spp* common. Snakes often present with mucous bubbling from the mouth and nose, wheezing, anorexia and occasionally with signs of septicaemia such as mouth rot and petechiations, “Blister disease” (pink discolouration and vesicle formation on the ventral scales) is typical for septicaemia. Treatment consists of antibiotic therapy according to antibiogram, nebulisation, physiotherapy, supportive care and high ambient temperature.

May develop disorientation and nervous signs characterised by torticollis and opisthotonus, “stargazing”. The prognosis for these patients is guarded to poor.

Dysecdysis, retained eye caps, dry gangrene and dehydration
Can be due to low humidity or disease. Instead of the skin pulling away in one smooth piece, the skin will be adherent to the body and come away in small strips. Frequent soaking will help. Very ill snakes may also shed poorly.

Dry gangrene is seen mainly on tail tips and toes. Ascending bacterial infection that leaves the tip dry and dead. Affected portion should be amputated through healthy tissue (assess for bleeding) and antibiotic treatment given.

Many reptiles will absorb water via the cloaca while soaking but make sure the water is shallow enough that drowning is impossible.

Anorexia, diarrhoea, regurgitation and constipation
Often the result of poor husbandry, endoparasitic or bacterial infections. Initial tube feeding may be necessary to stimulate an anorexic reptile to feed again.

New owners should be encouraged not to over handle snake hatchlings as this may stress them to the point of anorexia.

A canine critical care diet is ideal for tubing carnivorous reptiles and either a slurry of reptile pellets or vegetable baby food can be used for herbivores.

Diarrhoea may be caused by parasites, bacterial infections or toxins.

Regurgitation may be due to low environmental temperature, handling soon after a feed, stress or protozoal diseases such as cryptosporidium.

Cryptosporidium is a tissue cyst forming coccidian like parasite that causes hypertrophy of the gastric mucosa, chronic regurgitation and weight loss. Dx is on stomach flush or gastric wall biopsy. The infection is untreatable and infectious.

Constipation is a common problem in bearded dragons that are fed large invertebrate prey. The chitinous exoskeletons are poorly digestible. Treatment consists of overhydration, oral liquid paraffin, enemas and supportive care.

Obstipation or obstruction may be seen in cases of pica or with accidental ingestion of particulate substrates and may need surgical correction.

Congenital defects
Fairly common. Incubation temperature also has an effect. These individuals should not be used for breeding.

RECOMMENDED READING

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AN APPROACH TO THE ANOREXIC RABBIT

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ABSTRACT

A history of anorexia is one of the most common clinical presentations in the pet rabbit. The domestic rabbit still has the physiology and behaviour of a prey species and will respond to stress or pain with the same instincts as its wild ancestors. Anorexia is not a disease in itself but can be caused by any number of conditions including medical conditions, trauma and simple stress. Discerning the cause of the anorexia requires a detailed history, a thorough physical examination and potentially a variety of diagnostic procedures. Common underlying conditions include dental disease, gastrointestinal disorders, urogenital disease, hepatic disease, renal disease, respiratory disease, severe pododermatitis, arthritis, fractures and ingested toxins. Nutritional support of the rabbit during the diagnostic phase is vitally important to prevent or reverse hepatic lipidosis and to re-establish proper gastrointestinal tract (GIT) motility.[3]

RELEVANT PHYSIOLOGY AND ANATOMY

The rabbit is an obligate herbivore and a hindgut fermenter. The digestive system is adapted for a fibrous diet. Digestion in the stomach and small intestine is similar to that of monogastrics and the remaining food reaching the hindgut consists mainly of fibre (constituents of plant cell walls including cellulose, hemicelluloses and lignin). This fibre can be divided into two portions: fermentable and indigestible fibre. Both are important for proper gastrointestinal function. Excess carbohydrate reaching the caecum predisposes the rabbit to bacterial dysbiosis. This occurs commonly in young rabbits in commercial settings where concentrates are fed ad-lib at the expense of adequate fibre. The rabbits typically present a few weeks after weaning with severe mucoid diarrhoea, collapse and death.

The fibre that passes into the proximal colon is divided into two separate portions. Fibres of a greater length than 0.5cm are directed distally and are excreted as hard faecal pellets. These fibres stimulate healthy gut motility. Smaller particles are directed in a retrograde fashion into the caecum. This phase of colonic motility is named the “hard faecal phase”. The caecum functions as a bacterial fermentation vat and has a complex and delicate microflora. *Bacteroides* spp predominate in a mixed microflora including aerobic and anaerobic bacteria, both gram positive and gram negative. Small numbers of potential pathogens such as *Clostridium* spp may be present but are not harmful unless changes in caecal conditions allow their proliferation.

Volatile fatty acids are synthesised by the caecal microflora. These are absorbed by the rabbit as an energy source. The fermentation in the caecum reduces the fermentable fibre to a soft paste containing amino acids, enzymes, microorganisms and volatile fatty acids.

Following a circadian rhythm, usually in the morning and evening, the motility of the proximal colon reverses direction and the caecal contents are expelled and directed towards the anus. This phase is known as the “soft faecal phase”. The caecal contents are excreted as soft, odorous clumps of material with a thick covering of mucus. These caecotrophs are re-ingested by the rabbit directly from the anus and are further digested in the stomach and small intestine. The mucus coating protects the many beneficial bacteria from destruction in the extremely low pH (1-2) of the stomach.[1]

One of the most common underlying causes of anorexia in the domestic rabbit is dental malocclusion. The rabbit has aradicular hypsodont teeth that grow approximately 2mm per week. Pet rabbits commonly develop dental malocclusion due to genetic factors, inadequate bone mineralisation (due to a calcium deficient diet or inadequate access to UV light) and inadequate wear of the teeth both due to a diet low in fibre. A dental exam is a requirement for any rabbit workup. Sharp spurs commonly develop on the cheek teeth that cause pain on mastication and thus secondary anorexia and ileus.[3]

Rabbit dentistry is a speciality on its own so we will cover it only briefly here. Ad-lib access to high energy foods such as pellets often cause the rabbit to eat insufficient amounts of hay as they preferentially select out the most palatable food.

The rabbit's dentition is designed for a diet of hard grasses and the constantly growing teeth need to be worn down by the grinding mastication of hay.

With inadequate wear, the cheek teeth become overgrown and develop sharp spurs, which can cut the tongue or gums. The roots will also proportionately elongate which can cause exophthalmos, sinusitis and lachrymation. The rabbit will then, in a vicious cycle, be even less likely to eat fibrous foods. Eventually the pain causes anorexia.

Overgrown cheek teeth are corrected by burring them down to a normal flat occlusal plane. Rabbits that have developed this type of malocclusion should have their teeth re-evaluated regularly as repeated management is likely to be necessary.

HOW DO WE IDENTIFY THE ANOREXIC RABBIT?

Healthy rabbits graze almost continuously and will produce copious amounts of hard faecal pellets (up to 180) daily. A history of poor appetite is an early warning for the clinician that further diagnostics are indicated.[3]

Anorexia will result in a reduction of first the volume and then the size of the hard faecal pellets. We often try to offer tempting food to rabbits during the clinical examination. A rabbit that shows interest in the food but then ignores it or mouths and then drops the food is often a rabbit with a painful oral condition. A sick rabbit presents immobile, hunched over and oblivious to its surroundings. The typical rabbit stress response is to "freeze" before escaping and this response (with the associated raised cortisol and catecholamine levels) is a common sequel to pain, illness or fear. Abdominal auscultation may be used to evaluate borborygmus.

Be aware that rabbits can initially sit immobile on the examination table, in the "freeze" response and then jump and kick explosively. A rabbit on the table should always be under control to prevent accidents.

Diarrhoea is also a common presenting complaint. True diarrhoea is a serious condition, no solid faeces are produced and the rabbit is typically very ill. These animals need aggressive therapy in order to survive. Common causes of true diarrhoea include intestinal parasites, sudden diet change and bacterial dysbiosis from incorrect use of antibiotics. Antibiotics including Penicillins (especially if dosed orally), Cepalosporins, Tetracyclines and Clindamycin may commonly cause dysbacteriosis. Enrofloxacin, Trimethoprim Sulphas and Metronidazole are listed among the safer antibiotics.[3]

Owners often mistake uneaten caecotrophs for diarrhoea. These caecotrophs may be found in piles in the cage or may be found tangled in the perineal hair. Soiling of the perineum with also predispose the rabbit to fly strike. Animals with caecotrophic disorders will typically still pass solid faeces intermittently and will be bright, alert and responsive, often with a good appetite. Commonly, a gradual increase of good quality fibre (grass hay) in the diet will encourage caecotropy, improve gut motility, control obesity (which can make it impossible to assume the correct position for caecotroph ingestion) and make the gut flora more resistant to sudden stressors.[1,3]

Abdominal palpation and radiography may reveal a distended stomach with gas surrounding a firm (sometimes palpable) mass. Many normal rabbit stomachs contain hair ingested while grooming. In the past it was thought that gastric trichobezoars were a primary cause of anorexia in the rabbit.[1,3] We now understand that the hard mat of fur and fibre sometimes palpable in the stomach is simply dehydrated normal stomach content and is a sequel to, rather than a precipitating cause of anorexia.[3]

Occasionally a rabbit (especially a long haired breed such as the Angora) will develop a true pyloric obstruction. Both gastric and intestinal obstructions are emergencies and typically present with an acute abdomen and a collapsed rabbit. Gastrointestinal surgery on the rabbit is fraught with complications and is considered a last resort. The stomach and intestines are thin walled and fragile

and dehiscence of surgical incisions is common. Nevertheless, a truly obstructed animal will need aggressive surgical intervention.[3]

HOW DO WE APPROACH THE CASE?

It is imperative to establish the underlying cause of the anorexia. A thorough clinical examination including oral exam, abdominal palpation, faecal examination and potentially the use of other modalities such as radiography and ultrasound is indicated. Basic haematology and serum chemistries should be run.

Anorexia in the rabbit quickly leads to multiple metabolic derangements. Rabbits are unable to vomit and constantly produce saliva. During normal digestion water is also secreted into the stomach and proximal colon. Re-absorption of water occurs in the caecum and distal colon. For this reason any type of intestinal ileus or obstruction rapidly results in dehydration, electrolyte imbalances and distension of the gut with fluid cranial to the site of obstruction. As mentioned previously, anorexia can also result in dehydration of the stomach contents forming a so called “trichobezoar”. Rabbits with a trichobezoar will benefit from oral fluids and potentially from liquid paraffin. It was previously believed that the oral dosing of Pineapple juice helped to dissolve the fibrous mat, due to the proteolytic enzymes in the juice. More recent research indicates that it is likely the extra oral fluid that is making the difference.[3]

Early in the course of the illness rabbits may appear bright and alert but they are predisposed to the development of hepatic lipidosis. During periods of anorexia glucose absorption by the gut falls and there is a decrease in the amount of volatile fatty acids produced by the caecal microflora. This results in hypoglycaemia which stimulates lipolysis as well as the mobilisation of free fatty acids from the adipose tissue. The liver utilises β oxidation to metabolise this energy source and ketone bodies are produced. Rabbits do not have effective metabolic pathways to correct acidosis and are particularly susceptible to the effects of ketoacidosis. Triglycerides accumulate in the hepatocytes, further compounding the problem. Hepatic lipidosis occurs most readily in already obese animals.[3]

Liver failure and death are often the endpoint in the chain of events that begins with anorexia. Hyperglycaemia, disorientation and ataxia followed by profound depression may be seen in this terminal stage of the disease.[3]

HOW DO WE TREAT THE CASE?

Owners should be made aware of the severity and potential fatal outcome of a case of total anorexia and minimal faecal output in a rabbit. These rabbits should be hospitalised for intensive supportive care. Of primary importance is the maintenance of a positive energy balance to prevent excessive lipolysis and hepatic lipidosis. Tasty fresh greens should always be offered to the patient. Good quality grass hay is also needed.[3]

Anorexic rabbits often need assisted feeding in the initial phases of the illness. DO NOT wait too long. Rather begin syringe feeding approximately 10ml/kg of pureed vegetable baby food/soaked complete rabbit diet or Oxbow Critical Care formula for herbivores (most ideal choice) 4-5x daily.[3] Naso-oesophageal tubes may be placed if necessary (8FG works well) but are only used in patients that resist syringe feeding. Most rabbits will need an Elizabethan collar with the N-O tube and the stress of the collar as well as the limitations it places on movement and grazing are often counter-productive. Should we need to use a collar at our clinic, we trim it in such a way that the ears are unhindered and the rabbit has easy access to food. We do this by leaving larger flaps of collar laterally and trimming it shorter dorsally and ventrally.

Although the rabbit may not seem to be losing fluids via vomition or diarrhoea, a rabbit with anorexia and gut stasis should be considered dehydrated. Subcutaneous or intravenous fluids (depending on the level of compromise in the patient) should be administered. Lactated Ringers solution is a good choice.[3] Daily maintenance fluid requirements are approximately 60ml/kg/day.

Analgesics are indicated as gas distension of the inactive bowel causes pain which further compounds the problem. Opioids such as Buprenorphine (0.03mg/kg bid) are regularly used and are

reported to have minimal effects on gut motility. Non Steroidal Anti Inflammatories such as Meloxicam (0.5mg/kg once daily) are also used for pain control.[2]

The use of motility stimulants is a hotly debated topic. Many claim (rightly) that the best stimulator of intestinal motility is long stem unfermentable fibre. Nevertheless, we find that Metaclopramide (0.5mg/kg bid) and Cisapride (0.5mg/kg bid) definitely have a place in our treatment protocol. Naturally prokinetics are contra-indicated in cases of obstruction.[2]
Probiotics have been anecdotally reported to be of benefit in cases of dysbacteriosis. Commercially available probiotics do not contain the normal gut flora of the rabbit but do not seem to be harmful. Caecotrophs can be collected from a healthy "donor" rabbit by placing an elizabethan collar to prevent caecotrophy and can then be fed to the patient to re-colonise the caecum.[2,3]

Anti-ulcerogenics such as Ranitidine (5mg/kg p/o) and Omeprazole are indicated as gastric ulceration may occur rapidly in a stressed rabbit.[2]

It is important to house ill rabbits correctly. They need quiet quarters, away from possible predator species such as dogs. A bed of hay is often useful both as a good fibre source and as a familiar environment. A hiding box or a covered area should be offered. Rabbits naturally seek out dark, small spaces as retreats when they are frightened. A safe, walled off area should be available for supervised grazing outdoors. Anorexic rabbits will often be tempted to take fresh growing grass before any other foodstuffs.

CONCLUSION

An anorexic rabbit should be treated quickly and aggressively to prevent dehydration, hepatic lipidosis, acidosis and death. It is imperative to discover the underlying cause of the anorexia and address this while supportive care is administered. Owners should be made aware of the severity of the condition and should be educated on proper rabbit care and nutrition as many of the predisposing causes are prevented by good husbandry.

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PUBLIC-PRIVATE PARTNERSHIP

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ABSTRACT:

For ages, governments have used a mix of public and private endeavours to deliver services and to manage projects. These cooperative, long or short term arrangements between the public sector and one or more partners in the private sector has led in majority of cases to better management of resources leading to benefits to both parties and to stakeholders. In these arrangements, the risks are equally shared, therefore instilling a sense of improved risk management. As a result of better risk management, the concepts and plans of the projects are better conceived and implementation is more efficient, projects are completed on time, quality assurance is better and there is improved buy- in from the relevant stakeholders.

In the veterinary sector, the implementation of policies is characterised by resource constraints. In addition, profits of the animal and products industries or just their survival can be severely impacted by introduction of new policies and projects. This makes it necessary to share the risks between governments and the stakeholders. The high impact policies may on the other hand lead to greater benefits in the form of improved disease control efforts resulting from improved understanding and buy-in from the stakeholders resulting in setting of common goals, improved health assurances and improved market access of the products produced.

A brief evaluation of the current situation in the veterinary arena in South Africa was conducted by use of a questionnaire and proposals of areas of possible government, private sector and stakeholder collaboration are made for improved delivery of Veterinary Services.

A FRAMEWORK FOR TARGETED ALLOCATION OF RESOURCES FOR LIVESTOCK DISEASE SURVEILLANCE IN SELECTED PACIFIC ISLAND COUNTRIES

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The livestock sector contributes significantly to the food security and economy of the Pacific Island region but the extent of its 25,000 islands' borders and the vastness of its surface area represent a real biosecurity challenge for avoiding the incursions of transboundary animal diseases. Within this context, we conducted a structured approach to identify the diseases of priority in the region and determine how they could be introduced and spread within these islands.

The approach integrated social network and market chain analysis with a risk assessment on a regional level. This appears to be the first study that has applied this approach to a region rather than an individual country. The risk assessment first looked at farmer practices and the movements of pigs and poultry within four selected neighbouring countries using a questionnaire survey and social network analysis to predict how livestock diseases could potentially spread within the region. A participatory pig and poultry market chain analysis was then conducted and combined with a risk pathway analysis to identify the highest risk areas (risk hotspots) and risky practices and behaviours (risk factors) for the introduction and/or spread of foot and mouth and highly pathogenic avian influenza, which were identified as priority regional diseases. The involvement of animal health officials in the market chain analysis with risk pathway assessments formed the risk communication component of the model framework and was a practical way of communicating risk to animal health officials and improving biosecurity. The participatory approach helped officials better understand the trading regulations in place in their country and better evaluate their role as part of the control system.

The logical process developed under this study provides a practical framework that local authorities from the Pacific Island Countries and Territories (PICTs) can follow in the future for a more integrated and better harmonized animal disease risk management. The analysis of our results led to the identification of some limitations and gaps among the PICTs animal health systems and livestock sector that would need to be addressed for an optimal implementation of surveillance programmes. Results provide insights for more rational allocations of available resources and better targeted surveillance programmes and provide a strategy that will underpin food security and enhance biosecurity on a regional basis. The approach could be potentially applied to certain regions of Africa.

CLINICAL PRESENTATION OF CASES IN VETERINARY BEHAVIOUR PRACTICES IN SOUTH AFRICA AND ASSOCIATION WITH BREED

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Data obtained from four veterinary behaviour practices in South Africa was analysed to determine the prevalence of different clinical presentations as well as the association between presenting signs, and breed and gender. A group of dogs and cats presented to veterinary behaviour practitioners in 4 urban practices (the referred group, *n* = 628, consisting of 549 dogs and 79 cats) was compared to a group of dogs and cats obtained from the records of an urban dog and cat boarding facility, (the kennel population, *n* = 6906, 5037 dogs and 1869 cats), assumed to be healthy animals. The most common behaviour problem presented at veterinary behaviour practices was inter-dog aggression involving dogs in the same household (25% of presenting complaints in dogs – refer to Table 1). In cats, the most common problem was inappropriate elimination (house-soiling) with 35% of cat cases presenting with this complaint (Table 2).

The most common first complaints per breed were aggression to unfamiliar people in boerboels; obsessive compulsive disorder in bull terriers, aggression to familiar dogs in Jack Russell terriers, German shepherd dogs and Staffordshire bull terriers; hyperactivity in Labrador retrievers and Rottweilers and house-soiling in Persian and Siamese cats.

The types of complaints in cases with more than one complaint, were also analysed. The number of animals presenting with more than one complaint was 214. Aggression towards human family members and aggression towards unfamiliar humans (7,3% of combined complaints) most commonly presented together when cases presented with more than one problem followed by aggression towards familiar dogs and unfamiliar people (4,3%).

Table 3 represents the comparison between the referred population and the kennel population. Boerboels at 2,1% in the kennel population and 8% in the referred population and German shepherd dogs at respectively 4,4% and 9,3% were over-represented in the population of behaviour cases as compared to the population of healthy dogs. Labrador retrievers (3,3% in the kennel group and 1,9% in the referred group) and dachshunds (7,% and 3,4% respectively), although commonly seen for behaviour problems, were under-represented in the group seen for behaviour problems, when compared to the larger population (Table 4).

Table 1: Breakdown of behaviour problems in dogs

Behaviour problems - dogs	Prevalence
Aggression towards familiar dogs	25%
Aggression towards unfamiliar people	15%
Aggression towards familiar people	12%
Excessive vocalisation	11%
Hyperacitivity	10%
Fears and phobias	10%
Destructive behaviour	6%
Compulsive behaviour	6%
Elimination problems	3%
Aggression towards unfamiliar dogs	2%

Table 2: Breakdown of behaviour problems in cats

Behaviour problems - cats	Percentage
Elimination problems	35%
Intercat aggression in the home	25%
Anxiety, fears and phobias	14%
Intercat aggression involving strange cats	5%
Excessive grooming	4%
Roaming	4%

Table 3: Comparison of kennel population with test population – upper end

Breed	Ken no	Ken %	Ref no	Ref %	Diff
Boerboel	144	2,1%	47	8,0%	-5,9%
German shepherd dog	301	4,4%	55	9,3%	-5,0%
Bull terrier	73	1,1%	26	4,4%	-3,3%
Rottweiler	81	1,2%	26	4,4%	-3,2%
Labrador cross	27	0,4%	15	2,5%	-2,2%

Boerboel cross	21	0,3%	12	2,0%	-1,7%
Cocker spaniel	138	2,0%	19	3,2%	-1,2%
Staffordshire bull terrier	152	2,2%	20	3,4%	-1,2%
Border collie cross	25	0,4%	9	1,5%	-1,2%
Persian cat	3	0,0%	7	1,2%	-1,1%
Bullmastiff	35	0,5%	9	1,5%	-1,0%

Key: Ken = kennel population, Ref = referred population, no = number, Diff = difference between the kennel population and referral population

Table 4: Comparison of kennel population with test population – lower end

Breed	Ken no	Ken %	Ref no	Ref %	Diff
Yorkshire terrier	193	2,8%	13	2,2%	0,6%
Scottish terrier	100	1,4%	5	0,8%	0,6%
Golden retriever	123	1,8%	6	1,0%	0,8%
Labrador retriever	231	3,3%	11	1,9%	1,5%
Dachshund	488	7,1%	20	3,4%	3,7%
Maltese	393	5,7%	11	1,9%	3,8%

Key: Ken = kennel population, Ref = referred population, no = number, Diff = difference between the kennel population and referral population

AN EVALUATION OF SYNDROMIC DATA FROM RURAL POULTRY FARMERS AS A VIABLE DISEASE REPORTING TOOL USING EASTERN ZAMBIA AS A MODEL

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Rural Poultry production possesses a great potential for significant contribution to the attainment of food security in developing countries of the world. Unfortunately, successful production of rural poultry in developing countries like Zambia is hindered by high poultry mortalities which are mostly due to infectious poultry diseases. Furthermore, inadequate financial and human resources make it very difficult for veterinary services to carry out routine active poultry disease surveillance in these regions. As a result, most outbreaks lead to very high mortalities because of delayed response.

Syndromic surveillance is a well described tool used in developed countries for alerting authorities to disease incursions however, little work has been done to evaluate whether this could be a viable tool in countries where disease reporting infrastructure and resources is poor. Consequently, a syndrome based questionnaire study of 459 rural poultry farmers in Eastern Zambia was designed to gather data on previous encounters farmers had had with poultry diseases, as well as control measures they use to mitigate them. The survey took place between October 2014 and January 2015 and its main objective was to determine the viability of this data as an effective means of alerting authorities to disease incursion within rural farming enterprises.

Descriptive and logistic regression analysis were conducted using SPSS version 24, while poultry morbidity and mortality was simulated from the data provided using the Palisade software package @Risk version 7.0. Data provided by farmers, found the overall crude morbidity and mortality in rural poultry for eastern Zambia was 31% and 30% respectively.

Four significantly associated risk factors for poultry morbidity in this region were identified. The study also tentatively identified six poultry diseases from the thirty-four disease syndromes provided by farmers. Furthermore, thirty-six remedies and strategies farmers use to treat and control these diseases were revealed. From these remedies and strategies, only fourteen are conventionally accepted as remedies and disease control measures for poultry diseases.

When compared with the previous method of disease reporting involving morbidity reports submitted by field veterinary technicians, syndromic data apears to be better in identifying disease risks both qualitatively and quantitatively. Thus, it demonstrates that syndromic data obtained directly from farmers could be more beneficial for analysing poultry diseases and their significantly associated risk factors. It therefore, justifies the use of syndromic surveillance, as a cost effective form of targeted surveillance for resource constrained countries like Zambia. Lastly, this study also reveals the use of unconventional remedies for poultry diseases, which may indicate a gap in knowledge of poultry disease control among rural poultry farmers in this region.

A LIVESTOCK FIELD CENSUS CARRIED OUT IN GAUTENG PROVINCE – LESSONS LEARNT, GAUTENG, SOUTH AFRICA, 2016

Geertsma P.J., Govindasamy K.

INTRODUCTION

South Africa used to have a fairly reliable system of collection of animal numbers on our farms which depended on the spatial allocation of a stock inspector and or state veterinarian to a number of farms or to a district. The possible over-emphasis of the regulatory role of the provincial veterinary services may have contributed to emphasis of certain disease control activities to the detriment of effective collection of livestock numbers.

BACKGROUND

Gauteng Province has over the years attempted to develop a credible list of all livestock owners through regular farm visits. This has not yielded desired results and so a definite census was embarked upon as a project that all field staff would assist in. This report is the result of that effort and the data will be used for the next three years in Veterinary Services. The establishment of a credible updated databank of the numbers of animals and their geographical locations in Gauteng Province in support of a best practice veterinary management regarding effective planning, upliftment of resource poor farmers with potential to become commercialised, epidemiological interventions and statistical record-keeping and reporting

METHODOLOGY

The census was run over the months of May to December, 2016. All data was collected from the field with the use of a digitised form, digital pen and cell-phones. This technology allowed the simultaneous collection of the data in the field using the pen and form and a spatial coordinate was added on sending the form by the phone's GPS system.
Most farms were actually visited by the 31 Animal Health Technicians (AHT) in the field. The AHT had a detailed map of the area with farm names, portions and other geographic data to orientate the area. A large percentage of properties were not accessible due to the owners being absent, in such cases AHTs could estimate the numbers of livestock as seen from the road.

A manual was developed indicating definitions and how to conduct the survey. All technical field staff were trained on the form, definitions and completion of the form.

RESULTS/DISCUSSION

Over 12000 field observations were made. Aggregated data will be presented on the census, marketing channels and ethnicity of owners in the various production sectors Key lessons learnt during each of these phases will be presented.

INVESTIGATION OF SEASONAL PREVALENCE OF LOW PATHOGENIC AVIAN INFLUENZA IN A HETEROGENEOUS WILD WATERFOWL POPULATION IN PRETORIA

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Avian Influenza Virus (AIV) is a member of the the *Orthomyxoviridae* group and contains an segmented genome of single-stranded negative sense RNA. It is a highly diverse virus which consists of 16 haemagglutinin (H) and 9 neuraminidase (N) subtypes. The H and N glycoproteins occur in any combination in the viral envelope to form the serotype. AIV has been detected in more than 100 bird species from 26 different families, although waterfowl species are considered to be the reservoir of the low pathogenic form of the virus (LPAI), spreading the virus within and between populations by excretion into the shared environment. These birds are highly mobile, can live under variable densities with multiple exposures to pathogens and have a well-developed immunity to LPAI.

The Irene Country Club in Pretoria houses a variety of free-living wild waterfowl that belong to the *Anseriformes* species such as the Egytian geese, yellow billed duck, red knobbed coot, African sacred ibis and hadeda ibis. These birds would have contact with birds at other sites from other geographic regions across Southern Africa. We investigated the prevalence of AIV in wild ducks at the Irene sampling site over a period of 12 months. A total of 2870 faecal samples were collected and screened for AIV-specific genomic RNA (matrix protein gene), using real time reverse transcriptse PCR (rRT-PCR). Positive samples were inoculated into embryonated chicken eggs for virus isolation. A total of 15 (0.5%) samples tested positive for AIV, and one virus was isolated. The viral isolate was identified as an H3N6 strain using Illumina MiSeq sequencing. The highest frequency of AIV was detected in the months of February and March (late summer), with other peaks in July (winter) and November (early summer)

SEASONAL OCCURRENCE OF *THEILERIA PARVA* INFECTIONS AND CONTROL PRACTICES AMONGST PASTORALIST COMMUNITIES IN MONDULI DISTRICT, NORTHERN TANZANIA

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Abstract:
Background: *Theileria parva* causes an economically devastating tick borne disease which affects cattle in Central, Eastern and Southern Africa. Determination of seasonal infection rates for *T. parva* in wet and dry season is crucial for epidemiological understanding and for strengthening management practices. However, this information is lacking in the study area. The principle objective of this study was to estimate the prevalence of *T. parva* infection in contrasting seasons (wet and dry season) and identify risk factors associated with *T. parva* infections amongst pastoralist cattle of Monduli district, Tanzania.

Methods: A cross sectional study was carried out to determine the prevalence and seroprevalence of *T.parva* in Monduli District. A total of 960 cattle were randomly selected from ten study villages in both wet (April–May 2015) and dry seasons (August-September 2015). Seroprevalence of *T.parva* was assessed using polymorphic immunodominant molecule (PIM) enzyme-linked-immunosorbent assay (ELISA). While *T. parva* current infection was assessed using p104 nested Polymerase Chain Reaction (PCR) in both seasons. *Rhipicephalus appendiculatus* infection rates were examined through DNA extraction and confirmed by nested PCR. Information on animal and relevant farm management practices was gathered using a standardized questionnaire.

Results: The overall prevalence of *T.parva* was found to be 16.2 % and 32.5 % in wet and dry reason respectively. Multivariable logistic models evaluated the odds of PCR positive *T. parva* and the identified predictor risk variables were examined in this study. All cattle were sero-positive while prevalence of *T.parva* was 16.4% and 31.6% in wet and dry seasons respectively. Young animal age, dry season and study village were significant predictors of current infection with *T. parva*.

Conclusion: The results of this study reveal substantial variations of *T. parva* infections in the district. Also the findings suggest that timely and strengthen management interventions are necessary to alleviate more impact of East Coast fever in pastoral communities.

Keywords: *Theileria parva*, cattle, wet and dry season, maasai pastoralists, management practices, Monduli District,

ZOONOTIC EPIDEMIOLOGY OF BOVINE BRUCELLOSIS IN GAUTENG, SOUTH AFRICA, 2016

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INTRODUCTION

Bovine brucellosis is a bacterial zoonotic disease of cattle that is of international public health and economic importance. Gauteng province, in South Africa, has had an increasing trend of bovine brucellosis herd seroprevalence, from 17% in 2009 to 21% in 2013 despite it being targeted for eradication by legislature. The zoonotic impact of brucellosis is unknown in SA, negatively impacting the societal drive to control brucellosis. Evidence of integrated temporal and spatial epidemiological data of interacting cattle and human populations as well as environmental/management risk factors for endemic brucellosis is necessary to base and redefine governmental strategy and policy, for the effective control and management of brucellosis in cattle and people in Gauteng and South Africa.

AIM & OBJECTIVES

In this study we describe brucellosis positive cattle herd data, herd management data as well as data of the farm workers exposed to these herds, for the period 2014 – 2016. The seroprevalence of brucellosis as well as the knowledge and behaviour of these farm workers was determined. Secondly we explore selected risk factors associated with exposure of farm workers to bovine brucellosis.

MATERIAL & METHODS

A cross sectional design was used for a prevalence survey of farm workers in bovine brucellosis positive farms within Gauteng province using a structured questionnaire administered face to face. The farms were selected from farms identified through routine provincial government surveillance from 2014 to 2016. A multidisciplinary team, consisting of a medical doctor, a veterinarian and an animal health technician, conducted the field visit and interviews. The veterinarian administered a herd management questionnaire to the farmer or farm manager, whilst the doctor and assisting technician interviewed the farm workers. The medical doctor collected 5 ml of blood in serum separated tubes from each person and the blood was packaged on ice and delivered to the National Institute for Communicable Diseases that day. The samples were processed using the Elisa IgG (Vircell) serological test. Data from the questionnaires and results of the tests were captured in a relational ACCESS 2010 database and analysed using SPSS. 2 by 2 Univariate analyses to determine Odds Ratios and a Multilevel logistic regression model was used to identify and assess risk factors for seroprevalence in farm workers.

RESULTS & DISCUSSION

214 sample results from 41 farms tested. 41.5% (17/41) farms had at least 1 seropositive person. 11.2% (24/214) farm workers were seropositive on the Elisa IgG. Integrated cattle herd data, herd management data and farm worker data will be presented and described. We will also report of risk factors that were found to be significant in increasing the probability of exposure in farm workers on positive brucellosis farms. The implications of human exposure will be briefly discussed as well as the implications for zoonotic brucellosis surveillance and control in South Africa.

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PREVALENCE STUDY OF *BRUCELLA CANIS* IN PARTS OF THE THEEWATERSKLOOF AND OVERSTRAND MUNICIPALITIES OF THE WESTERN CAPE PROVINCE IN SOUTH AFRICA.

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Brucella canis is a zoonosis of dogs and other canids caused by a gram-negative proteobacterium *B.canis* in the family Brucellacea. Brucellosis in all species is a controlled disease in South-Africa. Many studies around the world have been conducted to determine the prevalence of *B.canis*, but no studies was ever conducted in South-Africa. *Brucella canis* is not considered to be endemic in South-Africa, so the sporadic occurrence of the disease in dogs in the Western and Eastern Cape Provinces of South-Africa prompted a study to determine the prevalence within South Africa.

The aim of this study was to form part of a larger project in the quest to learn more about the prevalence of the disease in South Africa. Thirty two percent of the animals (n128) in the study was sampled at both sterilisation campaigns and welfare kennels. The proportion of samples obtained from veterinary practices contributed 6.75% (n27), and represents the lower risk socio economic group that can afford veterinary health care. Sixty (15%) of the dogs was sampled from 5 different breeders. Dogs from private residences in both informal areas and formal suburbs were sampled during routine Rabies vaccination campaigns held in the study area. The majority of samples in the study was taken at private residences and constituted 172 (43%) of the total number of samples. The samples taken at private residences was divided into urban, peri-urban, Informal and farm categories. Thirteen (3.25%) of the dogs sampled were working dogs from the SAPS K9 unit and Helderstroom prison. Two samples was taken from each participating dog. One serum sample was taken for serologic screening by 2ME-TAT (2–Mercaptoethanol Tube agglutination Test) and then a whole blood sample was stored for confirmatory culture upon reaction of serology tests. All participants were required to complete a survey questionnaire. Owners also needed to sign a consent form where they were informed about and agreed to the study.

Six of the initial 400 samples were haemolysed and could not be interpreted. Eight of the 394 remaining samples tested positive on 2 ME-TAT, resulting in a prevalence of 2,03% on the

serological assay, the prevalence for the different groups were as follows, animal welfares 2, 42% (3 out of 124), Private veterinary practices 3, 70% (1 out of 27) and private residences 2, 33% (4 out of 172). Two of the 5 groups had no positive samples, these were breeders with 0 out of 59 dogs and working dogs with 0 out of 12 dogs. The difference between the three groups was not significant ($p>0.05$). Unfortunately none of the samples positive on serology could be confirmed by bacterial culture.

SPATIAL PLANNING, IMPLEMENTATION, MONITORING AND EVALUATION OF DOG RABIES VACCINATION CAMPAIGNS IN THE BUSHBUCKRIDGE MUNICIPALITY, MPUMALANGA PROVINCE, SOUTH AFRICA

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The Vaccination of domestic dogs against rabies is a regular and highly important activity of the local veterinary services throughout the RSA, especially in areas which experience a high incidence of rabies cases in animals. A vaccination coverage of at least 70% (temporary coverage) of the total resident dog population is targeted to be achieved during such vaccination efforts / campaigns, so as to facilitate the maintenance of a continuous level of protective immunity (valid vaccination coverage) of at least 35- 40%, which is regarded as sufficient to prevent the persistence of rabies in a population, due to the achieved herd immunity.
Differences between different human settlement types, land usage and infrastructure results in associated differences regarding the applied husbandry practices and population demographics of the respective local dog populations.
Measurable units are a basic requirement for the planning, implementation, monitoring and review of animal disease control programmes.
This is especially true for spatially defined animal populations and their respective proportions, which are targeted by animal disease interventions with preventative character, like rabies vaccinations.
Whereas such data is often readily available or comparatively easy to obtain in the livestock farming settings, adequate and current information on the sizes of domestic dog populations, as well as their demographic composition, is mostly lacking, and also prone to local variations and changes over time.
The logistical and strategic planning, as well as effective practical implementation of house-to-house dog rabies vaccinations also does necessitate the presence of clearly defined and easily distinguishable spatial entities.
To address the multiple experienced challenges with rabies control, an approach is now being used by the local veterinary services in the Bushbuckridge municipality in the Northeast of Mpumalanga Province, South Africa, which includes detailed geographical identification of vaccination areas, concomitant dog census and rabies vaccinations, as well as the integration of these information layers, for the efficient planning, implementation, real-time monitoring and analysis of dog rabies vaccination campaigns.

OVERVIEW OF THE PERCEIVED RISK OF TRANSBOUNDARY PIG DISEASES IN SOUTH AFRICA

ABSTRACT

Pig production is one of the most important animal agricultural activities in South Africa, and plays a definite role in providing food security for certain population groups in the country. As with all animal production systems, it is subject to the risk of outbreak of transboundary diseases. In the present overview, evaluations of the perceived risk of selected transboundary animal diseases of pigs, as collated from the willing participants from the provincial veterinary services of South Africa, are presented. A scenario tree revealed that infected but undetected pigs were the greatest perceived threat. The provincial veterinary services, according to participants in the study, face certain difficulties, including the reporting of disease and the flow of disease information amongst farmers. Perceived strengths in surveillance and disease monitoring include the swiftness of sample despatch to the national testing laboratory, as well as the ease of flow of information between the provincial and national agricultural authorities. The four factors were identified that were perceived to most influence animal health-service delivery: transport, access, livestock policy and resources. African swine fever was perceived to be the most important pig disease in South Africa. Because the decentralisation of veterinary services in South Africa was identified as a potential weakness, it is recommended that national and provincial veterinary services need to work together and interdependently to achieve centrally controlled surveillance systems. Regionally-coordinated surveillance activities for certain transboundary diseases were identified as needing priority for the southern African region. It is proposed that an emergency preparedness document be made available and regularly revised according to the potential risks identified on a continuous basis for South Africa.

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SEROPREVALENCE AND RISK FACTORS FOR RIFT VALLEY FEVER IN DOMESTIC RUMINANTS IN THE FREE STATE AND NORTHERN CAPE, 2015-2016

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This study estimated the prevalence of antibodies against Rift Valley fever (RVF) virus in domestic ruminants in an area affected by the South African 2010-2011 outbreaks and identified factors associated with seropositivity.

A cross-sectional study was conducted during 2015-2016 within a ~40,000 km^2 region between Bloemfontein and Kimberley. Farms were selected using random geographic points with probability proportional to density of livestock-owning households; livestock (cattle, sheep and goats) were sampled on the closest farm. A questionnaire was implemented to collect information concerning animal, management, and environmental factors. Sera were screened for RVFV antibodies using IgG ELISA. Data were analyzed using multilevel logistic regression.

On 232 farms, 3,001 animals (956 cattle, 1,525 sheep and 520 goats) were sampled. Estimated RVF seroprevalence, adjusted for clustering and sampling weights, was 30.5% (95%CI: 24.6-37.0%) in cattle, 14.2% (95%CI: 9.7-20.3%) in sheep and 8.8% (95%CI: 4.1-18.1%) in goats. Compared to animals <2y of age, seroprevalence was higher in animals 2-4y (OR=2.1, *P*=0.017) and >4y old (OR=19.7, *P*<0.001). Seroprevalence was also higher on private vs. communal land (OR=6.3, *P*=0.009), on farms that purchased animals in the previous year (OR=1.6, *P*=0.017), and in animals not kraaled at night (OR=2.6, *P*=0.010). Seropositivity was positively associated with the presence of perennial rivers (OR=2.2, *P*=0.004) and seasonal pans (OR=2.0, *P*=0.012) on the farm.

The low seroprevalence likely indicates a largely susceptible population. Seropositivity in animals <4y old, born after the most recent outbreak, raises the possibility that viral circulation occurred during the inter-epidemic period; this requires further investigation.

PROJECTED NUMBERS OF HISTORICAL HUMAN RIFT VALLEY FEVER AND CRIMEAN-CONGO HAEMORRHAGIC FEVER CASES IN SOUTH AFRICA

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Rift Valley fever (RVF) and Crimean-Congo haemorrhagic fever (CCHF) are important vector-borne, zoonotic viruses. In humans, RVF is typically mild with low mortality (1%), while CCHF causes severe haemorrhagic illness with a high case fatality (25%). RVF causes significant livestock morbidity and mortality, while CCHF is inapparent despite high seropositivity. During the 2010-2011 RVF epidemic in South Africa, there were approximately 300 reported laboratory-confirmed cases. Since 1981, 203 cases of CCHF were reported. However, the full public health burden of these diseases is poorly understood.

IgG seroprevalence against the viruses was assessed amongst livestock-owning households (LOHH) and veterinarians during a 4-month cross-sectional study in 2015-2016. The study area encompasses ~40,000 km^2 between Bloemfontein, Free State and Mokala Game Reserve, Northern Cape and includes known RVF epicentres and is within the range of the CCHFV *Hyalomma* tick vector.

Amongst 711 participants from 212 farms, RVFV seroprevalence was 9.6% (CI95%:6.9-12.2). At least one seropositive individual was detected in 25% of LOHH. Of 134 veterinarians, 8.2% (CI95%:6.2-10.2%) were seropositive. Given the participants' age distribution (mean 39y, 60% <40y), most were likely exposed during the 2010-2011 RVF epidemic than before in 1974-1975. Using the seroprevalence and estimated number of agricultural workers (2011 census), we project that the number of past RVFV infections in the study area is 979–2,960. The seroprevalence of CCHFV was 3.2% (CI95%:1.6-4.7%) and the projected number of actual infections is 227–1,140.

These estimates will improve the quantification of the public health burden of RVF and CCHF in South Africa.

THE INTERPRETATION OF LABORATORY DIAGNOSTIC TEST RESULTS FOR DISEASE DIAGNOSIS

Joule-Gaby Kangumba[1]

Laboratory diagnostic tests play a key role in modern animal health management practice and the control of infectious diseases. Diagnostic tests are applied to individuals or populations for the purpose of documenting freedom from disease in a country or region, preventing spread of disease through trade, contributing to eradication of an infection from a region or country, confirming diagnosis of clinical cases, estimating infection prevalence to facilitate risk analysis, identifying infected animals toward implementation of control measures, and classifying animals for herd health or immune status post-vaccination (OIE Terrestrial manual).

The interpretation of diagnostic test results depends on both the ability of the test to distinguish diseased from non-diseased subjects and the particular characteristics of the animal and setting in which the test is being used. This presentation discusses the diagnostic performance characteristics and interpretation of test results of tests that have been validated according to OIE principles of validation of diagnostic assays for infectious diseases. It begins with a summary of how basic criteria of validated tests are determined, with an emphasis on diagnostic sensitivity, diagnostic specificity and predictive values of test results, and then discusses how field workers should understand, interpret and use test results as tools to classify animals that have been tested in the field as diseased or non-diseased. A worked example taking both the screening and confirmatory tests commonly used in the country will be used for illustration.

Keywords: laboratory diagnostic tests, performance characteristics of diagnostic tests and interpretation of test results.

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DETECTION AND DISTRIBUTION OF BOVINE TRYPANOSOMIASIS IN MALAWI

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ABSTRACT

Polymerase chain reaction-restriction length polymorphism (PCR-RFLP) was used to identify trypanosome species in cattle from the three regions of Malawi. A total of 444 DNA samples were screened for trypanosomes using PCR-RFLP performed on whole blood samples collected from cattle between January 2016 and February 2016. A questionnaire was administered to the farmers of sampled cattle that contained sections on demographics and livestock management. Due to its zoonotic importance, *T.brucei* status was chosen to compare data among surveyed farms and Mann-Whitney U tests were employed for these comparisons. Prevalence information and collected questionnaire data were analysed using OpenEpi.

Out of the 440 cattle with DNA samples, 2% (n=9; 95% CI: 1-3) were positive for *Trypanosoma brucei*, 3% (n=14; 95% CI: 1-5) were positive for *Trypanosoma congolense* and 27% (n=120; 95% CI: 23-31) were positive for *Trypanosoma theileri. T.theileri* and *T.congolense* appeared randomly distributed across the country while *T.brucei* was restricted to the central and southern regions of Malawi. The majority of the respondents were farmers (92%; 95% CI: 82-97) that were literate with the median education level being grade 7. Most respondents were smallholder farmers with a median herd size of 7 cattle and owning 1.2 hectares of land. There were no differences between the *T.brucei* positive and *T.brucei* negative groups in respect to education level (p=0.340), cattle owned (p=0.449) and land owned (p=0.920).

MOLECULAR EPIDEMIOLOGY OF BOVINE TRYPANOSOMIASIS AMONGST PASTORALIST CATTLE: A CASE OF MONDULI DISTRICT, NORTHERN TANZANIA

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Abstract:
African trypanosomiasis is a devastating disease affecting both humans and animals in sub-Saharan Africa including Tanzania. Determination of seasonal pattern of this disease in wet and dry season is crucial for epidemiological understanding and for strengthening management practices in pastoral communities. The objective of this study was to estimate the prevalence of animal trypanosomiasis infections in contrasting seasons (wet and dry season) and identify risk factors associated with animal trypanosomiasis infections amongst pastoralist cattle of Monduli district, Tanzania.

In a cross-sectional study design, 960 cattle were randomly selected from ten study villages in both wet (April–May 2015) and dry seasons (August-September 2015). Trypanosomes infection was assessed using PCR, the internal transcribed spacer one (ITS1) gene. Tsetse flies were collected in study villages and Polymerase Chain Reaction was used to detect trypanosomes in the flies. Cattle-level and herd-level data were gathered using a standardized questionnaire.

The overall prevalence of trypanosomes was found to be 5.4 % and 4.2 % in wet and dry reason respectively with the majority of the infections due to *Trypanosoma vivax*. Also mixed infection involving *T. vivax* and *T. congolense* was identified in this study. Breed, age, sex, season, and other potential predictor variables examined were not significant for PCR positivity for trypanosomes.

The results revealed a wide spread of trypanosomes infections in the district despite of low clinical cases which may be due to some outlined potential reasons. Furthermore, this study provides baseline findings that may guide to design a better study to improve understanding of AT occurrence in contrasting seasons as well as tsetse flies infestations in pastoral communities.

THE COORDINATE CONFUSION

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BODY

Geographical coordinates can be recorded in several different formats. In South African Veterinary Services incorrect coordinates are often reported in disease reports due to confusion between the different formats. The official formats used by South African Veterinary Services are Degrees, Minutes and Seconds or Decimal Degrees, depending on the Province. Different provincial veterinary services have different disease reporting systems and formats.

This presentation will explain the different geographical coordinate formats (decimal degrees, degrees minutes seconds and degrees and decimal minutes, how to distinguish between them, their use and common errors experienced when reporting geographical coordinates. Several maps will be used to illustrate these points. Common errors that will be illustrated and discussed are swopping latitude and longitude, writing decimal degrees in the degrees minutes and seconds format and vice versa. This causes coordinates for disease reports to be reported incorrectly and some coordinates indicating the outbreaks falling in the incorrect province, another country or even in the ocean.

PREVALENCE AND RISK FACTORS FOR ANTIMICROBIAL RESISTANT *STAPHYLOCOCCUS AUREUS* ISOLATES FROM POULTRY MEAT PRODUCTS IN SOUTH AFRICA, 2015-2016

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The treatment of drug resistant *S. aureus* infections has become a serious challenge in health care settings and presents a major threat to global public health. Methicillin resistant *S. aureus* (MRSA) strains have been detected in a variety of food products of animal origin in countries throughout the world; however, the mechanisms for transmission of these pathogens to humans are not clearly defined. The aim of the study was to identify and evaluate the risk factors for antimicrobial resistant *S. aureus* isolates from poultry meat samples in South Africa. We investigated the hypothesis that specific factors related to the origin and type of meat products, type of facility, and practices at facilities contribute to the contamination of poultry meat products with antimicrobial resistant *S. aureus*. The *S. aureus* isolates were tested for susceptibility to 14 antimicrobial compounds using the Kirby-Bauer Disc Diffusion method. Risk factors were evaluated using logistic regression analysis of responses to questionnaires. Of the 311 samples tested, 106 (34.1%) were positive for *S. aureus* (95% CI 28.9%- 39.7%). The prevalence of antimicrobial resistance amongst 72 *S. aureus* isolates tested was 55.6% (n=40, 95% CI 43.4%- 67.3%). Out of 72 *S.aureus* isolates, the prevalence of MRSA was 20.8% (15 out of 72 isolates, 95% CI 12.2%- 32.0%). Multi-drug resistant strains were detected in 22.2% (95% CI 13.3%-33.6%) of isolates (16 out of 72 isolates). The associations between the presence of MRSA and origin of the product (p= 0.160), type of meat product (p= 0.962) and type of facility (p= 0.115) were not statistically significant. The presence of MRSA emphasizes the need for further studies to elucidate the possible health hazards for consumers. We recommend that comprehensive antimicrobial resistance surveillance and risk assessment be conducted at all levels of the food chain using a One-Health approach.

PREVALENCE OF *SALMONELLA* SPP. IN SLAUGHTER CATTLE, THE ABATTOIR ENVIRONMENT AND MEAT SOLD AT RETAIL OUTLETS IN GAUTENG PROVINCE

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Abstract

Salmonellosis is an important foodborne disease worldwide. There is a dearth of comprehensive recent information regarding the prevalence of *Salmonella* spp., in beef production in South Africa, primarily due to lack of targeted monitoring of foodborne pathogens. The aim of this study was to determine the prevalence of *Salmonella* spp., in slaughter cattle and environments in red meat abattoirs and meat products sold at retail outlets in Gauteng Province, South Africa. In a cross-sectional study, 517 various types of samples (meat, swabs, water) were collected seasonally from a random selection of 12 abattoirs (n=252) and 31 retail outlets (n=265) between November 2015 to November 2016. The isolation and identification of *Salmonella* spp., were performed using standard microbiological techniques. The prevalence of *Salmonella* spp. was 9.9% (25/252) and 9.8% (26/265) for abattoir and retail outlet samples respectively. In abattoir samples, the frequency of isolation of *Salmonella* spp. was 44% for effluents, 27% for walls and floors; 13% for perineal swabs; 12% for carcass rinsates; 10% for carcass swabs and 10% for faeces. For meat samples collected from retail outlets, the highest frequency of isolation of *Salmonella* spp. was in minced meat (16%) and the lowest in biltong (2%). The frequency of isolation of *Salmonella* spp. was 15% for cold meat, 10% for boerewors and 9% for brisket. Data from the study indicate the extent of contamination by *Salmonella* spp. in the selected abattoirs studied and, more importantly, the risk of salmonellosis posed to consumers of contaminated, improperly cooked meat sold at retail outlets in Gauteng Province.

ANTIMICROBIAL RESISTANCE PROFILES OF *LISTERIA MONOCYTOGENES* ISOLATES FROM RAW MEAT, PROCESSED MEAT PRODUCCTS AND READY TO EAT MEAT PRODUCTS IN SOUTH AFRICA

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Abstract

Listeria monocytogenes is an important foodborne pathogen associated with serious public health and food safety problems. *L. monocytogenes* is usually susceptible to most antimicrobials. However, over the last decade, increasing reports of multidrug-resistant *L. monocytogenes* from various sources have prompted public health concerns. The objective of this study was to determine the antimicrobial resistance of *L. monocytogenes* and the genetic mechanisms that confer resistance. A total of 168 *L. monocytogenes* isolates from raw meat, meat products and ready-to-eat (RTE) meat products were characterized using classical microbiological techniques and confirmed with *L. monocytogenes* MicroSEQ(R) Real-Time PCR. The antimicrobial resistance profiles of the selected isolates were determined by testing against 20 antimicrobials impregnated discs using Kirby Bauer disc diffusion method. Furthermore, the DNA was extracted from to establish the resistant genes, prophages and serotypes profiles. Here we report on the results of biochemical tests, antimicrobial resistance profiles and genetic mechanisms that confer resistance of the 168 isolates of *L. monocyogenes* obtained from meat and by-products samples. The highest resistance was observed against Nalidixic acid (n=168), Colistin sulphate (n=168) and Clindamycin (n=168) for all the isolates included for analysis. Further, 92.86% of the isolates (n=156) found to be resistant to Nitrofurantion, followed by Ceftriaxone 74.40% (n=125), Tetracycline 36.9% (n=62), Trimethoprim 35.71% (n=60), Erythromycin 27.38% (n=46) and 22.02% (n=37) against Ertapenem. The present study has shown high levels of *L. monocytogenes* antimicrobial resistance, which may pose a risk along the food value chain.

Key words: *Listeria monocytogenes* meat and meat products; antimicrobial resistance

FREQUENCY OF OCCURRENCE, AND ANTIMICROBIAL RESISTANCE PATTERNS OF *ESCHERICHIA COLI* O157 AND NON-O157 *E. COLI* ISOLATED FROM MEAT AND MEAT PRODUCTS IN SOUTH AFRICA

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Escherichia coli O157 and non-O157 *E. coli* are among important zoonotic foodborne pathogens due to the severity and complications that are associated with STEC-induced gastroenteritis. There has been a surge in antimicrobial resistance among *E. coli* O157 and non-O157 *E. coli*, which is a cause for concern. The aim of this study was to determine the frequency of occurrence, pulsotypes, antimicrobial resistance patterns and resistance genes among *E. coli* O157 and non-O157 *E. coli* that were recovered from meat and meat products in South Africa and 3 major ports of entry. A cross-sectional study was undertaken to analyse meat and meat products from diverse animal species (n = 2 015) for the presence of *E. coli* O157 and non-O157 *E. coli* using classical microbiological techniques and biochemical tests. In addition, *E. coli* O157 and non-O157 *E. coli* were detected directly from the samples using MicroSEQ(R) Real-Time PCR for STEC and MicroSEQ® *E. coli* O157:H7 Detection Kits. Thirty-four *E. coli* O157 and non-O157 *E. coli* were isolated and confirmed using classical microbiological and molecular techniques. Kirby-Bauer disc diffusion method was used for determination of antimicrobial resistance among the 34 *E. coli* O157 and non-O157 *E. coli* isolates against 10 antibiotics, namely Ampicillin, Amikacin, Amoxicillin-Clavulanic acid, Cefotaxime, Erythromycin, Gentamycin, Nalidixic acid, Oxytetracycline, Spectinomycin, and Sulphamethoxazole/Trimethoprim. The results were interpreted according to Clinical Laboratory Standards Institute guidelines. Real-Time PCR detected more positive samples (n = 74) compared to 34 isolates that could be recovered using culture. Antimicrobial susceptibility testing revealed that 28 of the 34 *E. coli* O157 and non-O157 *E. coli* isolates were resistant to at least one of the 10 tested antimicrobials. The majority of isolates (n = 19) were resistant to Erythromycin, followed by Ampicillin (n = 18), Amoxycillin-Clavulanic acid (n = 15), and Oxytetracycline (n = 12). A considerable number (n = 11) of the *E. coli* O157 and non-O157 *E. coli* were resistant to more than 3 antimicrobials. In conclusion, our findings show that the meat and meat products in some establishments in South Africa are prone to contamination by drug resistant *E. coli* O157 and non-O157 *E. coli.* This is important for highlighting the potential danger of these pathogens to policy makers in order for target-specific multifaceted approaches to be used as hurdles to curb the potential risk of foodborne infections among consumers in South Africa.

PREVALENCE OF BOVINE TUBERCULOSIS IN CATTLE AT THE WILDLIFE/ LIVESTOCK/HUMAN INTERFACE IN NORTHERN KWAZULU-NATAL PROVINCE, SOUTH AFRICA

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Bovine tuberculosis (BTB) is a chronic respiratory disease of cattle caused by *Mycobacterium bovis*.Bovine tuberculosis is a significant disease of livestock in developing countries especially in Africa. Although cattle are known as the primary host, wildlife animals such as the African buffalo tend to serve as maintenance host and this has a potential to spill over to cattle. A cross sectional study was carried out in UMkhanyakude communal area to determine the prevalence of BTB in cattle at the wildlife/livestock/human interface. Whole blood was collected from 380 cattle and a prevalence of 14% was determined using a modified commercial interferon-gamma assay. Additional confirmatory tests are needed to culture and genotype isolates from positive cattle and compare with isolates from wildlife so as to monitor BTB transmission at the interface.

Key words: Prevalence, interface, *Mycobacterium bovis*

COLLECTION AND PACKAGING OF DIAGNOSTIC SAMPLES

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There are several diagnostic modalities at our disposal to obtain a final diagnosis. However, these modalities are very dependent on proper sample collection. The methods differ greatly for the different diagnostic modalities. The presentation will mainly focus on sample collection during a post mortem for various diagnostic modalities and how these samples should be preserved. Special focus will be placed on controlled diseases such as rabies, brucellosis, bovine tuberculosis and which samples as well as how samples should be collected when there are no macropathology lesions.

The packaging of diagnostic samples is as important as the correct diagnostic sample for each animal disease.

All samples should be consider potentially infectious and should be packed accordingly.

All diagnostic samples must be packed in triple layer system and meet relevant Packing Instructions

Mainly: leak-proof primary receptacle, leak-proof secondary receptacle and outer packaging. Submission form must be included and the form should be in a sealed bag to protect from any leakage.

Temperature control should be consider and the inclusion of frozen ice packs is recommended if necessary

Labelling of the package is also important to be delivered to the correct address.

ZOONOTIC DISEASE AWARENESS OF ONE HEALTH STAKEHOLDERS, GAUTENG, 2016

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BACKGROUND

South Africa is a state party of the World Health Organization (WHO). The International Health Regulations (IHR) of the WHO came into force on 15 June 2007. All state parties were required to build core capacity to detect and respond to zoonotic pathogens by 2012. The IHR (2005) stresses the promotion of inter-sectorial cooperation between human and animal health sectors for zoonotic disease detection, prevention and control. Establishing mechanisms for detecting and responding to zoonoses and potential zoonoses is set down as indicator 17 of the IHR (2005) selected for reporting to the World Health Assembly.

A One Health Approach has been adopted in South Africa to meet the requirements of the IHR. One Health is the collaborative effort of multiple health science professions, together with their related disciplines and institutions – working locally, nationally, and globally – to attain optimal health for people, domestic animals, wildlife, plants, and our environment. The South African government plays a crucial role in this process by controlling and managing selected zoonotic diseases of global, and national public health importance.

An essential component of determining the capacity to detect and respond to zoonotic diseases of public health importance is to determine the baseline awareness of stakeholders who work within their disciplines to control these zoonotic diseases.

The aim of this study was to determine the existing critical knowledge of a multidisciplinary group of zoonotic disease stakeholders on 8 selected zoonotic diseases, (Rabies, Brucellosis, Anthrax, Leptospirosis, Tuberculosis, Rift Valley Fever, West Nile Virus and Cysticercosis) and to re-evaluate the knowledge after a presentation of awareness material on the 8 diseases.

METHODOLOGY

One hundred and thirty three stakeholders attended a Zoonotic Awareness day, on the 11 November 2016 in Gauteng province. These stakeholders comprised: emerging farmers (20%), farmers that participated in a zoonotic brucellosis study (5%), animal health technicians (10%), state veterinarians (5%), veterinary public health officials (5%), Gauteng Department of Agriculture and Rural Development and Gauteng Department of Health managers (5%), communications manager, environmental health practitioners (10%), health surveillance officers (5%), human communicable disease control managers (10%), University professors from the Faculty of Veterinary Science and the Faculty of Health Sciences (5%), laboratory managers from human and animal laboratories (4%), private medical doctors (4%),

private veterinarians (4%), students from the veterinary and health faculty and other universities (translation students) and guests from another province.

19 critical knowledge questions, designed by experts of each zoonotic disease, based on the 8 zoonotic diseases, was administered to this cohort through a focus group style of interviewing, using a multiple choice questionnaire, with translation students translating the questions between each question. The questionnaire was administered before a presentation of awareness material on the 8 selected diseases and after the presentation.

RESULTS:

The material and information effectively increased the knowledge of this group from only 35% knowing more than 60% of the critical facts on the pre-questionnaire to 70% knowing more than 60% of the critical facts on the post-questionnaire after the posters and brochures were presented. Results of the knowledge of each disease will be presented including the distribution of knowledge amongst stakeholders.

CONCLUSION:

In order to increase capacity to detect and respond to zoonotic diseases, it is vital to determine the existing awareness of key stakeholders to zoonotic diseases. A multidisciplinary awareness day and awareness material used in conjunction with a pre and post evaluation has proven to be effective in increasing the stakeholder critical knowledge of zoonotic diseases. It is recommended that this method be replicated to increase the capacity of zoonotic disease stakeholders to detect and respond to zoonotic diseases of public health importance.

BRUCELLA MELITENSIS – COMBATTING AN OUTBREAK IN THE FIELD

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Brucella melitensis is a serious zoonosis not often diagnosed in South Africa. Previous cases were only diagnosed when people contracted this disease.

After two people were diagnosed with brucellosis on a farm in Beaufort West in the Western Cape, brucellosis was diagnosed using complement fixation (CFT) in the extensive Boer goat herd on the farm. Subsequently positive CFT results were found in the cattle as well as sheep on this farm. The two seriously ill persons were treated successfully in a George hospital. At the same time, another person that suddenly became lame in Beaufort West was diagnosed as infected with *B. melitensis* using bacterial culture of bone marrow, but no link could be found to the positive farm.

The farm is 8000 ha of Karoo mixed veld and very extensive with a grazing capacity of 36 ha/LSU. Infrastructure such as fences and kraals are very bad, which makes working conditions very difficult. Identification of animals was also not up to standard. The 1100 Boer goats, 500 mixed sheep and 400 mixed breed cattle are farmed extensively and accurate stock figures were impossible to determine. Back tracing to find the origin of this outbreak was done but so far no answers could be found.

Initially a test and slaughter policy was implemented but this was later changed to a total slaughter out policy due to problems described above. In the end all the sheep and goats were slaughtered under official supervision. The cattle were tested and positive animals slaughtered.

After almost two years of testing and many other logistical difficulties the quarantine was lifted with certain conditions. This presentation describes these problems and a possible way to prevent these in the future. A national survey for *B. melitensis* is also of utmost importance for further eradication strategies.

INDIVIDUAL AND HERD SERO-PREVALENCE OF BOVINE BRUCELLOSIS IN IN NORTH WEST PROVINCE 2009-2013

McCrindle, C.M.E.[1]* & Manoto S.[1]

Bovine brucellosis is primarily a reproductive disease characterised by abortion, still births, weak calves, infertility and placentitis in cows; with epididymitis and orchitis in bulls. It is also an important occupational and consumer related zoonosis, causing fever, joint pains, urogenital symptoms and chronic disability. The OIE recommends routine active sero-surveillance for disease control in domestic cattle. In the literature there is often confusion about the actual sero-prevalence of brucellosis in cattle, as different researchers compare individual animal and herd prevalence, often in the same publication. A herd is considered positive if only one positive case is confirmed. Analysis of retrospective sero-surveillance data from 46 762 cows in North West Province showed a significant difference ($p<0.01$) between individual and herd prevalence in beef, dairy and communal farming systems. Over a five year period (2009-2013) the individual prevalence in communal cattle was 5.19%, while herd prevalence was 38.8%. In commercial beef herds individual prevalence was 3.02% while herd prevalence was 32.1%. For dairy cows, individual prevalence was 0.31% with a herd prevalence of 17.9%. It appears from the literature that individual prevalence of brucellosis may be more closely linked to the risk of brucellosis in humans. It is therefore critical to establish whether published research findings refer to individual or herd prevalence when estimating the risk of zoonotic transmission of bovine brucellosis. It was concluded that it may also be advisable to always use individual sero-prevalence when comparing outbreaks in different areas or countries.

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ABSTRACT ON RISK FACTORS FOR BOVINE BRUCELLOSIS IN KWAZULU-NATAL

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Despite the endemnicity of bovine brucellosis in South Africa and its zoonotic impact, little research has been done on its risk factors, control, vaccination levels and the effect of the disease on human health. Recently there has been an increase in bovine brucellosis in KwaZulu-Natal with little difference in commercial farms and those utilizing the dip tanks.

A case control study was conducted investigating risk factors on bovine brucellosis in both commercial and communal farms during the period October 2013 to October 2015. 177 farmers were interviewed, 102 of which were controls and 75 cases. A case farm is defined as a farm where there are two or more serologically positive cattle in herd on confirmatory CFT, but where vaccination was unlikely to be the cause. A control farm was defined as a herd within the same State Veterinary area, with no clinical symptoms, of bovine brucellosis and where all cattle tested negative within 6months of a case herd. The study was conducted in the Northern KwaZulu-Natal. Animal Health Technicians conducted interviews with farmers.

Risk factors were assessed, such as the size of the herd, vaccination status, use of AI or bull, presence of multiple farms by the same farmer, presence of other farm animals in the farm, whether the farm is a communal or commercial farm, government sponsored or self-owned, cattle were bought from neighbouring farm or afar, from auction sales or speculation, calving practise used, any history of abortion and farm management practices used in the farm or dip tank. Univariate analysis and multivariate logistic regression were used to calculate the Odds Ratio (OR) and their 95% confidence intervals.

We will present results from the project which will help us determine the risk factors that are driving bovine brucellosis in farms in KwaZulu-Natal at the commercial and dip tank level. The results will be used to influence the stakeholders and the policy makers to redefine the strategy for the management and control of bovine brucellosis.

SEROLOGICAL ANALYSIS OF *BRUCELLA* SERUM AND MILK SAMPLES WITH IN-HOUSE IELISA CONVERTED ON LUMINEX XMAP TECHNOLOGY

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Brucellosis is a zoonotic disease caused by *Brucella* species that affects livestock and humans. Bovine brucellosis is a serious problem in South Africa and despite it being a state controlled disease; the prevalence is increasing in most provinces primarily because diagnosis of the disease is challenging. This could be due to various factors namely; (i) the disease incubation period varies between animals and in calves, infection can only be detected when they reach maturity, (ii) diagnosis depends on serological surveillance, (iii) cross reaction of serological test with other closely related bacteria, (iv) culturing, the golden standard has low sensitivity and (v) no pathognomonic symptoms in livestock. The main aim of our study was to establish and convert an in-house iELISA for detection of *Brucella* specific antibody in serum to a liquid bead array, using the Luminex xMAP technology platform, to improve diagnostic sensitivity and specificity. The assay was developed using *Brucella abortus* str.99 antigen and protein A/G as the conjugate. Serum and milk samples collected from the cattle farms in Gauteng province were respectively tested with rose Bengal test, commercial iELISA and the milk ring test as well as with the in-house developed iELISA. There was good correlation between the established tests and the in-house iELISA. The conversion of the assay to Luminex xMAP Technology is currently in progress and the recorded data will be used to assist in the validation of the Luminex xMAP assay.

A COMPREHENSIVE NEXT-GENERATION SEQUENCING STRATEGY FOR WHOLE GENOME ANALYSIS OF SAT1 AND SAT2 FOOT-AND-MOUTH DISEASE VIRUSES

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Foot-and-mouth disease (FMD) is a highly contagious viral disease affecting cloven-hoofed animals including cattle, sheep, goats, pigs and antelope. It is an important disease since it is capable of spreading rapidly and extensively within and between countries and animal species, and can have serious economic effects due to direct production losses and indirectly through trade restrictions.

There are little data on the rate and extent of change and adaptation of certain serotypes of FMD virus (FMDV) once they have entered hosts, that affects the efficacy of vaccination. For this reason, it is important to have a reliable method to analyse whole genome sequences of FMDV to gain a better understanding of these changes in the different FMDV serotypes. Despite the fact that next-generation sequencing (NGS) is a relatively new technology, reliable comprehensive strategies for the sequencing of the whole FMDV genome have been developed. This project makes use of the method developed and outlined in Logan *et al.* (2014), implementing a next-generation sequencing strategy for whole genome sequencing of FMDV. This method is capable of being used on all FMDV serotypes, which means that the methodology is applicable worldwide.

In order to test the methodology, NGS was carried out on SAT1 and SAT2 FMDV cattle and goat, mouth and hoof vesicular epithelial samples. All samples used were prepared for NGS, namely through RNA extraction, gDNA depletion, RNA quantification, cDNA synthesis, DNA second-strand synthesis and double-stranded cDNA purification and quantification. The prepared samples then underwent Illumina NGS and the results were analysed using CLC Genomics Workbench computer software. Results were compared between samples collected from cattle and goats. The rate and extent to which genome polymorphisms occurred in SAT1 and SAT2 FMDV were also determined. The methodology was successful in yielding full-genome sequences of FMDV for both serotypes, with the exception of the 5' untranslated region (5' UTR) and 3' poly-A tail. More testing is required to include the 5' UTR and 3' poly-A tail in whole genome sequencing, possibly including Sanger sequencing for these specific areas. This methodology is beneficial for improving our understanding of infection and transmission FMDV, which will in turn aid us in the fight against FMD and limiting the negative effects associated with outbreaks of this disease.

Reference:

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ISOLATION AND WHOLE GENOME ANALYSIS OF A LYTIC BACTERIOPHAGE INFECTED *Bacillus anthracis* ISOLATE FROM PAFURI, SOUTH AFRICA.

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Abstract

Bacillus anthracis is a soil borne, Gram positive endospore forming bacteria and the causative agent of anthrax. It is endemic in Pafuri, Kruger National Park in South Africa. The bacterium is amplified in a wild ungulate host which then becomes a source of infection to the next host upon its death. While outbreaks have been documented in the area for over 30 years, the exact mechanisms involving the onset (index case) and termination of an outbreak are poorly understood due, in part, to a paucity of information about the soil based component of the bacterium's lifecycle. In this study we present an aspect of this in the form of a unique isolation of an environmental dsDNA *Myoviridae* bacteriophage from a *B. anthracis* infected wildebeest carcass. The 154,012 bp aggressively lytic bacteriophage hampered the isolation of *B. anthracis* from samples collected at the carcass site. Whole genome sequencing was employed to determine the relationship between the bacteria isolated on site and the bacteriophage dubbed phage Crookii. The bacterium contained the usual 4 *B. anthracis* prophages described previously, but did not contain any functional temperate phages, although remnants of another bacteriophage was collected from the unamapped reads during sequence analysis. This indicates possible multiple phage infection events of the bacterial strain over time. The isolates also did not demonstrate a trend toward developing phage resistance thus making the replicating bacterium continually available to lysis by phage Crookii. As such, this phage has potential applications in phage therapy and as an environmental disinfection agent. The unusual isolation of this bacteriophage also demonstrates the phage's role in decreasing the inoculum in the environment and impact on the life cycle of *B. anthracis* at a carcass site.

COMPARING IMMUNOGENICITY OF NON-LIVING ANTHRAX VACCINE CANDIDATES IN COMBINATION WITH SIMULTANEOUS ANTIBIOTIC TREATMENT IN GOATS AND USING PASSIVE MOUSE PROTECTION MODEL

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Potential advantages of non-living anthrax vaccines include the simultaneous use of antibiotics with the vaccine for the prophylactic treatment of valuable livestock and endangered wildlife during anthrax outbreaks. However the development and testing of new veterinary vaccines in target animals is increasingly difficult due to ethical and regulatory constraints. These problems are accentuated when testing vaccines against the highly lethal and fulminant anthrax due to high level bio-containment requirements. Little information exists on alternative methods of testing correlates of protection for new vaccine candidates in target animals. In this study we assessed the immunogenicity of subunit vaccine antigens, either in combination with or without antibiotics, and compared these to the live spore Sterne vaccine in goats. We also compared an *in vivo* passive mouse protection model to live target animal challenge to assess the protectivity of anthrax vaccines in goats.

The vaccine antigens comprised of recombinant protective antigen (rPA), spore-specific bacillus collagen-like antigen (rBclA) and formaldehyde inactivated spores (FIS). Vaccine candidates were administered in different combinations to groups of 5 (for *in vivo* mouse challenge) or 10 (for direct lethal challenge) goats. Immunogenicity in the goats was assessed by measuring specific antibody responses to the homologous antigens by ELISA and toxin neutralisation assay (TNA). The protectivity of these vaccines were evaluated in A/J mice after passive transfer of immune goat serum followed by challenge with a lethal dose of Sterne vaccine spores or by challenge of vaccinated goats with a lethal dose of anthrax spores. For the antibiotic treatment experiment two control groups were vaccinated twice with the Sterne live spore vaccine with or without Penicillin G (Pen G).

Goats receiving a combination of rPA, rBclA and FIS yielded the highest ELISA antibody and TNA titres and protected 73% of passively immunized mice and 80% of directly challenged goats 14 days post-challenge. Sera from goats vaccinated with rPA and rBclA alone protected 68% of challenged mice, while 50% of goats survived lethal challenge. In conclusion, the passive mouse protection assay proved to be a reliable correlate for protection and can help to reduce animal numbers in future challenge experiments.

There was no significant difference in the antibody responses between goats that received non-living vaccine candidates with or without Pen G treatment. Furthermore, comparing the immunogenicity between these groups (Pen G treated or untreated) and the twice-Sterne vaccinated

goats revealed equivalent development of titres, after the second vaccination. The control group vaccinated with Sterne vaccine and simultaneously treated with Pen G blocked the development of antibody titres in some of the treated animals. In conclusion, the current data indicate promising potential for further development of non-living anthrax vaccines in ruminants.

CLINICAL EXPRESSION OF AFRICAN HORSE SICKNESS IN SOUTH AFRICAN HORSES.

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African horse sickness (AHS), a disease of equids caused by the AHS virus (AHSV) (*Orbivirus*: Reoviridae), is a concern for equine-linked socio-economic groups, with mortality of up to 90% in afflicted horses. AHS occurs in peracute, subacute, mixed or mild infectious forms. Differential AHSV tropism for cardiac or pulmonary cells is suggested to cause the clinical variation. Clinical signs also vary with virus-dependent (dose, strain virulence, *etc*.) and host-dependent (breed, immune status, age, *etc*.) factors, but symptoms and lesions are thought to be pathognomonic to AHS, with differing mortality rates for each form. Despite the theory of clinically identifiable AHS, there are symptomatically similar viruses that are often mistaken for AHS or *vice versa*, most particularly Equine encephalosis (EE) with a seropositivity ranging between 53-100% in South African horses. Coinfection expression is unknown and a link between external symptomology and mortality or viral load has yet to be determined for AHS.

Suspected clinical cases of AHS were monitored through three outbreak seasons in South Africa. Virus identification and viral load estimation were determined through RT-PCR with serotype determination where possible. Intricate symptom tracking was performed throughout externally visible viraemia until recovery or death. Relationships between symptomology and virus-dependent factors were investigated.

The frequent recording of fever, subcutaneous oedema of the supraorbital fossa and/or head, lassitude and recumbence, inappetance, pulmonary oedema causing discharge from the mouth and/or nose, dyspnoea, and sweating suggest AHS-specific symptomology. Shared relationships were witnessed among symptoms, but clinical case symptomology could not discern orbiviral infection or AHS form. This is further evidence of continual misdiagnosis of orbiviral infection in the country. Symptomology was unsuccessful for the determination of viral load in the absence of further diagnostic tests or autopsy, and did not aid in predicting mortality. A confounding orbivirus, EEV, appears to alter expression of AHS, leading to increased mortality during coinfection. If this virus has such effects, the implementation of a vaccine against it may be necessary to lower mortality during coinfection.

NOTE: This paper has been/will have been submitted for publication, so prior to publication in the proceedings we request that, should it be in press or published, that the reference to the publication be made.

A FIELD INVESTIGATION OF THE AFRICAN HORSE SICKNESS OUTBREAK IN THE CONTROLLED AREA OF SOUTH AFRICA IN 2016

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ABSTRACT

An outbreak of African horse sickness (AHS) type 1 occurred within the South African AHS surveillance zone during April and May 2016 with control measures finally being lifted on 13 June 2016. Twenty-one cases of AHS were detected through both active and passive surveillance. This is relatively few compared to case totals in the same region (and of the same AHS type) in previous outbreaks in 2004, 2011 and 2014. The affected proportion of horses on affected farms was 0.07 (95% CI 0.04, 0.11). Seasonal weather conditions were conducive to high midge activity immediately prior to the outbreak but, with winter approaching, midge numbers decreased rapidly as the outbreak progressed. The spread of cases was localised with 18 occurring within 8 km of the index property. The other three cases occurred on two properties up to 21 km from the index property and wind data showed that these could plausibly have resulted from wind dispersion of infected midges, although secondary seeding of the outbreak in these locations was not evident. Control measures included farm level control using insect repellants and stabling and official implementation of a containment zone with movement restrictions for the duration of the outbreak. Outbreaks in the AHS control zones have a major impact on the direct export of live horses from South Africa to the European Union, its primary market. This outbreak will result in at least another two-year embargo on this form of export. Detailed peer-reviewed reports of field outbreaks assist field and animal health control officials by providing epidemiologic information and support the use of actual field data for AHS and other arboviral risk and disease modelling.

SEROPREVALENCE AND ASSOCIATED RISK FACTORS OF WEST NILE VIRUS IN EQUINE POPULATIONS IN SOUTH AFRICA

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Flaviviruses comprise various species occurring worldwide. Due to the sharing of epitopes within this genus, cross-reactivity amongst flavivirus species has proved its difficulty with regards to diagnostics. Of the mosquito-borne flaviviruses, West Nile virus (WNV) is the most predominant species occurring in horses. The virus is known to cause encephalomyelitis in infected horses. Little has been published on the prevalence of WNV in horses in South Africa (SA), despite several outbreaks. In addition, the risk factors associated with the spread and transmission of the virus has
not been well described in SA horses. For this reason, blood samples were collected from 1219 horses from all nine provinces of SA and tested for antibodies specifically to WNV. The serum neutralizing test was performed on all samples. A selected group of positive and negative samples were then also tested using a capture IgG sandwich ELISA. The serological evidence illustrated that the Free State Province had the highest percentage of positive samples with 78%, whilst the North West Province had the lowest percentile of 42% seropositivity for all tested samples. Overall, there was a total of 57% (700/1220) seropositive samples and 43% (519/1220) seronegative samples, illustrating an above average seropositivity of individuals within the tested equine population used in this study. Mosquitoes were caught using mosquito traps in two of the nine provinces; Mpumalanga and Gauteng. The pooled mosquito species were tested for the presence of WNV using a SYBR green nested real-time Reverse Transciptase-Polymerase Chain Reaction. The potential for cross-reaction between flavivirus epitopes and its effect on the prevalence estimates is discussed in the paper. Each owner of horses participating in the study completed a questionnaire pertaining to the necessary environmental and ecological factors thought to be associated with infection and transmission of WNV. Multiple logistic regressions were conducted to identify significant
associations between the various risk factors and seropositive horses. The significant risk factors associated with seropositive horses were agricultural activities of each region, contact with other horses, the presence of standing water pools, the water source from which horses drink, pest control methods, presence of rodents on properties and whether or not the individuals were stabled or lived outside. The main clinical symptoms significant for infection were fever and stiffness of the limbs
and lower back. The study found there to be a large seropositivity to WNV within SA equine populations, implying large exposure rates and the possibility that a high proportion of cases may be misdiagnosed as a result of asymptomatic presentation in infected individuals.

THE DESIGN AND FIELD IMPLEMENTATION OF A DIGITAL IDENTIFICATION SYSTEM FOR HORSES.

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Horse identification functions to establish ownership, pedigree, vaccination and health status of a horse and to prevent substitution at sales and competitive events. It aids in movement control, the control of infectious diseases and creates traceability. As trade and transport of horses increases internationally, so the need to accurately identify horses becomes more critical.

An identification document (ID) is a reliable and easy method of identifying horses. However, the current paper based identification document system has several limitations. Paper systems are laborious, time consuming, prone to inaccuracies and are not updateable. They do not allow for automatic data capture and do not lend themselves to international standardisation of identification. A digital system would overcome many of these limitations.

Our objective was to design and implement a digital identification system for horses for use by the National Horseracing Authority of Southern Africa (NHA).

An Android app and website were developed. Using this system, sire and dam information are pulled from the database and added to the horse's ID. The diagram and accompanying description of the horse are captured using the app. Descriptive fields are prepopulated to ensure accuracy and aid efficiency. Microchip numbers are imported and DNA sample information linked to the ID. Information captured through this app is automatically uploaded to the database. Once verified by authorised persons, the required output for a passport is generated. This ID can be updated at any time by authorised persons and it has an audit trail that details every change made.

Collected data are stored as objects in the database and are thus searchable. One would therefore be able to search for specific horses based on identification markings or descriptions, something which is currently very difficult.

In future, standardised descriptions would be provided automatically based on the markings drawn on the app. Standardised descriptions would allow the descriptions to be available in multiple languages and thus these horse IDs would be translatable. This standardised description feature would also assist the standardisation of horse identification internationally. Standardised animal identification systems are essential for tracking animals transported internationally.

Alpha and beta testing have shown that the system is simple to use in the field and that data integrity is good. The output fulfils the requirements of the NHA.

In conclusion, this system results in improved efficiency and accuracy of the horse identification process. Real time updates to databases mean improved traceability. While it has been developed for the NHA, the potential exists for it to be used for any equine breed society or registering authority in the world.

SEROPREVALENCE OF LEPTOSPIROSIS IN SLAUGHTER ANIMALS IN GAUTENG PROVINCE ABATTOIRS, SOUTH AFRICA.

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Abstract

Leptospirosis is a re-emerging infectious zoonotic disease caused by the spirochete bacterium *Leptospira* spp., distributed worldwide, especially in developing countries. The disease causes abortion and other reproductive problems in cattle, goats, sheep and pigs. In human cases, the disease presents as a febrile illness similar to malaria, viral hepatitis, influenza, dengue fever and typhoid fever, which can lead to kidney damage, meningitis, liver failure, respiratory distress, and even death. This study determined the seroprevalence of *Leptospira* spp antibodies, using the Microscopic Agglutination Test (MAT) with a panel of eight serovars, on sera samples from slaughtered animals in 10 randomly selected abattoirs within the Gauteng Province in South Africa. Out of the 256 sera samples collected and screened using MAT, 24.2% were seropositive for *Leptospira* antibodies. The serovars detected were Bratislava, Canicola, Icterohaemorrhagiae, Pomona, Tarassova, Swajizak, Grippotyphosa and Hardjo. The data from this study provide an update of the prevalence of leptospirosis and implicated serovars in livestock in the province which may contribute to the prevention and control of leptospirosis in livestock and humans in the country.

Key words: *Seroprevalence, leptospirosis, abattoir and Gauteng.*

RETROSPECTIVE DATA ANALYSIS ON *SALMONELLA* SEROTYPES IN ANIMALS AND ANIMAL PRODUCTS IN SOUTH AFRICA FROM 2007 TO 2014

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Introduction:

Salmonella species are a major cause of salmonellosis in both humans and animals in many countries worldwide. Serotype determination of *Salmonella* species is important for disease assessment, infection control, and epidemiological surveillance.

Aim:

In this review, the laboratory data were collated, analyzed and interpreted to demonstrate the prevalence and distribution of the various *Salmonella* serovars from different host species.

Materials and Methods:

The laboratory record books were reviewed and Salmonella serotyping data from 2007 to 2014 were transferred onto dedicated excel spread sheet. The data were cleaned and analyzed using descriptive statistics. Results were presented in table formats.

Result and Discussion:

In the current study, a total of 1293 cases of animal salmonellosis caused by 94 different serotypes were recorded from 2007-2014 inclusive at Agricultural Research Council-Onderstepoort Veterinary Institute, (ARC-OVI), South Africa. The three most common serotypes were *Salmonella enterica* subspecies *enterica* serovar Heidelberg (n=239), *Salmonella enterica* subspecies *enterica* serovar Enteritidis (n=170) and *Salmonella enterica* subspecies *enterica* serovar Typhimurium (142). These were followed by *Salmonella enterica* subspecies *enterica* serovar Anatum (n=72), *Salmonella enterica* subspecies *enterica* serovar Infantis (n=57). *Salmonella enterica* subspecies enterica serovar Schwarzengrund and Salmonella *enterica* subspecies *enterica* serovar Newport were recovered in 50 and 22 cases each, respectively.

Of the total cases recorded during the period under review, 210 (16.2%) occurred due to host adapted serotypes; viz, *Salmonella enterica* subspecies *enterica* serovar Enteritidis (n=170) and *Salmonella enterica* subspecies *enterica* serovar Dublin (n=40). *Salmonella enterica* subspecies *enterica* serovar Choleraesuis was not isolated. Of the total of 1293 incidents recorded during the period of the survey, 69.8 % (n=903) occurred in poultry and other birds, 13.1 % (n=169) in horses and 10.5 % (n=136) in cattle. Thirty four (2.6%) isolates were obtained from pigs and sheep. Thirty six (2.7 %) isolates were found from wild animals that include leopard (n=6), rhino (n=22) and crocodile (n=8).

Conclusion: Isolation of different *Salmonella* serovars from various host highlights the clinical significance of these bacteria. Moreover, it also highlights the potential zoonotic and health risk implications of the detected *Salmonella* serovars.

MYCOBACTERIUM TUBERCULOSIS INFECTION IN CATTLE FROM THE EASTERN CAPE PROVINCE OF SOUTH AFRICA

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Abstract

Mycobacterium tuberculosis (*M. tuberculosis*) is the main causative agent of tuberculosis in human. In the current study, *M. tuberculosis*, as confirmed by polymerase chain reaction (PCR) using primers targeting several regions of difference (RD4, RD9 and RD12) on the genomes, was isolated from cattle originating from two epidemiologically unrelated farms in the Eastern Cape (E.C) Province of South Africa. Although the isolates were genotyped with variable number to tandem repeat (VNTR) typing, no detailed epidemiological investigation was carried out on the respective farms to unequivocally confirm or link humans as sources of tuberculosis transmission to cattle, a move that would have embraced the 'One Health' concept. In addition, strain comparison with human *M. tuberculosis* in the database from the E.C Province and other provinces in the country did not reveal any match. The VNTR profiles identified in the current study will be included in both the veterinary and human genotyping databases to serve as references for future epidemiological studies. Our findings however, call for urgent reinforcement of collaborative efforts between the veterinary and the public health services of the country.

Keywords: *Mycobacterium tuberculosis*; cattle (*Bos taurus*); genotyping; zoonosis

PREVALENCE, SEROTYPES AND VIRULENCE CHARACTERISTICS OF Shiga toxin-producing Escherichia coli (STEC) FROM COW-CALF OPERATIONS IN THE GAUTENG AND NORTHWEST PROVINCES OF SOUTH AFRICA.

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Shiga toxin-producing *Escherichia coli* (STEC) are foodborne pathogens that are characterised by mild to severe diarrhea, hemorrhagic colitis (HC) and may be complicated by the hemolytic uremic syndrome (HUS) in humans. Ruminants, especially cattle are recognized as the main reservoir for STEC. The main STEC virulence factors are bacteriophages-encoded shiga toxins: (*stx1* and *stx2*), intimin (*eaeA*) which is responsible for the intestinal attaching and effacing lesions that are typical in STEC infections and the plasmid-encoded hemolysin (*hlyA*). The aim of this study was to determine the prevalence of STEC, serotypes and virulence profiles of associated with cattle STEC. Faecal samples were collected from 610 adult cattle on 6 cow-calf operations in Gauteng and Northwest Provinces. STEC were detected by culture and multiplex polymerase chain reaction (mPCR) that targeted *stx1, stx2*, *eae* and *hlyA*. In addition, 179 STEC were serotyped (O:H) and 100 isolates were virulotyped. The prevalence of STEC was 40% (245/610). The 179 STEC isolates were mostly non-O157 STEC which belonged to 40 O:H serotypes represented by 28 O serogroups and 12 H types. Virulotyping of 100 STEC isolates revealed the following frequencies for STEC virulence-associated genes and markers: *stx1*, 79%; *stx2*, 73%; *eae* 11%; *hlyA*,76%. More than half of the isolates (22/40) were serotypes that have been previously associated with human disease. Most of the isolates were *stx1, stx2 and hlyA* positive but lacked the *eaeA* gene. The 11 *eaeA* positive isolates possessed the *stx1, eae, hlyA* genotype except for two isolates that were *stx1, stx2* and *eaeA* and *hlyA* positive. In summary, STEC prevalence was high compared to the wide range of previously reported prevalences in cattle worldwide including Africa. The isolated STEC serotypes were mainly non-O157 STEC that have been previously implicated in human disease worldwide including South Africa. The isolates were mostly *stx1*, s*tx2* and *hlyA* positive but *eae*-negative. STEC that are associated with human disease in South Africa are mainly *stx1, eaeA, hlyA* positive while only a few human isolates carry the *stx1, stx2 eaeA* genotype. Possession of the *stx2* and *eaeA* is a common feature of STEC that are usually associated with severe disease in humans including HC and HUS while those that are *stx1, eae, hlyA* are frequently associated with mild disease. In conclusion, this study confirms that South African cattle are an important reservoir of STEC and a potential source of STEC for humans.

Sero-prevalence of *Brucella* spp. in slaughter animals in Gauteng Province abattoirs and assessment of risk factors posed to abattoir workers, using a One Health approach to educate as an intervention tool the prevention of zoonotic infection.

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Brucellosis is one of the neglected zoonotic diseases,a highly infectious and contagious zoonotic disease of humans, and a wide range of domestic animals, especially in the ruminants.To date, there are limited recent empirical data or published reports on the prevalence of brucellosis in the livestock industry in South Africa. Globally, abattoirs are used for passive and active surveillance of diseases of both economic and public health significance. Surveys by serological assays of slaughter animals may be used to detect newly introduced disease agents and in monitoring disease control and eradication programmes. This research goal was to determine the prevalence of *Brucella* spp. in slaughter livestock in abattoirs in Gauteng Province, and to assess risk factors that predispose abattoir workers to zoonotic infections such as brucellosis, and to use a 'One Health'multidisciplinary approach to educate the abattoirs workers on how to reduce the risk of contracting zoonoses while working at the abattoir.The Rose Bengal test (RBT) and indirect Enzyme Linked Immunosorbent Assay (*i*ELISA)were used to determine the seroprevalence of brucellosis in slaughter cattle and polymerase chain reaction (PCR) to detect *Brucella* DNA in lymphoid tissues of seropositive animals. Eleven consenting abattoirs were visited and 174 head of cattle were sampled comprising 81 females and 93 males. The seroprevalence of brucellosis was 11.5% (20 of 174) and 4.5% (8 of 174) by the RBT and *i*ELISA respectively, with 40% (8 of 20) agreement in seropositive animals between both tests. PCR detected *Brucella* DNA in the lymphoid tissues of 7 (87.5%) of 8 *i*ELISA-seropositive cattle. The fact that *Brucella*-positive cattle originated from 6 of the 11 abattoirs sampled indicates a zoonotic risk to abattoir workers at these facilities. Of the 100 abattoir workers interviewed, 79% were males and 21% were females. Thirty-seven (37.0%) believed that they cannot contract zoonoses from working in the abattoir. However, 91% (21% females, 70% males) of all workers interviewed had cut their hands at least once while performing their duties at the abattoirs,and 88% of the workers indicated that they do not seek medical attention whenever they experience symptoms of illness. A 'One Health' approach was used to produce educational materials such as brochures, mugs and posters, and these were used to sensitize the abattoir workers.

PREVALENCE AND CHARACTERIZATION OF SHIGA TOXIN-PRODUCING *ESCHERICHIA COLI* IN BEEF CARCASSES AND BEEF PRODUCTS IN GAUTENG

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Shiga toxin-producing *Escherichia coli* (STEC), particularly the O157 strains, are food-borne zoonotic pathogens. Foods of cattle origin have been implicated in various outbreaks and epidemiological studies have revealed that cattle are major reservoirs of STEC. We conducted a cross-sectional survey from Nov 2015 to Nov 2016, to investigate the prevalence and molecular characteristics of O157 and non-O157 strains of STEC in beef and beef products in the Gauteng province of South Africa.

A total of 265 swab samples of beef carcasses from 12 abattoirs and 399 beef products from 31 retail outlets were screened for STEC using a multiplex PCR. The overall prevalence in abattoir samples was 37% (55/149) in summer and 34% (39/116) in winter. In beef products it was 20% (27/137) in autumn, 14% (18/130) in winter and 17% (22/132) in summer; the highest prevalence was detected in boerewors (35%) followed by mincemeat (21%). The predominant serotypes detected were O113 (19.4%) and O157 (14.9%) in beef products, and O113 (14%) from abattoirs.

Our results demonstrate that STEC is present in South African beef and beef products. This may pose a real food-borne disease threat; further investigation of the epidemiology of the pathogen is required.

DEVELOPMENT OF REAL TIME PCR ASSAYS TO IMPROVE THE ACCURACY OF BOVINE AND PORCINE CYSTICERCOSIS DIAGNOSIS

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Cysticercosis is a parasitic disease caused by metacestode stages of the human tapeworms, *Taenia saginata* and *T. solium*. Consumer protection from bovine and porcine cysticercosis relies on meat inspection, although this method has been questioned in terms of accuracy and efficiency. Meat inspection may result in misidentification due to morphological similarities between taeniid larvae, other tissue parasites and occasionally neoplastic or non-infectious lesions. The aim of the study was therefore to develop real-time PCR (qPCR) assays for diagnosis of the metacestode stages of the two *Taenia* species and to evaluate their detection efficiency in comparison to meat inspection and conventional PCR. Mitochondrial *cox* 1 gene-specific primers and probes were designed from the gene of each species. *Taenia saginata* and *T. solium* control DNA samples were ten - fold serially diluted in triplicates to determine the sensitivity of the assays and DNA extracted from closely related *Taenia* species were tested to determine specificity of each assay. “Metacestode” stages of both *Taenia saginata* (n=71) and *T. solium* (n=2) were collected from positive carcasses in abattoirs and used as field samples. The standard curves generated and linear regression calculations made showed that both qPCR assays were successfully optimised. The *T. saginata* and *T. solium* assays showed detection limits of 0.013 ng/µl and 0.0034 ng/µl, respectively and both of them specifically amplified their target gene. The qPCR assays confirmed 63% (n = 71) *T. saginata* and 100% (n = 2) *T. solium* cysticerci respectively. On the other hand, the conventional PCR confirmed all of the visually identified cysticerci. Analytical sensitivity of the newly developed *T. saginata* qPCR was 37% lower than that of conventional PCR and this could be due to factors including DNA degradation and PCR inhibitory factors. Further experiments are therefore recommended to improve on the analytical sensitivity of this assay. Meat inspection records in South Africa show that cysticercosis, especially porcine cysticercosis is frequently observed at very low levels, hence only two *T. solium* cysticerci were collected.

AFRICAN SWINE FEVER VIRUS MAINTENANCE AND TRANSMISSION DYNAMICS IN THE SYLVATIC *ORNITHODOROS* VECTOR

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African swine fever (ASF) is a highly contagious and fatal haemorrhagic viral disease of domestic pigs caused by a large, DNA arbovirus with a genome ranging from 170 to 190 kbp in length, the African swine fever virus (ASFV). There is no treatment or vaccine available to combat the disease, and sporadic outbreaks of ASF have been reported from 1928 until present from within the Republic of South Africa (RSA). Control of the disease in the RSA relies on strict biosecurity measures and the establishment of a control zone that limits movement of pigs and pig products from high-risk areas. The sylvatic cycle, that involves warthogs and *Ornithodoros* soft ticks, plays a crucial role in the maintenance and distribution of ASFV and clarification of key epidemiological factors are needed in order to enhance understanding and to assist with the formulation of more effective disease control strategies. A comprehensive survey to confirm the presence of *Ornithodoros* ticks in game parks within the control zone in SA as well as those in neighbouring Swaziland was done to determine the presence of the soft ticks and their ASFV infection status. Characterisation of the 16S rRNA gene sequences of *Ornithodoros* ticks from each of the sampling sites revealed high levels of diversity and confirmed the presence of at least three geographically distinct lineages within SA. In an attempt to better understand how ASFV adapts and changes when it cycles between the invertebrate and vertebrate host, a transmission experiment in which naturally infected *Ornithodoros* ticks were used to establish an infection in domestic pigs was conducted. This study gives insight into the role that the sylvatic *Ornithodoros* vector plays in African swine fever virus maintenance and transmission dynamics in South Africa.

EVALUATION OF AFRICAN HORSE SICKNESS CASES TO *CULICOIDES* NUMBERS AND CLIMATIC VARIABLES

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African horse sickness (AHS) is a devastating vector-borne viral disease affecting all equines. The World Organisation for Animal Health (OIE) lists AHS as a notifiable disease and as such, it is a controlled disease in South Africa that must by law be reported to the state veterinary services. Cases are reported annually across South Africa with sporadic cycles of outbreaks being recorded. African horse sickness information i.e. reported outbreaks, cases and deaths was extracted from the website Department of Agriculture, Forestry and Fisheries (DAFF). These statistics were compared to the annual average *Culicoides* numbers collected per province and graphically presented. The average number of cases, average number of *Culicoides* and various climatic variables per month in Gauteng were also compared. Graphs were generated. The *Culicoides* numbers as determined with light traps increased during spring and summer, peak during March, and decreased steadily from April onwards until the next spring when it increased again. African horse sickness cases reflected the same general pattern as the *Culicoides* numbers. The best correlation was found between high numbers of midges to cases of AHS and rainfall.

AFRICAN SWINE FEVER OUTBREAK IN SOUTH AFRICA, 2016

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Domestic pigs infected with African swine fever (ASF), are a source of the disease and spreads the virus to other pigs during active outbreaks. During the acute phase of the disease, clinical symptoms like nasal discharge, diarrhoea, and reddening of the skin may occur. Infected pigs usually succumb within a week of the development of clinical symptoms. In South Africa, as in most other countries, ASF is a controlled disease and therefore the state veterinary authorities must be informed. In South Africa, ASF is usually restricted to four provinces in the north-eastern parts of the country. The ASF control zone, demarcated in accordance with the Animal Disease Act 35 of 1984, includes most of the Limpopo Province and parts of the North West Province, Mpumalanga and Kwazulu-Natal. The first outbreak outside the control area in more than 50 years occurred in 2012 affecting pork producers in Mpumalanga and Gauteng. The outbreak was associated with the movement of infected animals across provincial boundaries. This was followed by an unrelated outbreak of ASF in the North West and Free State provinces in 2016, highlighting the continuous risk of spreading ASF beyond the control zone. We have used DNA sequencing to characterise the virus isolates recovered from infected pigs, comparing it to contemporary and historical isolates from southern Africa. The ASF virus was characterised as genotype I.

SALIVARY GLAND TRANSCRIPTOME OF *RHIPICEPHALUS (BOOPHILUS) MICROPLUS*

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The cattle tick, *Rhipicephalus (Boophilus) microplus* is of veterinary and medical importance globally. Control of ticks is important and needed to prevent livestock diseases caused by tick-transmitted pathogens. Tick control measures have relied on the use of acaricides, however, this has several disadvantages such as development of acaricide-resistant ticks, environmental pollution and milk and meat contamination. Therefore, there is a need for alternative methods, and vaccines directed against tick feeding are considered as the best option in an integrated pest control strategy. This study aimed at characterizing and analysing the salivary gland transcriptome and proteome of *R. (B.) microplus.* Female ticks were collected at five different feeding stages. cDNA libraries were prepared from the RNA of female ticks from all feeding stages. The libraries were then sequenced by Illumina MiSeq platform. Proteomics experiments were also performed, where the salivary gland proteins were subjected to 1D SDS-PAGE; tryptic in-gel digestion and analyzed on the MS/MS mass spectrometry analysis. The transcriptome and proteome data were analyzed by CLC Genomics Workbench; Trinity and Minia and Mascot and X-Tandem databases, respectively. The results obtained herein indicated the presence of major secretory protein families such as Kunitz, lipocalins, serpins, cement proteins and metalloproteases, while the majority of transcripts coded for housekeeping genes. The correlation between the transcriptome and proteome was weak, especially for the housekeeping proteins. The outcomes of this study provided a deeper understanding and better insight about *R. (B.) microplus*. However, proteins identified in this study still need to be characterized and tested for antigenicity.

VETERINARY PROFESSIONALS AND ANIMAL RESEARCH

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Vivisection, is a word, that when used causes conflicting emotions and countless debates among people from all walks of life. It is quite understandable for people to not completely understand or grasp the importance of animal research, but as veterinary professionals, who owe their entire careers to this field of science, we have a moral and ethical responsibility to at least have a basic understanding of when, where and how animal research has enabled us to perform our jobs. Animal research has evolved from the 1st millennium and has vastly improved the way we live. Laboratory animal science is a challenging and ever changing field of study, it is the reason we as veterinary professionals know how to treat and prevent diseases in animals, or how we know the anatomy of an animal. It has considerable medical advancement implications for both man and animals. Up until recently it has been a field of study and work by a very few individuals (in South Africa). The South African Association for Laboratory Animal Science (SAALAS) with the assistance of other educational organisations and individuals, is trying to revive the interest in laboratory animal science as the need for qualified professionals is growing, be it veterinarians, nurses, animal health technicians or veterinary technologists. The word vivisection should make a veterinary professional stand up proud, as this field encompasses caring, compassionate individuals who are at heart dedicated to improving medical research while still meeting animal welfare requirements.

AFRICAN HORSE SICKNESS VIRUS EVOLUTIONARY DYNAMICS

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African horse sickness virus (AHSV) is a member of *Orbivirus* genus within the *Reoviridae* family. Nine antigenically distinct serotypes of AHSV (AHSV 1-9) have been identified. The genome of AHSV consists of 10 double stranded RNA segments and are replicated by a RNA-dependent-RNA polymerase which lacks proof-reading abilities and are therefore prone to mutations. The virus could potentially undergo genetic recombination and reassortment during co-infection of two serotypes in the same host cell. African horse sickness virus is an arthropod-borne virus transmitted by *Culicoides* biting midges. It is therefore proposed that the virus would evolve slower than other RNA viruses, due to constraints imposed by the ability to successfully replicate in both insect vector and vertebrate host. The evolutionary dynamic of AHSV over the period of more than 60 years was investigated. The effect of selection, substitution rate and recombination over time were determined. African horse sickness viruses isolates from the 1960's to 2014 housed at the OIE World Reference Centre for AHSV and BTV at the ARC-OVI were propagated in BHK-21 cells and dsRNA extracted. cDNA synthesis were performed using previously published methods and the DNA was submitted for Illumina Next Generation Sequencing. The complete genomes were assembled, deposited on GenBank and used in subsequent bioinformatics analysis. These included phylogenetic analysis, determining the substitution rates, selection pressure, intragenic recombination and reassortment. All ten segments were predicted to be under purifying selection pressure ($d_N/d_S < 1$), while four of the segments included selected sites under positive selection pressure (0.1 significance level). The Bayesian coalescent estimates of mean substitution rates for segments-2, -6, -7, and -9 were the highest of the AHSV genome segments, between 1.75 $x10^{-4}$ and 2.8 x 10^{-4} substitutions per site per year. These substitutions rates were similar to that (0.52 x 10^{-4} and 6.9 x 10^{-4} substitutions per site per year) observed in BTV. Estimated substitution rates ranged between 1.5 – 6.4 $x10^{-4}$ substitutions per site per year over the whole genome. Using RDP4, Bootscan and Simplot programs, intragenic recombination events were predicted in seg-1, seg-6, seg-7 and seg-10. These included both single and double cross-over events. Widespread reassortment events were detected, including the vaccine strains with field isolate. Similar to BTV, all ten segments were predicted to evolve under strong purifying selection with selected sites under positive selection. The high percentage sequence identity within serotypes reflects the strong selective constrain imposed on arboviruses by the necessity to replicate in both host and vector species. This study provides the first evidence of intragenic recombination in African horse sickness virus, though it was less abundant than in bluetongue virus. The study reported on widespread genetic reassortment, including between wild-type and vaccine viruses. This was the first study to investigate the evolutionary dynamics of nine AHSV serotypes collected over decades.

ANTIMICROBIAL ACTIVITY OF SILVER, NANO SILVER AND ANTIBIOTICS ON SELECTED MASTITIS CAUSING ORGANISMS.

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Antibiotic resistant microorganisms are becoming increasingly resistant to conventional drugs posing a health problem to animals and humans. There is a need to explore non antibiotic therapy as an alternative treatment. Nano structured antimicrobials and Silver nitrate have shown inhibitory effects against multi-resistant microorganisms known to be hard to destroy using conventional methods. The aim of this study was to evaluate the antimicrobial activity of antibiotics, bulk silver and Nano silver on mastitis causing organisms. The antimicrobial activity of three individual antibiotics, Silver nitrate and Nano silver against field strains of *Staphylococcus aureus* and *Streptococcus uberis* was compared. The Micro broth dilution and Kirby-Bauer method was used to test the inhibitory effects of antimicrobials. Isolates of 20 S*taphylococcus aureus* and 20 *Streptococcus uberis* were collected from milk samples from two farms in KwaZulu-Natal. All were subjected to different concentrations of Silver nitrate and Nano silver ranging 1.25µg to 20µg. Samples were read and recorded. Isolates were further subjected to the Kirby-Bauer method. Results were observed and recorded. Nano silver showed significant inhibition of *Streptococcus uberis* but not on *Staphylococcus aureus*. Silver nitrate showed inhibition of *Streptococcus uberis* at 20µg/ml and on *Staphylococcus aureus*. On the Kirby Bauer method *Staphylococcus aureus* isolates were 100% were susceptible to penicillin, 100% had an intermediate effect on erythromycin and 60% were resistant and 40% intermediate to streptomycin. *Streptococcus uberis* 40% susceptible, 60% resistant to penicillin, 80% were resistant and 20% intermediate for streptomycin, 90% were intermediate and 10% susceptible. With Nano silver and silver nitrate small zones of inhibition were observed.

VACCINATION OF ON-FARM CATTLE AGAINST HEARTWATER USING AN ATTENUATED TISSUE CULTURE VACCINE

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Heartwater (Cowdriosis) is an infectious, non-contagious, tick-borne disease of both domestic and wild ruminants caused by a rickettsia, previously known as *Cowdria ruminantium* but has recently been reclassified as *Ehrlichia ruminantium*. There is approximately 150 million animals that are at risk in the sub-Saharan Africa, with South Africa contributing 8.6 million. The disease affects mainly cattle, sheep, goats and some wild ruminants. The vectors responsible for the transmission of the agent belong to the genus *Amblyomma*, the most important two vectors being *Amblyomma hebraeum* and *A. variegatum*. Currently, the control methods of heartwater in the country is through acaricide application and vaccination. Both these control mechanisms are unsatisfactory, expensive and requires excessive effort. The only commercially available method of immunization is “infection and treatment”. Even though this procedure has been the only commercially available “vaccine” for many years, the spectrum of protection of the Ball 3 blood vaccine strain against other *E. ruminantium* strain is limited. Problems encountered with this procedure; blood should be kept at a temperature below freezing until before use and this is a serious challenge for rural areas, it requires intravenous administration, temperature monitoring, consequently trained staff is needed and the vaccine does not offer cross protection against many South African field strains. Therefore, there is a need for a vaccine that will offer a wider protection to heterologous challenge, replace the intravenous with intramuscular administration.

PRESENCE AND DISTRIBUTION OF *LISTERIA MONOCYTOGENES* IN SOUTH AFRICAN MEAT AND MEAT PRODUCTS.

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Listeriosis is an important foodborne disease worldwide. There is a scarcity of comprehensive recent information regarding the prevalence of *Listeria monocytogenes* in meat and meat products in South Africa, primarily due to lack of targeted monitoring of foodborne pathogens. The aim of this study was to determine the presence of *L. monocytogenes* in meat samples collected across nine provinces of South Africa. In a cross-sectional study, 2 013 various types of meat samples were collected from a random selection of abattoirs, meat processing plants and retail outlets between 2014 to 2016. The isolation and identification of *L. monocytogenes* was performed using standard microbiological techniques and Real Time-PCR. The prevalence of *L. monocytogenes* was 56, 4%, 38.8% and 4.8% for retail outlet, meat processing plant and abattoir samples respectively. In retail outlet samples, the frequency of isolation of *L. monocytogenes* was for 59.0% for processed meat, 25.5% for raw meat and 14.4% for Ready-To-Eat (RTE) meat products. For meat samples collected from meat processing plant, the highest frequency of isolation of *L. monocytogenes* was in processed meat (68.0%) and the lowest in RTE (5%). Geographical results revealed that samples from Gauteng (34%), North West (14.4%) and Mpumalanga (14.4%) provinces had the highest presence of *L. monocytogenes*, while Eastern Cape had the lowest presence of 2.1%. Data generated from the study indicate the extent of meat contamination by *L. monocytogenes* in South Africa and, more importantly, the risk of listeriosis to consumers of contaminated, improperly cooked meat sold at various outlets in South Africa.

DIAGNOSTIC TESTING AT PVVD, ARC-OVI

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Diagnostics form an integral part of disease surveillance and control. At the Agricultural Research Council – Onderstepoort Veterinary Institute (ARC-OVI) diagnostic tests done in the Parasites, Vectors and Vector-borne Diseases programme (PVVD) include tests of economically important parasitic diseases. Tests done include theilerioris, babesiosis, Leishmania, and Trypanosomiasis, These diseases affect a wide range of animals, cause severe losses in animals and revenue. With international trade and animal movement ever increasing, the risk of importation of disease exists. Specific tests require certain samples. Samples needed include serum, blood smears and EDTA collected blood. Most of the tests done at the laboratories at PVVD that test for babesiosis, Theileria and Leishmania is DAFF approved and SANAS accredited.

SEROTYPE-SPECIFIC RT-PCR AND SEQUENCING FOR DISCRIMINATING BETWEEN VACCINE AND FIELD AFRICAN HORSE SICKNESS VIRUSES

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African horse sickness virus (AHSV) is a *Culicoides* transmitted virus of the genus *Orbivirus.* It causes a non-contagious eponymous infection affecting equid species. The disease is endemic to southern Africa, where all nine serotypes have been described. The outer capsid protein VP2, encoded by the most variable genome segment 2 (Seg-2), is the primary target for AHSV specific neutralising antibodies and subsequently determines the virus serotype. Annual vaccination with homologous serotypes can protect horses. Commercial vaccine used in South Africa consists of polyvalent mixture of attenuated live viruses (ALV). The AHS vaccine produced by Onderstepoort Biological Product (OBP) Ltd., contains seven of the nine ASHV serotypes formulated as a trivalent and tetravalent application. A disadvantage of the vaccine is that it is not possible to discriminate between vaccinated and naturally infected animals. This problem is exacerbated by the possible reassortment and reversion to virulence following vaccine-associated infection. The purpose of this study was to design a serotype-specific PCR assay capable of distinguishing between vaccine and field isolates. Genome comparisons of more than 100 AHSV were performed and regions unique to each specific serotype were identified in segment-2. These sequences included the nine reference viruses, the seven ALVs and field viruses isolated from the 1980's to 2014. Single Nucleotide Polymorphisms (SNPs) were identified within these unique regions capable of distinguishing between the ALV / reference virus and field isolates of each individual serotype. Nine unique sets of primers were designed, each amplifying a unique serotype. The amplicons ranged from 222 to 458bp. The serotype of a virus was assigned based on the presence of an amplicon in PCR using the corresponding set of primers. In addition, the assay could be used to distinguish between natural and vaccine-associated infection based on the sequence of the amplicons. Six of the serotype specific amplicons had between 7 and 14 SNPs, discriminating between ALV and field isolates. An additional set of primers was designed to differentiate between field viruses and the ALV of serotype 7, since the latter contains a 670bp deletion in segment-2. The performance of the serotyping assay was assessed using 100 diagnostic samples not used during the design thereof. The PCR can be used as a fast, cost effective method to assign field viruses to a specific serotype. Since sequence analysis of the PCR product discriminate between ALV and field isolates, the assay could potentially be used to identify vaccine-associated infections of AHS.

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VETERINARY PHYSIOTHERAPY: REVIEW OF THE SCIENCE BEHIND THE MODALITIES USED

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ABSTRACT

Veterinary Physiotherapy is evidence based scientific profession. Majority of the modalities currently being used was adapted from the medical field. These modalities include manual therapy, hydrotherapy, electrotherapy, rehabilitation exercises and others. Research specific to the application of these modalities in the animal field is slowly emerging.

INTRODUCTION

Veterinary Physiotherapy for the surgical patient aims to speed up healing, alleviate pain, speed up recovery, empower owners as part of the rehabilitation team, recognise and prevent complications, prevent re-injury and to ensure optimal recovery to return to highest functional level. General protocols will be explored.

These are the main modalities utilised in treatment interventions:

- Manual Therapy – mobilisations, massage, stretching
- Electrotherapy
  - Therapeutic Laser
  - Therapeutic Ultrasound
  - Electric currents – EMS + TENS + FARADIC
  - Pulsed magnetic field
  - Shock wave therapy
- ICE + HEAT
- Rehabilitation exercises
- Hydrotherapy/UWT

MANUAL THERAPY

Manual therapy refers to the practice of passive or active assisted movement techniques applied by the therapist to address pain and impairments in the articular, neural and muscular systems (Goff and Jull, 2007) Includes Mobilisations, Manipulations, Stretching and different types of Massage.

Manual therapy Influences by

- LOCAL TISSUE
  - Physical and mechanical properties
    - Lengthen, elasticity, Aligning new fibers
  - Fluid dynamics
    - Blood, lymph, extracellular fluid
  - Repair process
    - Removal of metabolic waste
    - Influx of nutrients
- Neuro Physiological
  - Pain gate
  - Removal of irritants
- Neurological
  - CNS
  - Proprioception

Biomechanically it may effect in the following ways

- Joint displacement due to hysteresis (Herzog et al (1999)
- Vertebral mobilisation and manipulation increased dorsoventral segmental mobility in horses (Haussler et al (2007)

- Manipulation of equine T/L region produced small increases in segmental ROM (Gomez-Alvarez et al (2008)
- Massage (petrissage and effleurage) was applied to the caudal hind limb muscles in eight horses and resulted in a significant increase in both passive and active hind limb protraction (Hill et al, 2010)
- Dynamic stretching was proposed to have more effect on the tendon, than static stretching, increasing both the storage and release capability of the tendon (Mahieu et al 2007)

Neurophysiologically manual therapy has the following effects:

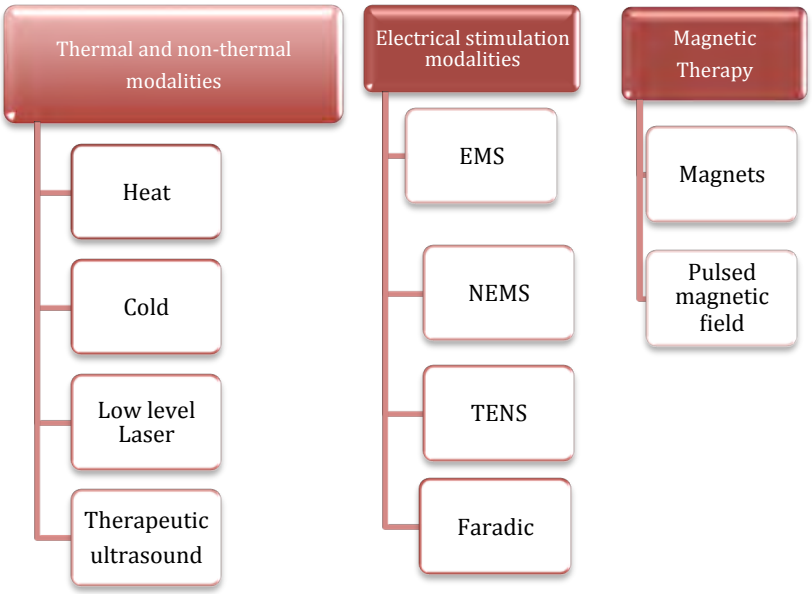
- Afferent input from specific locally induced movement may stimulate neural inhibition
    - At various spinal cord levels
    - At higher centres of nervous system (Christian et al (1998) Wright et al (1999)
- Passive manual therapy to the spine activates descending inhibitory pathways from the lateral periaqueductal grey of the midbrain.
    - This provides non-opioid mediated hypoalgesia Wright et al (1995), Vicenzino et al (1998)
    - Manual therapy effects are both hypoalgesic and sympathoexcitatory (Paungmaliet al (2003)a Paungmali et al (2003)b)

Manual therapy has a reparative effect by:

- Influencing reparative and healing processes within the neuromuscular system
    - Haussler, K (2009) Review of Manual Therapy Techniques in Equine Practice, JEVS, 29 (12) 849-869
    - Goff, L (2009) Manual Therapy for the Horse – A Contemporary Perspective, JEVS, 29 (11) 799-808 outlines the current evidence for the beneficial effects of manual therapy and its application to horses

All forms of manual therapy have reported levels of effectiveness for treating musculoskeletal issues in humans, but mostly only anecdotal evidence exists for animals – mostly horses. (Haussler (2009) High velocity, low amplitude thrust that induces therapeutic effects in articular structures, muscle function and neurological reflexes (Haussler (2009) Manually applied chiropractic type forces produce measurable spinal segmental motion (Haussler et al (1999) Chiropractic techniques can increase passive spinal mobility (Gomez-Alvarez et al (2008)

ELECTROTHERAPY



Therapeutic ultrasound

- Pressure wave/mechanical wave

- Sound wave is produced by a transducer
- Ultrasound turns electric energy into mechanical energy – via reversed piezoelectric effect
    - Certain crystals placed in electric field it changes crystal shape…this distortion causes compression and separation of molecules which is then propagated as longitudinal waves
    - The distortion of crystals causes the plate to vibrate at the same frequency and this distortion is then propagated into the next medium – gel….
    - Pulsed or continuous will give a Thermal effect or a Non-thermal effect
    - 1MHz – 2- 5cm penetration
    - Longer wave length, poorer absorption, need to use higher intensity
    - 3MHz – 0.5 – 2 cm penetration
    - Shorter wave length, better absorption, lower intensities as wave don't travel that far, better absorbed at superficial site

Pulsed magnetic fields

- Mechanism of action
        - Diseased/damaged cells have altered rest potentials (permeability to Na+ and K+)
        - Rest potential of cell is proportional to ion exchange occurring at cell membrane
        - Ions are affected by rhythm of pulsation when introduced to PEMF
        - Ion exchange is responsible for O2 utilization of cell
        - Lack of O2 utilization is a problem with delayed healing and arthritic joints
        - Enhance cartilage repair
        - Stimulating chondrocytes
        - Increase in collagen synthesis
        - Increase in osteogenesis
    - Some research suggest:

      Cartilage:
        - 3 times per month, 6hr /day, 75Hz, 1.6 mT
        - Increase cartilage thickness
        - 75 Hz, 1.5 mT, 90 days, 6hours/day = reduced NSAID needed

      Bone:
        - 15 Hz, 3hr/day, 7 day/week, 10 weeks
        - Reduced osteotomy gap size
        - 100Hz, PW 25usec = increased bone contact

      Tendon:
        - 30 min/day, 27.12 MHz
            - 69% increase n tensile strength
        - 17 Hz, 15 min, 5 mT
            - Increase physiological alignment of collagen fibres
        - 15 Hz, 12 mW, 8 h/day
            - Increase tensile strength and increase peritendinous adhesions

      Nerves:
        - 2Hz, 0,3 mT, 4h/day during 1-5 day post injury
                - Increase functional recover

      Spinal cord:
            - Might help motor function recovery and lesions volume size after acute spinal cord injury.

CRYOTHERAPY

- Transfer of Thermal energy
    - Between environment and body surface
    - Between tissues and fluids of body
- Increased metabolic activity
    - Van't Hoff's Law – any chemical reaction that can be increased will increase temperature (metabolic >13% with every 1°C)
- Increase in capillary permeability

- Heat - > metabolism>accumulation of metabolites>increase acidity>dilation of arterioles>decrease hydrostatic pressure in capillaries> Increase flow of fluid through capillaries into tissue

TENS

- The treatment of pain through electrical stimulation is accomplished through the following four ways:
  - Pain Gate Theory (Melzak and Wall 1965)
    - Selective stimulation of larger diameter fibres in peripheral nerves, which in turns helps to block nociceptive activity in smaller, afferents at segmental level
    - TENS works better as it has higher frequency with longer pulse durations
  - Descending inhibitory mechanisms (Baxter & Barlas 2002)
    - Release of endogenous opiate-like (endorphins) substances in response to higher intensity levels at a lower frequency.
  - Localised blocking of peripheral nerve fibres
  - Decrease of tone in tight muscles
  - Increase in blood flow

EMS

- Parameters
  - 100,200,300 micro seconds (humans say 100 best)
  - Waveform – less NB, no research
  - Amplitude – as high as comfortable
  - On/off – 1:2 – with function, 1:1 if not
  - Ramp – 2-4 suggested, 1 use
  - Polarity – black (-, cathode) red (+,anode)
  - Time – 7-10 – 30 min
- Frequency
  - Tetanic for strengthening
    - 30-60 Hz recommended, 5 Hz used
    - 20Hz – prevent atrophy of slow-twitch
    - 30 Hz – prevent atrophy of fast twitch
    - 2Hz & 10 Hz – remediation of disuse atrophy
- Myofascial Tender Points
  - TENS
    - 60 Hz, 20 min, strong but no muscle contraction
  - EMS
    - 10Hz, visible contractions, 20 min
    - TENS more effective for immediate pain relief of MTP than EMS
    - EMS better effect immediate release of muscle tightness
- More effective with active exercise
- For strength gain – no added benefit
- Lots of research for Ems plus exercise
- Fracture healing
  - 3cm prox to fracture, 2nd electrode proximal to first
  - 4Hz, 50 microsec, 20 sec on, 15 sec off, 5 sec ramp
  - 1hr per day, start day 4 after Sx X 25 days
  - Increase mineralized callus
  - Bone greater torsional parameters for stiffneess, max torque withstand & required more energy to failure

LASER THERAPY

Different class lasers available. Anything with an average power output of less than 1 watt will not be sufficient in treating chronic and deeper musculoskeletal problems. Therapeutic lasers will have different wavelengths and its best to have a laser with more than one wavelength setting. As different wavelengths will be absorbed in different cells. It is also important to understand that if a deeper or more chronic problem needs to be treated with a laser it wont be sufficient to use a less powerful laser

for longer. Even though the mathematics will suggest it will produce the same amount of Joules into area, it is the average power output that will ensure proper uptake in cells.

Latest research also suggests that a pulsed setting produces better results than a continuous setting.

Classification:

- Class1 : 0-0.4 mW (laser, printers, CD)
- Class2: 0.4-1mW (Pointer)
- Class 3 A: 1-5 mW (firearm sights, pointers, therapy)
- Class 3B: 5 -500mW (therapy, light shows)
- Class 4:anything bigger than 500mW(Surgery, cutting, therapy)

Interactions can be phototermal, photodisruptive or photochemical.

Effects start at cellular level:
Increased ATP and protein synthesis resulting in increased growth factor response
Accelerate/stimulate cell reproduction and growth leading to faster repair of damaged tissues
Reduces inflammation and reduces pain by reducing prostanglandin E

Anti-inflammatory effects:

- Cellular membranes stabilised by increasing ROS influencing the Sodium potassium pump and calcium uptake.
- Superoxide Dismutase production will moderate the free radical activity
- Histamine/ Serotonin/NO release will lead to vasodilation which decreases ischemia and increases oxygen and nutrient influx required for cell repair and removal of cellular debris
- Increases angiogenesis-capillary and lymphatic
- Increase in leukocyte activity – macrophage clean up
- Increase in prostaglandin synthesis (PGI2)-anti –inflammatory
- Decrease of interleuking 1-a – which is a pro-inflammatory component
- Decrease amount and duration of inflammation

Wound healing:

- Increased circulation
- Increased ATP production
- Increased DNA synthesis, protein synthesis, growth factor
- Increased cell proliferation and maturation
- Increased collagen production
- Reduction of scar tissue formation
- Faster wound closure
- Increase phagocytosis
- Increase granulations tissue
- Increase epithelisation

Research:

- Ten daily sessions of 1,200 J and 12 W of laser therapy (wavelength of 1,084, 810 and 980 nm) added to a flow of cold air at -30 °C (Archilles Tendon)
    - Gave quicker and better pain relief. It also gave the patient a full functional recovery and greater satisfaction.
        - Notarnicola A et al 2014
- Laser therapy iLux-Triax® and tecar therapy Pharon® in the treatment of low-back pain, with or without leg pain, can significantly reduce pain and improve the quality of life in patients with degenerative and inflammatory problems.
    - Osti et al 2015
- 15 treatment sessions with the experimental protocol (2600J) suggested greater effectiveness of Laser than of US therapy in the treatment of LBP, proposing HILT as a promising new therapeutic option into the rehabilitation of LBP.
    - Fiore et al 2011

VETERINARY PHYSIOTHERAPY: CARDIORESPIRATORY AND ICU PATIENTS

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ABSTRACT

Physiotherapy interventions for cardiorespiratory patients in the medical field are instrumental in the recovery of the patient. This field is slowly growing within the veterinary profession.

INTRODUCTION

Cardiorespiratory physiotherapy is very big field within human medicine. There is lots of research making it an evidence-based practise. It is however still a growing field in the animal industry with big potential.

The main areas:

- Obstructive pulmonary disease
- Restrictive pulmonary disease
- ICU patients
  - Ventilators
- Cardiac patients
  - Many of the evidence-based techniques used in human care, are built on studies performed on animals, a number of them on dogs. (Gee & Williams 1979, Zidulka et al 1989, Tomkiewics et al 1995)
- Typical patients
  - Pneumonia (bacterial, viral (interstitial), fungal)
  - Upper respiratory tract obstruction (ex during anaesthetic recovery in brachiocephalic dogs)
  - Aspiration pneumonia (tick poisoning, mega-oesophagus, myasthenia gravis, after snakebite or other poisoning, laryngeal paralysis)
  - Pulmonary oedema from cardiac disease (left-sided heart failure)

GOALS

- Mobilisation and removal of airway secretion/mucus
- Manual techniques
- Postural draining
- Non-invasive ventilation
- Breathing techniques (Challenging in canine patients)
- Respiratory muscle strengthening
- Improve pulmonary function
- Mobilisation

MANUAL TECHNIQUES

Localize the mucus -Auscultation
”Loosen the mucus up and move it forward”

- Percussions
- Vibrations
- Shaking

Above mentioned techniques are done in postural draining positions.

Postural drainage

- Postural drainage positions for dog has been well described (Manning et al 1997)
- Clinically, the sicker the dog, the more cooperative in being positioned and greater importance

- Positions may need to be modified to ensure patients compliance or because of concomitant pathologies
- Saturation and heart rate should be monitored.

Postural drainage positions

Areas of lungs to be drained	Postural drainage position
Lateral segment of left caudal lung lobe	Left lateral recumbancy with the hind end elevated 40°
Left and right caudal dorsal lung fields	Sternal recumbancy with hind end elevated 40°
Left and right caudal ventral lung fields	Dorsal recumbancy with hind end elevated 40°
Left and right cranial ventral lung fields	Dorsal recumbancy with front end elevated 40°
Left and right cranial dorsal lung fields	Sternal recumbancy with front end elevated 40°
Right middle lung lobe	Dorsal recumbancy. Pillow placed under the right side of the thorax. Hind end elevated 40°, front end rotated one quarter turn to the right
Lateral segment of the right caudal lung lobe	Left lateral recumbancy with hind end elevated 40°

Non-invasive positive pressure ventilation

- Mechanical ventilation that does not include intubating the trachea
  - Common treatments (of humans) with pulmonary oedema, hypoxia and/or hypercapnia.
- Several varieties;
  - Positive end expiratory pressure (PEEP)
  - Non-invasive intermittent positive pressure ventilation (NIPPV)
  - Positive expiratory pressure mask (PEP)
  - Continuous positive airway pressure (CPAP)
  - Bi-level positive airway pressure (BiPAP)
  - Nasal inspiratory pressure support ventilation (NIPS)
- They all work a little bit different but basically; The patient breaths against a positive pressure which keeps the alveoli open.
- Improves saturation and assists with mucus loosening

Studies show that:

- PEEP improves lymphatic drainage and increases lung volume in dogs with pulmonary oedema
- PEEP reduces hypoxia as previous unventilated alveoli are re-expanded. Also in dogs.
- PEEP is more effective at reducing extravascular lung water when applied immediately when pulmonary oedema occurs
- NIPPV, CPAP and PEP reduces intubation rates, length of time from weaning from mechanical ventilation, complication frequency and in-hospitality mortality rates of humans with hypercapnic respiratory failure.
- PaO2 has been shown to improve within the first hour of CPAP

- Non-invasive ventilation reduces respiratory muscle fatigue

Breathing techniques

- Expiration pressure – rib springing
- Exercise-based
- Breathing assist techniques" developed to improve saturation by reducing respiratory rate and encourage more effective inspiration and lung inflation
- Perioral stimulation and intercostal stretching shows significant improvements in oxygen saturation
- Intercostal stretching slows the expiratory rate of dyspnoeic dogs (although difficult to perform on small dogs with respiratory rates in the vicinity of 60 breaths per minute)

ICU

- Prevention of pneumonia and other respiratory complications
- Prevention of neuro-musculoskeletal complications
  - Loss of ROM
  - Atrophy
  - Pressure sores
- Mobilising patients to be discharged from ICU when ready.
- CPAP has been used in dogs with respiratory signs including pulmonary oedema, hypoxia and hypercapnia.
  - Biggest challenge? To achieve an air-tight seal without causing the animal undue stress
  - Anaesthetic masks achieve sufficient seal but blow the positive pressure directly into the dogs face and constrict the nasal region
  - Transparent hoods better that blows the positive pressure over the top of the dogs head from behind has been better tolerated, but is harder to achieve an airtight seal.
  - The effective delivery of CPAP to non-intubated dog is still on the trial phase.

CARDIAC REHABILITATION

- Combination of exercise, psychological and educational interventions
- Exercise-based rehabilitation of patients with cardiac disease showed lower rates of all-cause mortality and lower systolic blood pressure. (Taylor et al 2004) - human
- Exercise rehab is recommended in the post-acute phase of pulmonary oedema and other cardiac conditions.
- Physical deconditioning occurs as a result of the immobility associated with severe illness.

Programmes

- Pre-exercise assessment is essential
  - Eg 6 minute walk test
- Exercise programmes should be designed to include warm-up, aerobic conditioning and cool-down phases.
- Differs according to patient's typical level of daily exertion – patients with physically demanding lifestyles need to achieve a higher intensity level of rehabilitation than those with more sedentary lifestyle.
- Carefully graded throughout the rehabilitation programme
- At least two exercise sessions per week.
- Programme recommended to be a minimum of eight weeks.

Canine cardiac patients

- Limited literature available on the effects and benefits of exercise in canine patients suffering from cardiac disease.
- Teach owner to reliably monitor their dogs response to the intensity of the exercises – respiratory and heart-rate monitoring.
- Controlled exercise programme focusing on cardio
- Weight management
- Treat other issues? Often geriatric dogs, joint stiffness, muscle atrophy?
- Enhance Quality of Life

VETERINARY PHYSIOTHERAPY: THE NEUROLOGICAL PATIENT

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ABSTRACT

The neurological patient can become a difficult case to manage. Veterinary physiotherapy utilises modalities such as PNF and other manual techniques to aid in normalising tone, addressing proprioception and optimising function. Many patient specific rehabilitation exercises and a carefully setup programme will put the focus on functional recovery, making patient management easier for owners and veterinary staff members.

TYPICAL PATIENT CASES

- Spinals
  - Type I and type II disc
    - Conservative
    - Surgical
  - CCVM
- FCE
- Spondylosis
- Degenerative Myelopathy
- Cauda Equina Syndrome
- Peripheral Nerve injuries
- Plexus injuries

MAIN AIM OF TREATMENT

- Function
  - Retraining motor control patterns
- Owner education/home environment
- Address origin of problem/dysfunction
  - Surgical site
  - Prolapsed disc
- Address secondary pain or compensation
  - Wounds
  - Scuffing wounds
  - Muscle pain – back pain, shoulder region
  - LMNL vs UMNL

LMNL

- Decreased tone
- Absent/decreased reflexes
- Hypometric gait/scuffing
- Acute atrophy
- Rx
  - Address origin of problem
  - Stimulate nerve
  - Maintain until nerve function regained
    - Muscle mass
    - Function

UMNL

- Increase tone
- Compensation
- Owner education
- Increased tone

- Increased reflexes
- Hypermetric gait
- Chronic atrophy

- Rx
  - Address origin of problem
  - FUNCTION
  - Motor patterns
  - Owner education
  - Decrease tone
  - Maintain muscle mass

FUNCTION AND MOTOR CONTROL

- Brain
  - Motor templates
  - Retrain and repeat
- UMNL
  - Nerve not the problem
  - “pathway” is the problem
- NB to retrain pathways….
- Use “key segments” to initiate movement
  - Head
  - Shoulder girdle
  - Hip girdle

EXAMPLE

Working with a tetraplegia patient the aims for functional, motor control retraining will be in the following sequence.

1. Lateral recumbence to sternal lying (note hind leg position)
   - Head control in sternal lying
   - Ability
   - Endurance
   - Fx in position/mvt
2. Sternal lying to sitting
3. Sitting to standing
4. Standing
5. Walking

Rx

- PNF
- NDT
- Sensorimotor techniques
- Postural reactions
- Manual therapy

PNF

- Relaxation, re-education, stabilization, strengthening and co-ordination training of body
- Progressive, function orientated, repeated several 100 times!!
- Use
  - Touch, voice, joint compression/distraction, repetition, sensory motor!!
- Run, walk, scratch
- Lateral recumbence, Sternal recumbence, sit, stand, walk
- Any dog like activity!! With lots of touching!!
- Tone
  - To decrease tone
    - Weight bearing
    - Sustained stretching
  - To increase tone

        - Weight bearing
        - Movement with shaking
        - Ice sweeping
        - Quick stretch outside range of movement trying to re-educate
        - RS
- Contractures
    - Use MT

NDT

- BOBATH
    - Normalise tone
    - Inhibit incorrect postural responses
    - Promote normal development of postures and movement
- Relearn basic movement patterns
    - Avoid learned non-use
- Facilitate normal tone, posture and balance
- Sensorimotor
- Keep evaluate and change
- Postural reactions
- Physioball (front feet, body)
- Weight shifting
- Treat to introduce head as control point in movement
- Static position – push, treat disrupt
- Advance – hopping, stepping

VETERINARY PHYSIOTHERAPY: THE SURGICAL PATIENT

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ABSTRACT

Veterinary Physiotherapy for the surgical patient aims to speed up healing, alleviate pain, speed up recovery, empower owners as part of the rehabilitation team, recognise and prevent complications, prevent re-injury and to ensure optimal recovery to return to highest functional level. General protocols will be explored. This paper will look at a basic outline of treating the surgical patient.

GENERIC PROTOCOL

These protocols will change and needs to be adapted taking into account

- Type of surgery
- Contra indications & precautions
- Original injury/reason for surgery
- Surgeons technique/surgeon protocol
- Patient signalment to set realistic goals
- Pre-operative functional ability and other injuries/weaknesses

Even though protocols are a great tool to try standardise treatment interventions, they bring about an enormous amount of problems. Working in an ever-changing growing veterinary medical field, with a patient that is never exactly the same living in home environments that are also not comparable. For these reasons clinical reasoning skills are crucial when applying protocols.

GENERAL GOALS

- Phase one:
  - Immediately after surgery and in initial inflammatory phase of tissue healing through to reparative stage
  - 3-4 weeks
    - Resolve pain and inflammation
    - Stimulate early tissue healing
    - Preserve muscle mass
    - Joint ROM and articular homeostasis
    - Prevent mechanical dysfunctional compensatory postures and movement strategies by patient
- Phase two:
  - Targets challenging the healing tissues during remodelling and maturation stages of healing to improve strength and mobility, mobilise scar tissue and enhance function

ACUTE STAGE

- Lifestyle management
  - Reduce risk of re-injury
  - Educate owners (play, toileting,leash walks,no stairs,jumping, slippery floors)
  - Slings?
- Manage inflammation
  - Low doses modalities – US, Laser, PEMF after 24-48 hours
  - ICE
  - Gr I Maitland mobs – distractions, glides, compressions
  - NMES
- Enhance or maintain ROM
  - PROM – avoid pain and end of range
  - Active ROM – easy WB exercises, Functional things
- Proprioception

- ROM, Maitlaing Gr I – Ruffini ending or Pacinian corpuscles in joint capsule (awareness)
- Massage – Golgi tendon organs and muscle spindles
- WS - - proprioception
- NMES – early strengthening and propioception

- Axial skeleton
  - NBNB

SUBACUTE STAGE

- Resolve pain and inflammation
- Stimulate early tissue healing
- Preserve muscle mass
- Joint ROM and articular homeostasis
- Prevent mechanical dysfunctional compensatory postures and movement strategies by patient
- Strengthening
  - Muscular support
  - Start increase leash time to 20 min
- Hydro
- Soft tissue stretching and ROM
  - All joints – affected and others
  - Muscle stretch
- Proprioception
  - More challenging - wobble boards etc

MID STAGE

- Targets challenging the healing tissues during remodelling and maturation stages of healing to improve strength and mobility, mobilise scar tissue and enhance function
- Strengthening and proprioception
  - Increase time, distance terrain, speed, steeper hills
- Gait re-training
  - Maybe taping? Bandaging etc needed?

END STAGE

- Strengthening and proprioception
  - If all good
    - Destination jumping
    - Longer cavaletti poles sessions
    - Gradual off leash return
    - Warm up, off leash, cool down

OTHER ASPECTS TO REMEMBER

- Owner involvement and understanding
- Home visit
  - to help recognise problem areas
  - ADVICE OWNER
- Patient behaviour
  - Very active?
  - Destructive?
  - Puppy? Socialising?
  - Pre-surgery treatments to condition patient?

VETERINARY PHYSIOTHERAPY: WHAT IS IT ALL ABOUT AND HOW CAN IT CONTRIBUTE?

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ABSTRACT

Veterinary Physiotherapy is starting to play an important role in managing the veterinary patient holistically. This profession addresses the neuro-musculoskeletal systems, (as well as cardiorespiratory), recognising functional dysfunction and altered biomechanics, which leads to symptomatic pain and compensation patterns and inevitable altered function. Veterinary physiotherapy aims to relieve, recover and prevent altered biomechanics and dysfunction to ensure optimal function for patients, regardless of the origin of the problem (surgical, traumatic, age).

DISCUSSION

Veterinary Physiotherapy aims to address injury, dysfunction recovery, prevention and maintenance in a wide field of different animals and levels of activity. Ranging for the geriatric to the elite agility dog, not compromising on the general pet. The fields include working with surgical, neurological, geriatric and athletes. The cardiorespiratory demand is growing. Even though the patient function and goals will differ for each of these, the end goal is optimal performance.

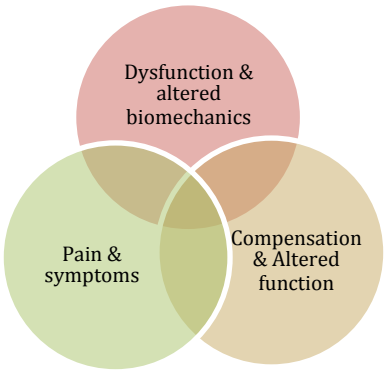
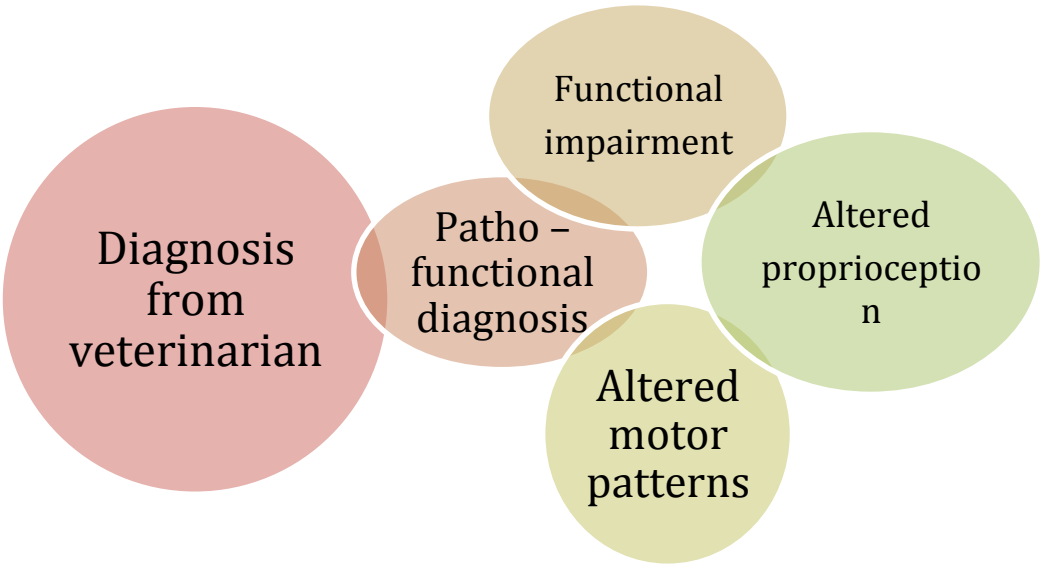


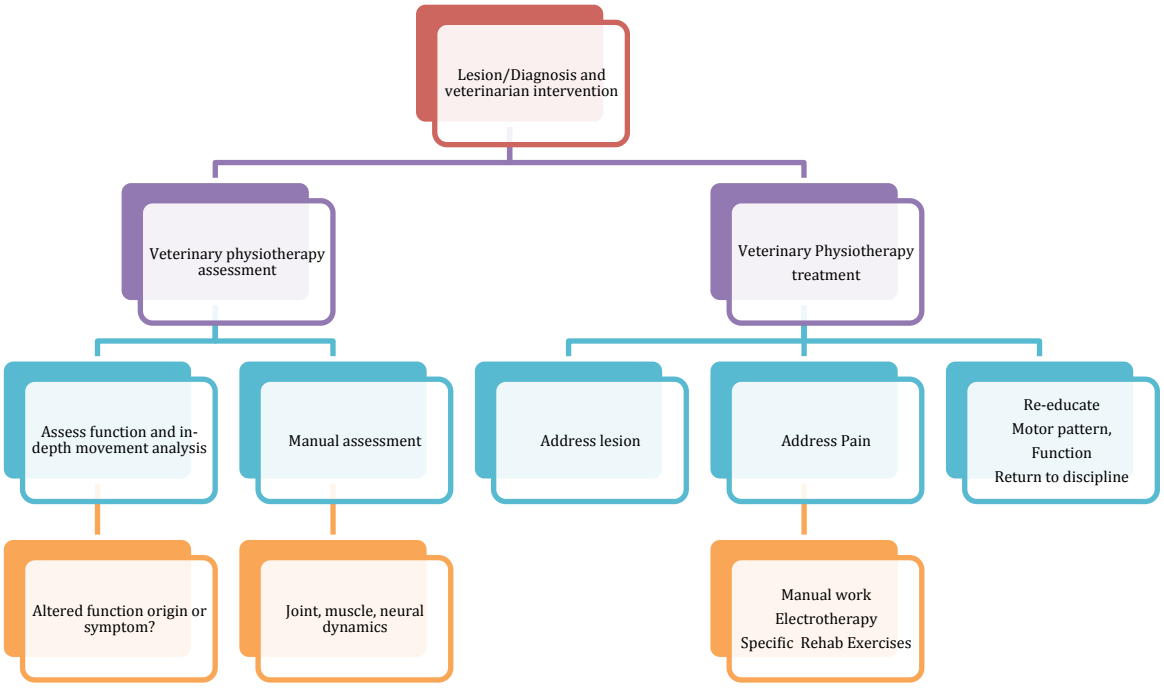
Fig 1. Neuromusculoskeletal

Looking at figure 1. When evaluating patients we can divide our findings into the above-mentioned groups. It is crucial for us to identify the true dysfunction. This dysfunction will lead to altered biomechanics giving rise to pain and symptoms and leading to compensation and altered function and inevitably poor performance.

For a Veterinary Physiotherapist to be successful in achieving their goals in treatment it is therefore crucial to differentiate between symptoms and the origin of the problem – on anatomical structure level. This is where a diagnosis from the veterinarian is vital. A definitive diagnosis gives the therapist the ability to identify a patho-functional diagnosis, recognising the dysfunction of the anatomical structure, the functional impairment, altered motor patterns and altered proprioception. The functional impairment, altered proprioception and altered motor patterns are the aspect of dysfunction that can limit the animals performance for months and years after the original dysfunction was addressed. These aspects must be identified and specifically addressed to ensure a better outcome long term.



Below is the layout of a typical evaluation and intervention. The ability to clinically reason is central to the success of a Veterinary Physiotherapist.



Case example:

- 5 yr old agility border collie
- Veterinary Diagnosis –Lx spondilosis.
- Veterinary treatment – rest, pain medication (?retire)
- Veterinary Physiotherapy:
  - Function: altered standing position, altered gait to compensate for discomfort, altered proprioception,
  - Pain: Epaxials, gluteus, Lx joints, FSJ,
    - Discomfort over SIJ – DSIL, ST attachments? Primary vs secondary?
    - Discomfort – Hip mobility and Iliopsoas?

        - ??? Thoracic limbs
    - Reduced lateral and DV mobility in lumbar spine – joint restriction
    - Reduced ability to stabilise– weak stabilisers
    - ? Atrophy or weakness HQ
- Treatment:
    - Pain:
    - Laser – muscle + joint pain
    - Manual soft tissue work - muscles
    - Myofascial release –muscles
    - Stretches – static and functional
    - Reduced mobility in lumbar spine
    - Mulligan mobilisations – lateral glide with physiological lateral flexion + DV glides
    - Reduced ability to stabilise – weak stabilisers
    - Re-educate stabilisers
    - Taping, theraband
    - Specific strengthening exercises to return to normal discipline as needed

The main treatment interventions used within the field of Veterinary Physiotherapy includes Manual Therapy – Looking at mobilisation techniques utilising physiological and accessory joint movements. Different types of massage, myofascial techniques and other soft tissue mobilisation techniques will be included here. Physiological manual therapy has an effect on the local tissues and on the neurological dimension.

Rehabilitation exercises specific to achieve identified dysfunctions are paramount to a longterm successful treatment and in preventing further re-injury. These can include the use of pilates balls, wobble balls/boards, cavaletti poles, taping and many more. The use of hydrotherapy and UWT's can also assist in achieving specific outcomes.

Electrotherapy modalities can include EMS, TENS, Faradic, ultrasound, Laser therapy, Shock wave therapy, and pulsed magnetic therapy.

With a difintive diagnosis from the attending veterinarian, a veterinary physiotherapist's assessment and intervention can speed up recovery, sometimes prevent re-injury, and with a carefully planned rehabilitation program patient can return to their highest level of function. Veterinary Physiotherapists manual skills and knowledge of the neuro-musculoskeletal system can also assist in finding the origin of the problem – especially when it is of soft tissue origin.

DIAGNOSIS AND MANAGEMENT OF OTITIS EXTERNA

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ABSTRACT

Otitis externa is one of the most common reasons why a dog is brought to a veterinarian and accounts for up to 15% of all canine veterinary case presentations. The causes of otitis externa are divided into primary and secondary causes and predisposing and perpetuating factors. All these factors should be considered when diagnosing and treating otitis externa in order to successfully manage these cases. Otoscopy and cytology of the ear canals are essential diagnostic procedures in every case of otitis externa. Other diagnostic procedures such as culture and sensitivity and imaging such as radiography, CT and MRI are indicated in some cases. Proper ear cleaning is indicated in most cases of otitis externa. Topical treatment is indicated in almost every case and systemic treatment in some cases as well. Biofilms are a newly recognized complication with especially *Pseudomonas* otitis externa. Management of specific causes of otitis externa such as allergic otitis externa and *Pseudomonas* otitis externa will be discussed. Owner compliance is crucial for successful management of otitis externa.

INTRODUCTION

Otitis externa (OE), defined as inflammation of the external ear canal from the pinna to the tympanic membrane (TM), is one of the most common reasons why a dog is brought to a veterinarian. It accounts for up to 15% of all canine and 6% of all feline veterinary visits[13]. Otitis externa (OE) affects up to 20 % of dogs and 6.6 % of cats[5, 10, 16].

ANATOMY OF THE EXTERNAL EAR CANAL

The external ear canal is a cartilaginous tube that extends from the external acoustic meatus to the pinna. It is lined with epithelium and continuous with the skin. The dermis contains large numbers of sebaceous and ceruminous glands that produce cerumen. Excessive cerumen and exfoliated cells are transferred towards the pinna via a lateral outward migration[15]. Canine ear canals are 5-10cm long and 0.5-1cm wide, L-shaped and comprise the pinna, vertical and horizontal ear canal and the TM, the barrier between the outer and middle ear. The TM is a slightly opaque structure divided into two parts: the pars tensa, a thin, dense and grey structure forming the larger ventral part and the pars flaccida a pink, looser and smaller dorsal part. The C-shaped manubrium of the malleolus is attached to the medial surface of the TM. The tympanic cavity is situated in the tympanic bulla, an air-filled cavity that contains the middle ear ossicles and communicates with the nasopharynx via the Eustachian tube[15].

CAUSES OF OTITIS EXTERNA

Cases of OE usually have a multifactorial aetiology. These causes and factors are classified as primary and secondary causes and predisposing and perpetuating factors[14, 26].

Primary causes

These are the factors that directly initiate inflammation and create ear disease.

- Parasites: mites, ticks and flies can affect the ears. *Otodectes cynotis* is the most common cause of OE in cats, especially kittens. *Demodex canis* and *Demodex cati* can cause OE, while *Sarcoptes scabiei* and *Notoedres cati* affect the ear pinnae[15].
- Dermatophytosis: a rare cause of OE, been described in young cats[15].
- Allergies (Atopy and Cutaneous adverse food reactions): Atopic dermatitis (AD) and cutaneous adverse food reactions (CAFR) are the most common primary causes of OE, especially of recurrent or chronic otitis in dogs. OE is reported as a clinical sign in 55% of atopic and 80 % of CAFR dogs and in 15% of atopic and 7% of CAFR cats[5]. Otitis may be the

only sign in 5 % of atopic and 25 % of CAFR dogs and may be uni- or bilateral[5]. Allergic otitis causes diffuse erythema of the ventral pinna and vertical ear canal. Other dermatologic signs of allergy are often present, but not in all cases. *Malassezia* infections are the most common secondary infections. Secondary bacterial infections are less common, but when present, are usually *Staphylococcus pseudintermedius* and in more chronic cases, gram negative bacteria such as *Pseudomonas aeruginosa* and *Escherichia coli*. AD is a common underlying cause of otohematomas[5,26].

- Contact allergy may occur to any topical medication applied to the ear, especially neomycin containing medications. The allergic reaction results in ongoing inflammation and a white to purulent discharge with mature non-degenerate neutrophils. There is often a history of an initial response followed by relapse and pain on administration[5].
- Foreign objects: Plant seeds are the most common foreign body found in the ear, but any foreign object or material, e.g. ticks, sand dirt, hair tufts and dried medication small enough to lodge in the ear canal can cause inflammation and subsequent problems. Small children can also introduce foreign bodies into a pet's ear[5, 14].
- Autoimmune conditions can cause ulceration and inflammation of the pinnae and, less commonly, the ear canals, but other clinical signs are usually more prominent. Pemphigus foliaceus is the most common. Other autoimmune conditions include pemphigus vulgaris, bullous pemphigoid, vasculitis and systemic lupus erythematosus[5, 26].
- Juvenile cellulitis causes severe purulent otitis in young puppies[5, 26].
- Keratinisation disorders may result in scaling and seborrhoea of the pinnae and ear canals. These include hypothyroidism, hyperadrenocorticism, sebaceous adenitis and idiopathic seborrhoea in Cocker Spaniels. Hypothyroidism is most common. These conditions usually affect the rest of the skin and body as well[26].
- Metabolic disease e.g. Zinc responsive dermatitis[14].

Secondary causes

The normal ear canal contains small numbers of commensal bacteria and yeast. Inflammation caused by primary causes results in a proliferation of these and pathogenic micro-organisms inside the ear canal. Acute inflammation results in infections with *Staphylococci* or mixed bacterial and yeast infections. Chronic inflammation causes a modified micro-environment which results in a change in the bacterial population. This, together with treatment of chronic cases with repeated courses of topical antibiotics leads to the development of a less predictable, more resistant bacterial population, especially Gram-negative bacteria, such as *Pseudomonas*[26].The most common bacteria causing infections of the ear canal are cocci (*Staphylococcus, Enterococcus* and *Streptococcus*) and/or rods (*Pseudomonas, Proteus, Corynebacterium* and *Escherichia coli*). *Pseudomonas* is the most common bacterium involved in chronic recurrent bacterial OE. Yeast infections are most commonly caused by *Malassezia pachydermatis*, and in rare cases by *Candida* organisms[14, 26].

Predisposing factors

Factors that increase the risk of developing OE and include:

- Breed Predilection: Dog breeds with a genetic predisposition to allergy and certain breeds including Golden and Labrador Retrievers, Cocker and Springer Spaniels, and Poodles, have a high incidence of OE. Anatomic breed characteristics e.g. pendulous ears and narrow or stenotic ear canals can be predisposing factors. Excessive hair in the ear canal, which can cause slower drying and increased moisture in the ear canal, can also be a predisposing cause[5, 26].
- Way of life: Excessive moisture in ears caused by swimming or the doggy parlour, or in hot humid conditions stimulates the production of more cerumen which can block the ear canal and promote yeast and bacterial proliferation. Vigorous hair plucking and over aggressive ear cleaning can cause inflammation and lead to infection[5, 26].
- Obstructive ear disease**:** Ceruminous gland tumours are the most common and can be benign adenomas or malignant adenocarcinomas. Benign tumours only cause clinical signs when they reach a certain size, whereas malignant tumours cause clinical signs early on and can therefore also be classified as primary causes[26]. Other malignant tumours include squamous cell carcinomas, mast cell tumours and melanomas. Benign tumours/growths include basal cell tumours, papillomas and inflammatory polyps in young cats[6, 14].

Perpetuating factors
These are responsible for continuing the inflammatory process. They include otitis media and chronic anatomic and pathological changes in the ear canal itself[14].

- Otitis media is defined as inflammation of the middle ear cavity. Incidence is 16% in acute OE and 50-80% in chronic OE. The most common infectious agents causing middle ear disease are *Pseudomonas aeroginosa, Staphylococcus pseudintermedius, Proteus, Escherichia coli* and *Malassezia pachydermatis*. Inflammation of the middle ear results in purulent, mucous exudate in the tympanic bulla. This either occurs by an infection of the outer ear followed by a TM rupture, by an infection from the nasopharyngeal area via the Eustachian tube into the middle ear or in rare cases by haematological spreading. Otitis media should be considered when a patient presents with neurological disease affecting the head including vestibular disease, Horner's syndrome, or facial nerve damage. The diagnosis of otitis media in dogs can be difficult due to the L-shaped conformation of the ear canal, which makes it hard to visualise the TM. Many patients with otitis media have an intact TM (up to 70% of cases), giving the impression that the middle ear is normal. Most canine patients with otitis media also have chronic OE with pathological changes to the ear canal. It is often theorized that otitis media is an extension of OE that was either not treated, improperly treated, or resistant to treatment[5].
- The changes in the epithelium and ear canal structures can become so severe that they perpetuate otitis. The chronic changes include proliferative change of the ear canal wall, canal stenosis, calcification of the pericartilaginous fibrous tissue, hyperplasia and hidradenitis of ceruminous and sebaceous glands and dilatation or rupture of the TM[14, 26].

A PSPP form can be used to assist the veterinarian to address all the causes and factors that play a role in a particular case (the PSPP form can be downloaded from www.animaldermatology.com)[14].

DIAGNOSIS

Diagnostic steps may include signalment and a good history, full clinical and dermatological examination, otoscopic examination, cytology, culture and sensitivity and imaging, e.g. radiographs, CT and MRI.

Signalment and history
Breed, age when started with OE and a full history are crucial, as this provides information about possible primary and predisposing factors.

Clinical examination
Full clinical and dermatological examination of the entire dog is very important, not only the ears should be examined. Clinical signs may include head shaking; scratching, rubbing and pawing at ear(s); pain, pruritus and redness of the ears, smelly ears, discharge from the ear canal, crustiness on pinnae/inside of the ear, a hot spot close to affected ear and an otohaematoma. With deeper ear problems, neurologic signs, such as head tilt and/or nystagmus, vestibular disease, Horner's syndrome, facial nerve paralysis and deafness, may occur. Unilateral keratoconjunctivitis sicca may result from facial nerve damage[3, 10, 14].

The different types of OE are classified as:

- *Erythematous OE*: the external ear canal and pinnae are erythematous, without significant discharge. This may be seen in early atopic OE and can indicate the presence of inflammation without infection[8].

- *Erythroceruminous OE*: the most common, presents with erythema and a ceruminous discharge. Erosions and ulcers are rare. The inflammation, discharge and chronic pathological changes vary from mild to very severe. The ears are usually more pruritic than painful. *Staphylococci* and *Malassezia* are the most common organisms. The exudates are usually yellow-brown with bacteria and chocolate brown with *Malassezia*, although there is overlap and mixed overgrowths are seen[8, 19].

- *Suppurative OE*: more common with chronic OE, characterised by erythema, inflammation, pain, ulceration, haemorrhage, a foul odour and a purulent discharge (usually yellow to green). Most

cases are associated with Gram-negative infections, especially *Pseudomonas* infection. Occasionally staphylococci or streptococci can also be found. *Malassezia* is rare[8, 19, 21].

- *Otodectes OE*: characterised by a dry, black to dark brown, coffee ground type of discharge.

Otoscopic examination (Otoscopy)
Otoscopy is performed to evaluate the ear canals at the first consultation and at follow-up visits to evaluate efficacy of treatment and ear cleaning. Both ears should be examined even with unilateral OE, the least affected ear first. In cases with severe inflammation, pain, ulceration and stenosis of the ear canal, systemic (and topical) glucocorticoids should be given for 7 to 14 days to resolve inflammation and decrease stenosis[8, 24].

The canine ear canal is not straight. The vertical canal runs ventro-rostrally before turning medially into the horizontal canal. A ridge of cartilage projects from the medial wall at the junction between the horizontal and vertical canals. This area is especially vulnerable to trauma from otoscopy. The pinna should be held up to straighten the ear canal, and the otoscope cone is inserted first in a rostroventral direction and then once around the medial ridge, is directed medially to view the horizontal canal. The healthy ear canal should have a smooth, non-inflamed lining[8, 20].

Otoscopic examination should assess the presence and nature of any discharge, evidence of ectoparasites, presence of foreign bodies, appearance of ear canal lining, any ulceration, patency of ear canal and degree of stenosis, the appearance and patency of the TM and the presence of neoplasms or polyps[8, 20]. It is important to clean and disinfect the otoscope properly after use. Water, wiping and 70% alcohol are not effective disinfectants for this purpose. Soaking in 2% chlorhexidine has been shown to be very effective[17].

Biofilms
Biofilms are a very important, newly recognised entity, commonly seen in chronic OE cases. All common bacterial (*Staphylococcus, Pseudomonas*) and yeast (*Malassezia*) pathogens of canine OE are capable of forming biofilms[28]. Biofilms form when microbes attract one another, join, adhere to a surface and produce a slimy, glue like, protective matrix that anchors them to the surface. This matrix acts as a colony, not an isolated organism. The outer layers of bacteria protect those inside the colony[28]. Biofilms are clinically important because they inhibit cleaning and antimicrobial penetration. The impeded antimicrobial penetration results in a sudden drop in the antimicrobial concentration of a topical ear medication, which results in bacteria being exposed to either high, low or intermediate antimicrobial concentrations. The bacteria exposed to high concentrations will be eliminated. The low concentrations will result in unaffected bacteria which will act as a reservoir and lead to treatment failure. The bacteria exposed to intermediate concentrations of antimicrobials will either be eliminated or allow more resistant mutants to survive and proliferate. The end result is treatment failure and recurrence of a more resistant isolate[19, 21]. Clinically, biofilms appear as a dark brown to black, adherent, thick and slimy discharge[19, 21]. They are common and under-diagnosed, although they can be easily identified on otoscopy or cytology.

Cytology
Cytology is inexpensive, highly informative, easily self-taught and can be performed in-house. It is mandatory to perform cytology in every case of OE[13]. Samples should always be collected after otoscopy, collected from both ears, the least affected ear first. A clean cotton bud should be used for each ear canal. The best samples are collected from the junction of the vertical and horizontal ear canals[7, 9, 16]. Collected material is gently rolled onto a glass slide and stained with DiffQuik stain. Purulent discharges should be air dried and fixed with the alcohol fixative before being stained with the other two stains. Waxy deposits on the glass slide may be heat fixed e.g. with a cigarette lighter. This helps prevent loss of material during staining[20]. These samples are stained without using the alcohol fixative to avoid dissolving the material collected. The blue stain is effective for staining *Malassezia* yeasts[7]. In cases of suspected *Otodectes* or *Demodex*, material is placed onto a glass slide with a drop of liquid paraffin, a cover glass is added and examined on low power[9, 11, 20].

Cytology samples are evaluated for the presence of mites, yeasts, bacteria (rods/cocci), leukocytes, other cells and biofilms. The numbers of each are scored 0 – 4 according to a scale[11]: 0 - no organisms, 1 (1 – 3), 2 (4 – 10), 3 (11 – 30), 4 (> 30) organisms per high power field (HPF), after

evaluation of 5 to 10 areas under HPF. Cytology assists to choose appropriate treatment and helps to monitor progression of disease or response to therapy[7, 16, 20].

Normal ear canals may have micro-organisms present on cytology. There may be up to two yeast and up to 5 cocci bacteria per HPF in normal canine ear canals[5]. Rod bacteria and inflammatory cells are never normal. Normal ear cytology also shows keratinocytes, wax and lipid[5]. Abnormal cytology seen in dogs with OE may include large cocci in pairs or clusters that suggest *Staphylococcus, Streptococcus* and *Enterococcus* are slightly smaller cocci and tend to form chains[20]. Rod bacteria that may be *Pseudomonas, Proteus* and other Gram-negative species as well as *Corynebacterium* all look very similar. All bacteria stain dark blue with DiffQuik stain, Gram stains are necessary to distinguish them[20]. Peanut-shaped yeasts are characteristic of *Malassezia* infections. Most cocci and *Malassezia* infections are associated with overgrowth in the absence of neutrophils. Large numbers of degenerate neutrophils with intracellular bacteria are usually seen with *Pseudomonas* infections, occasionally with severe *Staphylococcus* infections. Neutrophils are also seen in contact allergy reactions, and both red blood cells and neutrophils are common with ulceration of the ear canal[11, 20]. Biofilms have the appearance of mucoid slime and appear as unevenly thick veil-like material that obscures bacteria and cells on cytology[19, 20, 21].

Culture and sensitivity

Bacterial culture and sensitivity are not required in every case. Micro-organisms can easily be identified by cytology. *Malassezia* and cocci bacteria usually have a predictable sensitivity, but rod shaped bacteria do not and are frequently resistant to many antibiotics. Culture and sensitivity is indicated for severe chronic proliferative cases AND cases with rod-shaped bacteria and inflammatory cells on cytology AND where empirical treatment has not resolved the infection AND where all other causes of failure of therapy have been ruled out as well as for Otitis media[12]. Opinions are divided as to the value of bacterial culture and sensitivity in OE. Sensitivity is based on systemically achieved antibiotic levels, not topical levels. Topical medication reaches a 100 - 1000 fold higher concentration and will overcome apparent in vitro bacterial resistance in most cases, rendering culture and sensitivity of lesser value[12, 16, 18].

Another consideration when interpreting culture and sensitivity results is that bacterial cultures from ear canal samples often grow three and sometimes more isolates, sometimes normal flora, per specimen. It is therefore important to interpret culture results together with cytology findings. Other reasons for variable results include concurrent antimicrobial treatment, sample location and variability among labs. Samples should preferably be taken 48 hours after the last dose of oral antibiotics or beyond the appropriate dose interval for topical or parenteral antibiotics. If appropriate withdrawal times are not possible but cytology indicates presence of micro-organisms, enriched cultures may be necessary. It is therefore important to communicate recent or on-going antibiotic therapy to the laboratory[12, 16, 18].

Diagnostic imaging

Diagnostic imaging is primarily indicated in cases with recurrent or severe otitis, neurological signs (vestibular syndrome), nasopharyngeal polyps and otitis media. The imaging techniques available include conventional radiographs, computed tomography (CT) and magnetic resonance imaging (MRI). These imaging techniques are useful adjunctive diagnostics but they each have advantages and disadvantages to their usefulness[1].

Conventional radiographs are used most commonly and can be performed at most veterinary hospitals. The radiographic views needed to evaluate the ear canal structures and middle ear are challenging and require anaesthesia or heavy sedation of the patient. Variability in the size and shape of various canine skulls make precise positioning difficult for certain breeds. The dorsoventral (or ventrodorsal) and rostrocaudal (open-mouth) views are generally the most useful. The dorsoventral view allows a side by side comparison of the bullae and petrous temporal bones. The disadvantage is that the bullae are superimposed on the petrous temporal. The rostrocaudal open mouth view has the advantage of a side by side comparison of the bullae and external ear canals but disadvantage that positioning is difficult. The diagnostic value of radiographs is questionable as radiographic detection of a soft tissue density in the middle ear cannot distinguish between fluid or tissue[1].

Computed tomography (CT) is based on the same physical principles as radiography and produces images that resemble radiographs. A major advantage of CT is that structures can be examined

without the confusing effect of superimposition. CT enables a detailed examination of the ears and adjacent structures, is very sensitive and specific for stenosis and occlusion of the ear canals, bulging or rupture of the TM and is an excellent choice for imaging the middle ear[1].

Magnetic resonance imaging (MRI) is fundamentally different in principle from radiography or CT and, therefore, produces images with very different properties. MRI is preferable to CT for examining the soft tissue components of the external ear, the inner ear and the brain. Structures such as the tympanic bullae and paranasal sinuses are difficult to examine using MRI[1].

MANAGEMENT OF OTITIS EXTERNA

The basic principles of successful OE management include the following: identification and treatment or management of all primary and secondary causes and predisposing and perpetuating factors; use of systemic (and topical) glucocorticoids to increase patency of a stenotic ear canal before attempting full examination, otoscopy and flushing; complete cleaning and flushing of the ear canal initially and long term cleaning; elimination of swimming if possible; using a sufficient volume of topical medication to fill the ear canal; continuation of antimicrobial treatments for 2 weeks after cytologically resolved infection; a long term maintenance program to prevent recurrence and surgical options for ears that cannot be medically salvaged[12].

A treatment plan should be formulated that is tailored specifically to each patient after skin and ear evaluation, otoscopy, cytology and ear canal cleansing. It is unfortunately not always possible to completely resolve or successfully treat all causes and factors of OE in a specific patient. Allergic disease can often be managed only partially due to atopic dogs experiencing seasonal flare ups and food allergic dogs accidentally eating the wrong food. Predisposing breed characteristics, such as high numbers of ceruminous glands, pendulous ears, stenotic ear canals and excessive hair, cannot be eliminated, only managed. Similarly, it may not be possible to stop water-loving Golden Retrievers from swimming. Such cases may require long-term management. This should be discussed with the owners so that they have realistic expectations of what to expect[12].

Ear cleaning/flushing

Ear cleaning is an important component of managing OE and is indicated when discharge within the ear canal prevents visualisation of the TM or areas of the ear canal lining. Benefits of ear cleaning include facilitation of examination of the ear canal, removal of micro-organisms, exudates, biofilms and small foreign bodies, exposure of the ear canal lining to topical therapy and removal of debris and purulent material which greatly improves the efficacy of topical antimicrobials, especially polymyxin B and aminoglycoside. As mentioned before, cases of OE with severe inflammation, pain and proliferation that require ear cleansing, require systemic glucocorticoids for 7 to 14 days before the ears are cleaned. The steroid therapy decreases oedema, ceruminous gland hyperplasia and stenosis and renders the microclimate in the ear less suitable to bacteria and yeast and prevents maceration during the flushing procedure. If the ear canals do not “open up” with the systemic glucocorticoid therapy, medical treatment is likely to fail and surgery may be necessary[16, 24].

Types of ear cleaners

In cases with a ruptured or non-visualized TM, only warmed sterile saline or water should be used. Many different ear cleaners are available for dogs and cats with an intact TM. Ear cleaners commonly contain one or more of the following: cerumenolytic, astringents or drying and antimicrobial agents.

Cerumenolytic agents soften, emulsify and dissolve waxy ceruminous build up and debris. They need 10-15 minutes to work and should be applied prior to cleansing. These agents should be flushed out during ear cleaning and are not safe if the TM is ruptured. Dioctyl sodium/calcium sulfosuccinate are active ingredients with good cerumenolytic activity[13, 16, 24, 27].

Drying or astringent agents prevent maceration of the ear canal. They are either combined with cerumenolytic agents or used solely after deep ear cleansing or as a prophylaxis after swimming and bathing in dogs prone to OE. Examples are isopropyl alcohol, acetic acid, benzoic acid, malic acid, boric acid, lactic acid and salicylic acid as well as sulphur and aluminium acetate[13, 16, 24, 27].

Antimicrobial agents include acetic and boric acid or ketoconazole that have an antifungal effect and acetic, lactic or boric acid (or other low pH maintaining agents), salicylic acid, chlorhexidine (at less than 0.2%), TrisEDTA or parachlorometaxylenol (PCMX) that have antibacterial effects. Tris EDTA potentiates the antiseptic capacity of chlorhexidine[13, 16, 27].

Several commercial ear cleaners are available in South Africa, containing one or more of the active ingredients mentioned above. An example of an effective ear cleaner containing a combination of active ingredients is Epi-otic® (New) (Virbac) which contains disodium EDTA, salicylic acid, PCMX (Polychlorometaxylenol), docusate sodium and monosaccharide complex.

The veterinarian should preferably perform the initial cleaning/flushing. General anaesthesia is required in most cases. It is important to place an endotracheal tube during general anaesthesia, because respiratory contamination can occur if large volumes of flushing fluid were to flow into the nasopharynx via the Eustachian tube during the flushing procedure in cases with a ruptured TM. After the initial cleaning, regular home cleaning by the owner is important for controlling and preventing chronic OE. It is important to demonstrate to owners how to clean the ears. In most cases an owner should clean the ears one to three times weekly and after swimming, bathing or grooming for general maintenance[16, 24].

Treatment of biofilms and mucus

Biofilms can be physically broken up and removed by thorough flushing and aspiration. Topical Tris EDTA and n-acetylcysteine can disrupt biofilms, facilitating their removal and enhancing penetration of antimicrobials. TrisEDTA damages bacterial cell walls, increases antimicrobial efficacy and keeps the ear canal at pH of 8.0, which is optimum for function of aminoglycosides and fluoroquinolones. It should be given 20 to 30 minutes before the antimicrobial but can be co-administered. It is well tolerated and non-ototoxic. TrisEDTA has additive effects with antibiotics including gentamicin, fluoroquinolones, silver sulphadiazine and chlorhexidine (0.2% or less safe for the middle ear)[19, 21].

Topical and systemic antimicrobial therapy

Topical therapy is preferred wherever possible as it reaches concentrations of 100 to 1000 times higher than systemic drugs. Topical antimicrobial therapy is indicated if cytology identifies infection. The integrity of the TM determines which topical agents should be used. Systemic therapy rarely reaches therapeutic concentrations in the skin of the ear canal and within fluid and waxy exudates. It is indicated in cases where the epithelial lining is extensively eroded and ulcerated, in cases of otitis media, when the TM is ruptured and when the ear canal cannot be treated topically (e.g. stenosis or compliance problems or if topical adverse reactions are suspected)[19, 21].

Topical antimicrobials

All commercial veterinary topical ear products contain an antifungal, antibacterial and glucocorticoid. Choosing appropriate topical treatment is based on the history, clinical examination, and cytology result and with regard to good antimicrobial stewardship[8]. The volume of topical ear medication administered is very important. Counting drops increases administration time and because the nozzle is not inside the canal, reduced penetration of medication will occur. Squeezing the bottle is not accurate either, as the nozzle is inside the canal and under- and overdosing can occur. A 1 ml syringe is most accurate to measure the correct volume of ear medications. The new ear product Easotic® (Virbac) contains a pump applicator that ensures that the correct volume of medication is given. The following volumes have been recommended: cat 0.15-0.2 ml; dog (5-10 kg) 0.25-0.5 ml; dog (10-20 kg) 0.5 ml; dog (20-40 kg) 0.75 – 1 ml; dog (> 40 kg) 1 – 2 ml[31].

Topical antimicrobial agents

Fusidic Acid: Bacteriostatic, effective against Gram positive cocci

Aminoglycosides: Bactericidal, broad-spectrum with excellent Gram-negative bactericidal effect. Include neomycin, gentamicin, amikacin and tobramycin. Gram-negative bacteria (including some *Pseudomonas*) have less resistance to amikacin or tobramycin than to gentamicin or neomycin[27]. Aminoglycosides show decreased effectiveness in acidified ears, enhanced antimicrobial activity in alkaline environment and are inactivated by purulent material[17]. Neomycin is less potent that other aminoglycosides but is effective against Gram-positive bacteria. Neomycin has been implicated as a cause of contact dermatitis in the ear[3, 16].

Polymyxin B: Bactericidal, effective against Gram negative bacteria, ototoxic, inactivated by cellular debris, therefore prior ear cleaning is important[3, 16, 27].

Fluoroquinolones: Bactericidal, effective against Gram-positive and -negative bacteria. Decreased effectiveness in acidified ears. Enrofloxacin injectable solution, diluted in sterile saline, can be used topically when TM is ruptured[3, 16, 27].

Silver Sulfadiazine: Broad-spectrum antibacterial, excellent activity against *Pseudomonas* and *Malassezia*. Inactivated by purulent material[5, 16]. Available as a cream (recipe given later).

TrisEDTA: Commonly used, either as pre-soak or carrier vehicle for treatment of Gram-negative infections[3, 27].

Ototoxicity: Antibiotics such as gentamycin, tobramycin, amikacin, neomycin, and polymyxin B are potentially ototoxic, therefore if there is no TM, these antibiotics should be avoided[19, 21]. Enrofloxacin and silver sulfadiazine appear to be safe in the middle ear. The ototoxicity of gentamicin appears to depend on the preparation. Topical application of injectable solutions of gentamicin appears to be safe. Vehicles may also be ototoxic e.g. propylene glycol.

Topical anti-fungal drugs
Miconazole, clotrimazole, ketoconazole and silver sulphadiazine are effective for the treatment of *Malassezia* infections. Nystatin has mixed efficacy and thiabendazole poor efficacy[19, 21, 27].

Systemic antimicrobial treatment
Indications for the use of systemic antimicrobials include the presence of neutrophils with bacteria on cytology; significant proliferative changes in ears; bacterial and *Malassezia* otitis media and owner inability to administer topical antimicrobials, e.g. patient's personality or owner's own limitations[3].

Empirical systemic antimicrobials
Clindamycin, cefalexin and amoxycillin/clavulanic acid are good first line drugs for staphylococcal infections. Cefovecin is appropriate if compliance and/or administration are likely to be difficult[19, 21].
Cephalexin, enrofloxacin and marbofloxacin are good empiric choices for rod infections. Fluoroquinolones cause cartilage damage in dogs < 12 months old (18 months in giant breeds), neurotoxicity at high doses, and blindness in cats (especially with injectable enrofloxacin).

Otitis media treatment
The tympanic bulla should be flushed clean with or without a myringotomy. Culture and sensitivity should be performed in all cases on samples obtained from the middle ear. Systemic antimicrobials should be given for at least 6 - 8 weeks. The initial choice should be based on cytology. Empirical systemic options for bacterial infection include enrofloxacin, marbofloxacin and cefalexin and itraconazole for *Malassezia* infection[19, 21]. Enrofloxacin (recipe given later) may be installed into the tympanic cavity. A study investigated medical treatment of otitis media. There were 86 dogs in the study, 95% had a ruptured TM. All cases were treated medically: 82% resolved, 7% were lost to follow up, 11% needed surgery, 7 of resolved cases later relapsed[22].

Anti-inflammatory treatment
The choice largely depends on the severity of the OE. Topical therapy is preferred as this delivers the drug to the affected site avoiding systemic exposure. Systemic treatment is necessary if there is stenosis, severe fibrosis or mineralisation, or if topical therapy cannot be safely administered. It is usually possible to switch to topical therapy once the ear canals have opened. Animals better tolerate topical therapy once the pain and inflammation has decreased[16].

Topical glucocorticoids
Glucocorticoids are an important part of OE therapy. They reduce inflammation and associated pruritus, exudation, swelling and tissue proliferation and hyperplasia. The glucocorticoids in commercial veterinary topical ear products are appropriate for managing mild to moderate inflammation in OE, but more severe inflammation requires longer courses of more potent products. Once the OE has resolved, topical glucocorticoids should be used at the lowest frequency that controls the inflammation. Potent corticosteroids such as betamethasone and fluocinolone may cause systemic effects over time because of local absorption and should not be used continually in chronic OE cases[19, 21, 27]. Systemic absorption can result in cutaneous atrophy, comedomes and demodicosis. Hydrocortisone aceponate, a novel topical diester glucocorticoid, is a highly active anti-inflammatory with potency similar to dexamethasone. It is an ingredient in a new topical ear medication, Easotic® (Virbac)[31]. Absorption through the skin and ear deactivates drug and no systemic effects occur.

Systemic anti-inflammatory treatment
Systemic glucocorticoids are often of value in treating OE. They decrease inflammation and pain associated with OE and as mentioned before, are especially useful in cases with considerable swelling, hyperplasia and proliferation of the ear canal. Prednisolone (1 mg/kg every 24 hours, tapering) for one to two weeks is sufficient to control inflammation and stenosis in most cases. Severe cases may respond better to betamethasone or dexamethasone. Glucocorticoids do not remove

hyperplastic epithelium or glands, so if there is no response to the more potent glucocorticoids after 7-14 days, the stenosis is rather the result of increased tissue growth rather than inflammation. Systemic ciclosporin has shown efficacy in some chronic OE cases. Long-term systemic prednisolone or ciclosporin should be at the lowest frequency and dose that prevents recurrence of the OE in cases where chronic topical maintenance glucocorticoids do not control the allergic OE[19, 21].

DIAGNOSIS AND MANAGEMENT OF ALLERGIC OTITIS EXTERNA

A diagnosis of allergic OE is made by ruling out all other causes of OE and pruritus in general. A proper diet trial is essential to rule CAFR in or out in every case of chronic recurrent OE. Secondary ear infections should be resolved prior to or during the early stages of the diet trial. Clinical, otoscopic and cytologic examinations should be repeated every 3 weeks during the diet trial.

Treatment of Acute and Infrequently Recurrent Allergic OE

These cases are usually successfully managed with commercial veterinary topical ear products that contain an antibiotic, anti-fungal and glucocorticoid combination. A short course of oral glucocorticoid is indicated for severely inflamed ears at 0.5 – 1.0 mg/kg/day of prednisone/prednisolone in the dog and 1.0 – 2.0 mg/kg/day prednisolone in the cat[8, 30].

Long Term Management of Allergic OE

Successful long term management of allergic OE requires ongoing regular anti-inflammatory treatment to maintain remission and prevent relapses of infection. Such proactive therapy gives a much better prognosis than reactive treatment, where treatment of each bout of infection gives short-term relief, but misses the ongoing inflammation in the absence of infection. This allows chronic inflammatory changes to develop that will result in more frequent and severe infections. These cases will eventually need a total ear canal ablation[19, 21].

Proactive ongoing anti-inflammatory treatment can be accomplished as follows: All secondary bacterial/yeast infections should be resolved with a combination topical product. Thereafter, long term maintenance therapy should continue with topical glucocorticoid products without antimicrobials in the absence of infections[30]. There are no registered glucocorticoid-only products for OE in veterinary medicine[19, 21]. Available options include human eye drops, ear drops and ear cleaners that contain glucocorticoids only (off label use in animals), twice-weekly hydrocortisone aceponate (3 drops/day) application in the ear canal which has been shown to control OE in atopic dogs[2] or compounded medications (off label) e.g. 0.1% dexamethasone solutions in acute cases (2mg/ml dexamethasone mixed 1:1 with saline) and 0.01% dexamethasone solutions which have been shown to be safe for long term use, e.g. twice weekly. When using glucocorticoids long term, it is important to consider potency and possible adverse effects. Routine ear cleaning once or twice weekly is an important component of the ongoing management of these cases. Dexamethasone can be added to ear cleaners as a compounded medication (0.01%) and used for long term anti-inflammatory control. During acute flare ups, rapid remission can be achieved with a short course of oral glucocorticoid. The overall control is easier if underlying allergy is well controlled (e.g. hypoallergenic diet for CAFR, allergen specific immunotherapy, antihistamines, essential fatty acids, glucocorticoids, oral cyclosporine for AD)[30].

DIAGNOSIS/MANAGEMENT OF *PSEUDOMONAS* & OTHER RESISTANT GRAM-NEGATIVE OE

Pseudomonas aeruginosa is the most common and most resistant Gram-negative bacteria associated with OE in dogs. *Pseudomonas* is a motile Gram-negative bacillus abundant in the environment but uncommon in canine ears. It grows in moist environments and is more common in tropical climates. Dogs with hairy, narrow canals and pendulous pinnae are predisposed to *Pseudomonas* infection. *Pseudomonas* does not infect “normal” tissue, so primary causes should be identified[23, 25]. These are not always evident and obvious. Chronic moisture associated with swimming may act as a predisposing and a perpetuating factor. *Pseudomonas* has mechanisms for evading the host’s immune response. These include the production of toxins and proteases (e.g. exotoxin A and lecithinase) and biofilms that protect *Pseudomonas* against the host immune system and topical medication[25]. The development of more resistant strains of *Pseudomonas* poses a continual challenge. *Pseudomonas* OE cases often have a history of chronicity and of failed, repeated or multiple therapies[23].

Diagnosis can be made by cytology that is characterised by a predominant population of rods with degenerate neutrophils, phagocytized bacteria, red blood cells and biofilms. Mixed infections with cocci and yeasts may also be present. Bacterial culture and sensitivity is indicated for severe chronic cases of OE where rods and inflammatory cells are found on cytology, when otitis media is present, when systemic antibiotics are deemed necessary, and in cases not responding to empirical therapy[25]. Samples for culture should be obtained from the junction of vertical and horizontal canals and from the middle ear in cases of otitis media. Where otitis media is suspected, radiographs or CT for better evaluation of middle ear involvement are indicated[23].

Management of *Pseudomonas* OE

The principles are the same as for any chronic OE case. Ear cleaning is very important with warm water, saline and trisEDTA all good choices. TrisEDTA is useful because it increases permeability of *Pseudomonas* cell membranes by binding calcium and magnesium ions. Ear cleaning should be continued during treatment and afterwards for long term maintenance and prophylaxis[30].

Topical antibiotics

First-line drugs include gentamicin and polymyxin B, both contraindicated with perforated TM, enrofloxacin and silver sulfadiazine. Commercial veterinary ear drops are not safe for use in the middle ear. Compounded aqueous solutions of enrofloxacin, gentamicin and silver sulphadiazine, used off label, are reported to be safe even if TM is ruptured. Pre-soaking the ear canal with trisEDTA helps to potentiate aminoglycoside and fluoroquinolone antibiotics[23, 25]. TrisEDTA may be a useful adjunctive treatment for chronic cases of *Pseudomonas* otitis where biofilms have developed, if gentamicin or neomycin is to be used as a topical treatment[29].

"Off label" topical treatment (No toxic effect to middle ear):

- Fluoroquinolone/Dexamethasone drops (50 ml): 5 ml Baytril® 10%, 9 ml dexamethasone (2 mg/ml), 36 ml trisEDTA or sterile saline[16].
- Silver sulphadiazine drops (1% solution): 1 ml silver sulphadiazine: 9 ml sterile saline, sterile water or trisEDTA[16.] A study by Buckley and colleagues demonstrated that trisEDTA significantly potentiates the bactericidal activity of silver sulfadiazine against multi-drug resistant *Pseudomonas aeruginosa*[4].

Second-line drugs include amikacin, tobramycin, ceftazidime and ticarcillin as off-licensed preparations. A culture and sensitivity should be performed before using these. Amikacin, tobramycin and ticarcillin all have ototoxic potential and should only be used when other safer products are deemed unsuitable[30].

"Off label" topical treatment (Ototoxic, not when ruptured TM):

- Injectable amikacin (50 mg/ml solution) – 1 ml per ear OID[30].
- Tobramycin - use eye drops or 8 mg/ml injectable solution 0.15-0.3 ml/ear OID[30].
- Ticarcillin – Ticarcillin and clavulanic acid (Timentin®) - 3.1 g vial; reconstitute with 26 ml (100 mg/ml); freeze in 2 ml aliquots; thaw and use each 2 ml aliquot over 2 days; 1/2 ml in each ear BID[30].
- Ceftazidime – 2 g vial of ceftazidime reconstituted with 10.5 ml sterile water. Divide the 12 ml of solution into three 4 ml aliquots. Add 10 ml of sterile water to 1 of the aliquots and freeze the others. In 1 week defrost 1 frozen 4 ml aliquot, add 10 ml of sterile water and make another fresh bottle[10].

Systemic antibiotics

These are indicated where cytology reveals neutrophils, there is poor response to topical treatment, the ear canals are hyperplastic or proliferative and/or there is otitis media. *Pseudomonas* are resistant to many antimicrobials through low cell wall permeability, β-lactamases, clavulanate-resistance and efflux pumps[19, 21]. They develop further resistance if treatment is ineffective as they have a large genome to express resistance genes and mutations, and are capable of plasmid, transposon and bacteriophage transfer. Marbofloxacin (5 – 10 mg/kg once a day, PO) and enrofloxacin (15 - 20 mg/kg once a day, PO) are first-line systemic antibiotics for *Pseudomonas* OE[23]. If the bacteria are considered resistant to the fluoroquinolones, gentamicin (5 – 10 mg/kg SC OID), amikacin (10 – 15 mg/kg SC OID), ticarcillin (60 - 75 mg/kg SC BID), carbenicillin (10 – 20 mg/kg IV TID) and ceftazidime (30 – 50 mg/kg SC BID) remain viable options but require parenteral administration[7, 30]. The last three antibiotics are not licensed for animals and should only be used where clinically justified and with informed consent of the owners[19, 21]. Patients on aminoglycosides should be monitored for nephrotoxicity with urinalysis for protein and tubular casts and serum for urea and creatinine every 1-2 weeks[30].

Glucocorticoids
Pseudomonas infections are not a contraindication to glucocorticoid therapy as glucocorticoids can reverse the ototoxic effect of *Pseudomonas* infections. Potent glucocorticoids should be avoided in severe *Pseudomonas* infections as they can suppress neutrophil activity. Once the infection has been resolved, it is crucial to keep the environment within the ear canal unfavourable for the growth of *Pseudomonas*. This requires control of inflammation (topical corticosteroids as well as addressing any underlying diseases) and keeping the ear canal clean and dry[16].

SURGICAL TREATMENT

Total ear canal ablation with lateral bulla osteotomy is indicated for end stage ear canals with hyperplastic epithelium, hypertrophic glands, collapsed and/or stenotic horizontal ear canals, calcified peri-auricular soft tissue, ruptured tympanic membranes and concurrent otitis media. This surgical procedure removes the entire lining of the external ear canal and tympanic bulla, and provides a definitive cure for these animals. It is a complicated procedure with serious potential complications and should be performed by experienced surgeons. Lateral ear resection (Zepps) is rarely successful and is no longer indicated[14].

FOLLOW UP

Successful management of any case of OE is strongly linked to regular follow-up visits, initially every 2–3 weeks until the OE has been resolved, or a maintenance plan has been established. Otoscopy and cytology should be performed at each follow up visit. Otoscopy should reveal a clean ear canal and clearly visible TM. Client compliance and ear cleaning techniques can be evaluated at the same time. Topical therapy is continued for 2 weeks after no more micro-organisms and inflammation (neutrophils, purple staining strands of nuclear material) are seen on cytology. Owners should be warned that OE often recurs and that further investigation into causes and factors will be required if this occurs. Ongoing ear cleaning is beneficial to prevent further accumulation of cerumen and reduces the likelihood of reinfection. It is important to not repeatedly prescribe further topical therapy for subsequent episodes of OE even though clients may be insistent. This will encourage the formation of resistant organisms and is likely to result in the development of chronic disease[19, 21].

REASONS FOR TREATMENT FAILURE AND POOR COMPLIANCE

These include not cleaning appropriately, not treating long enough, not resolving otitis media, proliferate and end-stage ears, poor owner compliance and communication, inappropriate antibiotics, failure to address the PSPP causes and factors, poor patient compliance and failure to maintain chronic cases[19, 21].

Improved owner compliance
This may be achieved by using once daily and/or palatable medication, using drugs that the owner is able to and wants to administer, convincing the owner of the importance of correct treatment, giving written instructions, demonstrating how to administer topical therapy and how to clean ears, good follow up and communication, minimising the number of different drugs or treatments, using analgesia to facilitate cleaning and topical medication, recommending regular revisits to assess the ear disease and emphasising that management and control is the aim, not usually cure of chronic otitis[19, 21].

CONCLUSION

It is essential to address all PSPP causes and factors when managing otitis externa, especially chronic cases. Where some of these cannot be treated, ongoing management will be necessary. Veterinarians should discuss this with the owners and make them aware that relapses can occur. Early aggressive treatment of primary and secondary causes and proactive treatment of chronic cases is crucial. Biofilms are important clinical components, which should be diagnosed and treated. It is important to check a patient's ears regularly, with otoscopy and cytology performed at every follow up visit. It is very important to keep ears clean. With routine cleaning at home, clients will be able to assess the ears regularly and notice relapses early, when they are more easily treated. Successful management depends on good communication with the owner.

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INVESTIGATION AND TREATMENT OF CANINE ATOPY

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ABSTRACT

Canine Atopic Dermatitis (AD) has been defined as a genetically predisposed inflammatory and pruritic allergic skin disease with characteristic clinical features. It is associated most commonly with IgE antibodies to environmental allergens. The disease has no pathognomonic clinical signs that permit a definitive diagnosis to be made upon initial owner interview and clinical examination. A definitive diagnosis can thus be challenging to make. A set of practical guidelines that can be used to assist practitioners in the diagnosis of canine AD have been published. It is imperative that initial investigations be aimed at ruling out of other skin conditions with clinical signs that can resemble, or overlap with canine AD.

RULING OUT OF OTHER SKIN CONDITIONS WITH CLINICAL SIGNS THAT CAN RESEMBLE, OR OVERLAP WITH, CANINE AD

The evaluation of a pruritic dog requires a step-by-step thought-process and approach that should lead to a definitive diagnosis. The differential diagnoses and role of complicating factors (Table 1) need to be narrowed down using information derived from the history, the findings on physical examination, diagnostic tests (where necessary), and response to treatment. Basic sampling methods and diagnostic tests, which may be required to rule out most of the common differentials are flea combing, skin scraping, hair plucking and cytological examination of skin and ear samples. Depending on the complexity of the case, the following steps may be performed over a series of visits, or all at once.

Table 1: Important differential diagnoses for pruritic skin diseases in dogs

Ectoparasitic skin diseases
- Fleas
- Scabies (*Sarcoptes scabiei*)
- Demodicosis
- Cheyletiellosis
- Otoacariasis (*Otodectes cynotis*)

Microbial skin infections
- Staphylococcal pyoderma
- Malassezia dermatitis

Allergic skin diseases
- Flea allergy dermatitis
- Atopic dermatitis
- Food intolerance/allergy
- Insect bite hypersensitivity
- Contact dermatitis

Neoplastic disease
- Cutaneous lymphoma

STEP 1 – CONSIDER THE POSSIBILITY OF FLEAS

While the clinical signs in a dog with flea infestation are variable, the location of skin lesions and pruritus associated with flea allergy dermatitis (FAD) are most commonly found at the lumbosacral area, tail base and caudomedial thighs. A flea infestation is associated with increased flea counts, whereas in dogs with FAD this may not be the case. In addition, clinicians must be aware that many atopic dogs may suffer from concurrent FAD, which may complicate the clinical diagnosis. To exclude

FAD or flea infestation as a possible cause of pruritus in a particular case, clinicians should apply the following guidelines:

- The prevalence of fleas and associated hypersensitivities depends on the geographical area in which the animal lives.
- In dogs with pruritus and/or lesions in areas of the body that are not primarily affected by fleas (e.g., the paws or ear canals), FAD may not be the sole cause of pruritus.
- Clinicians should check all pruritic dogs for fleas or flea faeces on direct examination or brushing the hair coat (flea combing). To exclude FAD when fleas or flea faeces cannot be found, an effective flea control program should be initiated.

STEP 2 – CONSIDER THE POSSIBILITY OF OTHER ECTOPARASITES

Besides fleas, other ectoparasites may be associated with pruritus (e.g., sarcoptic mange, cheyletiellosis, otoacariasis) or can be found as a concurrent disease (e.g., demodicosis). Although the majority of these parasites favour specific body areas, they can be difficult to distinguish clinically. Prior to an allergy investigation, every attempt should be made to rule out potential ectoparasitic skin diseases. Various sampling methods such as skin scraping, hair combing, hair plucking, ear swabbing, and acetate tape impressions can be used to collect specimens.

STEP 3 – CONSIDER THE POSSIBILITY OF STAPHYLOCOCCAL INFECTION AND *MALASSEZIA* OVERGROWTH

Pyoderma
Bacterial skin infections caused by *Staphylococcus pseudintermedius* (SP) are common in dogs with AD. The typical lesions of superficial pyoderma, such as papulopustular eruption and epidermal collarettes, are often distinctive enough to make a clinical diagnosis on gross appearance alone. However, the initial diagnosis should be confirmed by examining cytological samples. Samples from pricked pustules will most likely yield definitive results, while samples from papules and epidermal collarettes may be less rewarding.

Staphylococcal pyoderma is in most cases a secondary problem associated with underlying pruritic and non-pruritic diseases such as canine AD, but also other allergies as well as endocrinopathies. The pyoderma often causes a change in the overall level or distribution pattern of the pruritus. In these cases, eliminating the pyoderma will determine if the primary disease is itself pruritic, and what its severity and distribution pattern may be.

***Malassezia* dermatitis**
The most effective diagnostic test for the identification of *Malassezia* organisms is skin cytology from affected areas such as skin folds, areas with lichenification and oily seborrhoea. In general, clinical signs associated with the cytological presence of yeasts reflect a yeast overgrowth or infection. However, in dogs with hypersensitivity, few organisms may elicit pruritus and associated skin lesions. For this reason, a diagnosis of *Malassezia* dermatitis should be based on the clinical and cytological findings and confirmed by a response to antifungal therapy

STEP 4 – CONSIDER THE ROLE OF CUTANEOUS ADVERSE FOOD REACTION (CAFR)

Food related pruritus can be caused by two different mechanisms, one, a non-immune mediated reaction (food intolerance), the other an immune mediated reaction which includes IgE-mediated hypersensitivity (food allergy). Because reactions to food components can present clinically as canine AD, or serve as a flare factor in canine AD, dogs with CAFR may be indistinguishable clinically from canine AD. The presence of gastrointestinal signs, such as diarrhoea, vomiting, tenesmus, soft stools, flatulence, and increased number of bowel movements is more typically seen with food-induced canine AD. In any canine AD case that has year-round clinical signs, CAFR can only be ruled out by effective strict elimination diet trials, since accurate diagnostic commercial tests are not currently available. Unfortunately, there are no diets that have been shown to be effective in all cases of CAFR. Therefore, in some cases, especially when gastrointestinal signs are present, multiple different diet trials may be needed until a sufficient control of the clinical signs has been achieved.

Ideally an elimination diet trial should be performed with a diet to which ingredients the dog has never been exposed before. Unfortunately, most commercially available diets contain a wide range of

ingredients and by-products, making the selection of an appropriate diet difficult. Most over the counter diets as well as some prescription elimination diets may be contaminated with traces of other food components. Although hydrolysed diets are offered as an alternative option, the protein source is based on either chicken or soy. For this reason some dogs allergic to chicken and/or soy may not respond to such diets. The most common food allergens in dogs are: beef, dairy, chicken products and wheat, and to a lower degree soy, lamb, pork, fish, and maize.

A diet trial is performed by instituting a strict trial with a diet containing commercial or home-cooked novel (e.g., rabbit, kangaroo, venison, horse, etc.) or hydrolysed protein ingredients. Any strict elimination diet trial should be fed exclusively for a minimum of 8 weeks to achieve complete clinical remission in most cases. If the condition improves, the diet should be continued to determine if there is complete or only partial control of the clinical signs. If a dog is not responding to a commercial elimination diet a second attempt with a home-cooked diet should be performed. Home-cooked diets are considered the most limited ingredient diets if done properly. All diet trials should be continued until the veterinarian examines the dog. This is important as some owners may not recognize a partial response or be aware of lesions still present when a dog appears to have improved. Dietary involvement is confirmed if there is a relapse of clinical disease when the original diet is re-introduced. Clinicians should be aware that poor owner/patient compliance is a common problem. Typical pitfalls during a diet trial are: feeding table food, raw hides, treats, “hiding” medication in food, using flavoured tooth paste, giving medication in gelatine capsules, using flavoured drugs (e.g., NSAIDs, antibiotics, chewable heartworm or flea preventative), and dogs eating other animals’ faeces. Clients need to realize that very small amounts of other foods or food additives ingested, even intermittently, can prevent a favourable response. Crumbs on the floor and even licking another pet’ s empty bowl may result in a poor outcome. The client’ s job is to make sure the dog ingests nothing but the prescribed diet and water.

Once steps 1– 4 of the diagnostic work-up has been completed, a clinical diagnosis of canine AD should be considered if the pruritus is still present.

DETAILED INTERPRETATION OF THE HISTORICAL AND CLINICAL FEATURES OF CANINE AD

The initial clinical feature of canine AD is pruritus, which can include scratching, rubbing, chewing, excessive grooming or licking, scooting, and/or head shaking. Depending on the allergens involved, the pruritus may be seasonal (e.g., pollen) or non-seasonal (e.g., dust mites, food). At the beginning the pruritus may be alesional or associated with primary skin lesions such as erythema and occasionally papules. The face, concave aspect of the ear pinnae, ventrum, axillae, inguinal area, perineal area and distal extremities are most commonly affected in canine AD, but breed associated variations of body sites affected by canine AD have been identified (Table 3). In more chronic stages secondary skin lesions will occur due to self-trauma, chronic inflammation and secondary infections. Typical secondary skin lesions are excoriations, alopecia, lichenification, hyperpigmentation, crusting, and seborrhoea.

A new tool to assist with the interpretation of the clinical findings when confronted with a pruritic dog is application of clinical criteria known as “Favrot’ s criteria” (Table 2). These include a set of criteria that have been developed from a large case series of confirmed cases of canine AD. The use of complex statistical analysis allowed a set of clinical features to be identified that had maximum association with canine AD. The analysis revealed two sets of criteria, which yield varying levels of sensitivity and specificity for the condition. Clinicians can use whichever set best serves their needs. For example, use of a set of criteria that yields the highest specificity is more likely to ensure that a particular case actually has canine AD. However, this set would exclude some pruritic dogs that were suffering from the disease. A set yielding the highest sensitivity is more likely to capture cases of canine AD, but it could allow some dogs with other conditions to be classified as atopic when in fact they were not. Further guidance about application of these criteria sets is shown in Table 2.

MANAGEMENT OF CANINE ATOPIC DISEASE

Practicing in South Africa we face serious limitations because of the agents available. Following a proper work up (including the failure of a food trial), I will generally discuss all of the following options with owners as a means of trying to achieve lifelong control of the pruritus:

Derm Defense
This is a helpful, side effect free way of trying to manage the disease. Many owners will opt this as their first choice and at least give it a try before resorting to pharmacological means of control. It should be combined with shampoo therapy. Many dogs respond favourably to its use. It may reduce the requirement and/or dose for/of systemic drugs.

Corticosteroids
Not a viable long term option unless the owners give informed consent and are well counselled in regards to the negative impact this class of drugs has on dogs. Depo steroid injections have no place in long-term management of pruritus in dogs.

Topical cortisone (eg. Cortavance Spray, otic mixtures containing cortisone used a few times a week) can play an important and helpful role is spot treatment of especially hairless areas of the body surface that the dog targets.

Short-term oral prednisolone may be used to control flare-ups of pruritic disease previously stable/well controlled. Typically these short protocols look something like this: 1mg/kg at 12-hour intervals for the first day; 1mg/kg once the next day; 1mg/kg once after skipping a day. There should 3-5 days rest between these cycles. This is an acceptable way of getting a very pruritic dog through a diet trial or flea exclusion trial but is generally not an acceptable way of life-long control. Remember, essential fatty acids have a helpful adjunctive role to play in long-term management. Antihistamines have a very inconsistent effect in dogs.

Cyclosporine
Safe and effective in cases that are confirmed atopics. Owners need to be well directed in its use. Although effective, the drug has a slow onset of action (3 weeks) and as such it is not the sort of drug that can be used as an on-off switch to control flares. It is only used in cases that need on-going and continuous pharmacological control of pruritus. Side effects are not uncommon in the first 3 weeks of use and some dogs (although few) never tolerate it. It is expensive and seldom a consideration in dogs over 15 kg.

ASIT
Safe and effective in around 60% of confirmed atopics (following the failure of a hydrolysed protein food trial). It may take many months for benefit to be observed. It is cheaper than cyclosporine but nevertheless price may be an issue for some owners. In RSA the vaccines are made up based on in vitro serum testing, as intradermal testing is not practical.

Shampoo therapy
No atopic dog should be without chronic shampoo as part of its management protocol. This manages the surface carriage of the allergen and reduces the surface carriage of pathogens. Initially, in skins that are proven to be secondarily infected, a chlorhexidine-containing product (eg. Pyoderm) would be a good choice. Longer term a maintenance product should have an antimicrobial effect as well as an effect on surface irritation.

Therapies not yet available in South Africa:
Oclacitinib (Apoquel). Hopefully this drug will be available by 2019 and Cytopoint (monoclonal Ab against IL-31).

Table 2. A new tool to assist with the interpretation of the clinical findings when confronted with a pruritic dog is application of clinical criteria known as “Favrot’ s criteria”. These include a set of criteria that have been developed from a large case series of confirmed cases of canine AD.

		Use	**Reliability**
	Set 1	High specificity – fulfil 6 criteria High sensitivity – 5 criteria should be fulfilled	5 criteria Sens 85.4% Spec 79.1
1	Age of onset<3 years		
2	Mostly indoor		
3	Corticosteroid-response pruritus		
4	Chronic or recurrent yeast infections		
5	Affected front feet		
6	Affected ear pinnae		
7	Non-affected ear margins		
8	Non-affected dorso-lumbar area		
	Set 2		5 criteria Sens 77.2% Spec 83%
1	Age of onset<3 years		
2	Mostly indoor		
3	Alesional pruritus at onset		
4	Affected front feet		
5	Affected ear pinnae		
6	Non-affected ear margins		
7	Non-affected dorso-lumbar area		

A QUICK OVERVIEW OF AUTOIMMUNE SKIN DISEASE OF THE DOG AND CAT

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DISEASE OF THE EPIDERMIS

Pemphigus foliaceus

Incidence and Prevalence

There is very little epidemiological data on Pf. In one study at Cornell in the USA the incidence in a dermatology referral clinic was 3 cases per 1000 dogs seen. In another study at Michigan State University Pf accounted for 1% of skin biopsy specimens submitted. In another study of canine autoimmune skin disease, Pf accounted for a third of the cases seen with autoimmune skin disease.

Signalment

Genetic factors are likely to play a role in the disease, as there are definite breed predispositions. Breeds with an increased incidence in published work include bearded collie, Akita, Newfoundland, Schipperke, Doberman, chow, Shar-Pei and dachshund. In some regions chows and Akitas have a significantly higher incidence than other breeds. There is no sex predilection. The age of onset is most commonly between about 4 and 6 years but the range is from less than 1 to 16. Sunlight exposure is an environmental factor that induces disease flare.

Clinical signs

In most dogs, lesions initially appear on the face, principally on the dorsal muzzle, planum nasale, periocular skin and ears. In these areas, the pattern usually is strikingly bilateral and symmetrical. In the largest case series, lesions were restricted to the face in 15 of 91 dogs (16%). In rare canine patients, the dermatosis exhibits a generalized distribution from the onset, but in most cases, lesions will develop towards regionalization or generalization over 3 to 12 months. In the largest retrospective study, generalized skin lesions were present in 60/91 dogs (66%), and in these dogs, crusts were most prevalent on the trunk (58%). A remarkable finding of canine PF is the predilection of lesions for the footpads. Indeed, footpad involvement is seen in one third of dogs with PF, and rare canine patients exhibit lesions restricted solely to this location. Of note is that mucosal lesions are only rarely seen in dogs with PF (2/91; 2%).

The nature of skin lesions is comparable in most dogs with PF: transient pustules evolve rapidly into erosions and crusts, the latter being the lesions most commonly seen. The pustules of PF are usually large and confluent, and they often exhibit polycyclic borders. Many hair shafts can protrude from these pustules, in contrast to the lesions of bacterial folliculitis where only a single hair can be seen coming from each pustule. Alopecia and generalized exfoliative erythroderma are seen occasionally. Pruritus is present in one fourth to one half of dogs with PF, whereas systemic symptoms consisting of anorexia, depression, fever and weight loss are encountered usually in dogs with widespread erosive lesions

The clinical signs of feline PF are reminiscent of those seen in dogs with this disease. Pustules are extremely transient, however, and the phenotype is dominated by erosions and yellowish crusts on the face, ears, and on the feet. Pedal lesions, consisting of suppuration or crusts, can be seen on or around the footpads or ungual folds of claws. The phenotype of feline PF is usually mild and fairly localized, but generalized lesions can be seen also. The distribution of lesions of feline PF is usually bilateral and symmetrical.

Cytology

Classic cytology smears prepared from intact pustules or from the underside of crusts demonstrates acantholytic keratinocytes. Isolated to clustered (e.g. ‘rafts’) free-floating rounded keratinocytes admixed with non-degenerated neutrophils and rarer eosinophils are typically seen. The presence of acantholytic keratinocytes and neutrophils is not specific of PF, however. Similar microscopically findings have been found in canine and equine cases of pustular dermatophytosis, a PF mimicker in which *Trichophyton* fungi invade the stratum corneum and induce subcorneal acantholytic neutrophilic pustules. Keratinocyte acantholysis has also been reported in dogs with bacterial skin infections.

Diagnosis
The diagnosis of PF may be given to animals that suffer from a skin disease satisfying ALL three criteria below:

- Clinical examination: pustules rapidly evolving in shallow erosions and crusts with predominance to the face and feet
- Histopathology: superficial epidermal or follicular pustules rich in neutrophils and often-clustered acantholytic keratinocytes
- Differential diagnoses: rule out of other acantholytic neutrophilic pustular diseases, especially exfoliation associated staphylococcal pyoderma and pustular dermatophytosis due to corneophilic dermatophytes.

Treatment and outcome
Immune suppression remains the initial therapeutic intervention of choice for Pf in dogs and cats. Of note is that all recommendations for immunosuppression initially were based on interventions used for treatment of the human disease, and prospective clinical trials were not performed to optimize protocols for animals. Historically, standard-of-care treatment of canine Pf relied on the induction of immunosuppression with oral glucocorticoids, such as prednisone or prednisolone, at daily dosages varying from 2 to 6.6 mg/kg divided once or twice daily. If lesions decreased in extent and severity with this regimen, then the dose and/or administration frequency of glucocorticoids was reduced with the goal of rapidly achieving alternate day intake. In many canine patients, however, glucocorticoid therapy alone appears incapable in halting or slowing the progression of skin lesions. In these cases, cytotoxic drugs are usually added. Azathioprine (2–2.5 mg/kg, orally, once daily), cyclophosphamide (25 mg/m^2 body surface area, orally, once daily) or chlorambucil (0.2 mg/kg every 24–48 h) have been proposed as adjunct cytotoxic drugs. Finally, rare dogs with Pf exhibit lesions that respond to tetracycline and niacinamide (250–500 mg of each, three times daily).

In cats with PF, glucocorticoid monotherapy usually is effective for achieving clinical remission. Historically, the oral glucocorticoids of choice have been prednisone (4– 5 mg/kg daily) and triamcinolone (0.6–2 mg kg−1 daily). Recent pharmacological data, however, have shown that oral prednisone is not very well absorbed and/or converted into prednisolone in cats, therefore suggesting that oral prednisolone is a better choice than prednisone in this species. In cats whose Pf lesions fail to respond to glucocorticoids, chlorambucil (0.2 mg/kg, orally, once daily) is the cytotoxic drug most commonly used. Even though azathioprine has been used successfully to treat cats with PF this drug is no longer used widely as it is known to cause profound neutropenia and thrombocytopenia in feline patients. In one study, complete remission of Pf lesions occurred in 15 of 15 cats (100%) using triamcinolone alone, in eight of 13 (62%) using prednisone alone, and in nine of 11 (82%) using a prednisone/chlorambucil combination.

BULLOUS AND ACANTHOLYTIC DISEASES OF THE EPIDERMIS AND DERMAL-EPIDERMAL JUNCTION

Bullous pemphigoid
This is a very rare vesiculobullous subepithelial autoimmune skin disease of the dog and cat. Since the lesion is sub-epidermal, intact vesicles and bullae are seen more commonly than with other bullous autoimmunities such as pemphigus vulgaris in which the intra-epidermal lesions are much more fragile. Dogs and cats of any age may be affected and there appears to be a genetic factor involved in pathogenesis.

Clinical signs of BP overall appear similar in dogs and cats. Lesions consist primarily of turgid vesicles, erosions, ulcers, and crusts. In dogs, the phenotype is usually mild as skin lesions can be seen, in grouped arrangement, on the concave ear pinnae, abdomen, axillae or mucocutaneous junctions. Oral vesicles and erosions have been seen in 3/5 dogs with BP. In cats, few lesions usually are present. Erosions predominate on the face and oral injuries consistently are observed.

The first step in the diagnosis of BP in animals involves histopathological examination of lesional skin biopsy specimens. In dogs with BP, subepidermal accumulation of neutrophil and eosinophil granulocytes is the earliest microscopic lesion seen. Inflammatory subepidermal vesicles form secondarily. The most important differentials include pemphigus vulgaris, EM and drug eruptions.

Pemphigus vulgaris is a more sever disease and oral lesions are much more common in this disease compared with bullous pemphigoid.

Predisposed breeds include the Doberman, Collie and Dachshund.

Too few cases of BP in animals have been diagnosed to provide ample information about the natural progression of the disease and its response to therapy. Nevertheless, long-term remission of BP has been seen in two dogs and one cat following short courses of corticosteroids. Some canine patients reportedly respond to a niacinamide tetracycline
combination regimen These anecdotal findings suggest that BP lesions usually respond to treatment and can exhibit a self-limiting behaviour.

Pemphigus vulgaris
PV is one of the rarest autoimmune blistering skin diseases of dogs; it can occur at any age with some breeds appearing to be predisposed (e.g. German shepherd dogs, collies), and males outnumbering females. In most dogs, lesions first develop in the oral cavity or at mucocutaneous junctions, and they will usually spread in extent and severity from a mucosal-predominant (mouth, ears, nose, periocular and anogenital) to a mucosal-and-cutaneous phenotype. On haired skin, lesions develop over pressure points and at intertriginous areas secondary to friction. In most dogs with PV, primary lesions consist of flaccid transient vesicles that evolve rapidly into irregular erosions with epidermal sloughing. Direct Nikolskiy's sign is usually positive. Dogs with severe disease exhibit systemic signs such as lethargy, anorexia and ensuing weight loss. Milder 'atypical' variants of canine PV appear to exist. The histopathology of canine and feline PV is characteristic: there is initial widening of intercellular spaces around basal cell keratinocytes that – possibly secondary to friction – rapidly evolves into suprabasal epidermal clefts without inflammation. In 40% of dogs with PV, a fatal outcome has been reported; they either died spontaneously or were euthanized at their owner's request. Nevertheless, partial or complete remission can be induced with immunosuppressive regimens as described for canine PF.

Paraneoplastic pemphigus
There are only 3 cases of para-neoplastic pemphigus reported in the literature. These were characterised by flaccid vesicles, erosions and ulcerations in the oral cavity, nose and vulva in one case and by similar lesions on all mucosae, mucocutaneous junctions as well as in haired skin in the others. All three dogs were systemically ill. Biopsies revealed supra-basal epithelial acantholysis typical of pemphigus vulgaris as well as some features of erythema multiforme. Two of the cases also showed a lichenoid reaction. All three dogs died and at necropsy a thymoma, metastatic thymic lymphoma and undifferentiated sarcoma were presumed to be the basis of the skin disease.

INTERFACE DISEASES OF THE DERMAL-EPIDERMAL JUNCTION

Discoid lupus erythematous
Discoid lupus erythematous (DLE) is an immune-mediated disease with lesions often localized to the nasal planum and dorsal muzzle and, less commonly, the pinnae, periocular skin, and lips. Discoid lupus erythematous is a relatively benign cutaneous disease with no systemic involvement, and it is the second most common immune mediated dermatitis of the dog. The initial presentation of DLE may be characterized by erythema, depigmentation, and scale with loss of normal appearance and texture of the nasal planum that frequently progresses to erosion or ulceration and crust, and may extend onto the dorsal muzzle. Lesions may be exacerbated by sun exposure and may have a waxing and waning course. In the early phases, sun avoidance, topical sunscreens, topical glucocorticoids, and systemic vitamin E and omega-3/omega-6 fatty acids may be helpful in treating these diseases. Commonly used therapy for progressive cases includes the addition of tetracycline and niacinamide. The author has had some success with topical Cortavance application. For more severe or refractory cases, oral glucocorticoids may be needed in addition to other systemic immunosuppressive drugs such as azathioprine or chlorambucil. While vitamin E, tetracycline, and niacinamide have minimal long-term adverse effects, their success may be more limited or may require frequent oral dosing (e.g., *tid* in the case of tetracycline/niacinamide) for optimum results. Glucocorticoids, azathioprine, and chlorambucil are generally more successful in managing these cases, but they may be associated with systemic toxicity, potential long-term adverse effects, and must be monitored closely; therefore, they should be used with caution. Topical therapies for localized lesions of DLE and PE have, to date, consisted primarily of medium- to high-potency corticosteroids and may be plagued by

limited success, epidermal atrophy, and potential adverse effects from systemic absorption. Topical cyclosporine has been used as an adjunctive therapy for localized lesions of DLE and PE with anecdotal reports of benefit. In one study of 10 dogs with DLE topical 0.1% Tacrolimus was used for treatment of localized lesions either as a sole therapy (n=2) or as an adjunctive treatment (n=10). Eight of 10 dogs with DLE were improved following 8 weeks of topical application. In six of the eight dogs that improved, other medications were discontinued. No adverse effects in clinical or laboratory parameters were noted throughout the study.

Erythema multiforme
EM is an acute reaction pattern of the skin and mucous membranes characterised by distinctive clinical and histological findings. A wide range of triggering factors may elicit it and the pathogenesis is incompletely understood. It is hypothesised that it represents a host-specific T-cell mediated hypersensitivity reaction directed towards various antigens (especially drugs and infectious agents).

The cutaneous eruption of EM occurs rapidly and lesions are approximately symmetrical. Early lesions are annular, erythematous macules and papules and plaques. These lesions enlarge centrifugally and often coalesce to form bizarre polycyclic patterns. The centre of the lesion may then become cyanotic and haemorrhagic. These have become known as the classic target or doughnut lesions. These lesions may also become vesicular or bullous with a necrotic epidermis sloughing to leave an erythematous oozing erosion/ulcer. Mucosal lesions have a similar onset and symmetry but are mostly vesicular/bullous and ulcerative. The ulcers are often haemorrhagic and coated by a greyish-white necrotic pseudomembrane of epithelium and fibrin.

Lesions typically involve the ventrum (especially the axillae and groin) and multiple muco-cutaneous junctions, the oral cavity, pinnae, and foot pads. Lesions may be mildly to moderately painful. Pitting oedema may be present on the distal limbs of some dogs and some dogs will show systemic signs of illness (anorexia, fever, depression). Cats are rarely affected but the few described cases had ear and facial lesions as well as involvement of the lips, paws and target lesions on the ventral abdomen.

Drugs are the most common presumed trigger and usually result in lesions developing within 3 weeks of initiating treatment with the offending agent. Commonly involved drugs include trimethoprim-sulpha, chloramphenicol, amoxicillin and amoxicillin-clavulanate. Dietary protein (beef and soy) have been associated with EM. Infectious agents associated with EM include *Pseudomonas* otitis externa and Staphylococcal folliculitis.

Lupoid onichitis
Lupoid onychodystrophy is a lupus like disease that causes onychomadesis (claw loss). It is uncommon in dogs, with the highest incidence in young adults and middle aged animals. The disease has an acute onset of nail loss. Initially only a few claws are affected but with time all claws are symmetrically affected. Typically the nails are the only affected part of the animal. They are brittle, deformed, and may fall off exposing a painful bed. The nail beds may become secondary infected and are often sufficiently painful to cause lameness.

Treat any secondary infections in the first instance. Essential fatty acids dosed orally for 3 - 6 months have proven to be effective for lupoid. If efa's are ineffective Vit. E (200-400 IU PO q 12 h) may be effective. Tetracycline and niacinamide combination may also be used (<15 kg use 250mg of both agents PO q 8 h; >15 kg use 500mg per dog the same way). Improvements should be seen within 6 months. Long term steroids usage will also result in resolution in very refractory cases. In a disease that is usually responsive to efa's is however hard to justify chronic glucocorticoid usage. Prognosis for nail regrowth is good and some dogs can be taken off all treatment after 6 months whereas others will need some form of life long management.

NECROTIZING DISEASES OF THE EPIDERMIS

Erythema multiforme
See above.

Toxic epidermal necrolysis
This is a rare life-threatening disease that is hard to separate from EM at times (although in humans they are seen as separate entities). Triggers described include drugs, bacterial infections, parvovirus,

food, neoplasia and possibly herpesvirus. Severe cases of EM and TEN are where distinct differentiation of these disease becomes muddied as both of these diseases are rare and present with severe skin signs (involving several mucosal surfaces and systemic malaise.

TEN is associated with extensive vesiculo-bullous and ulcerative lesions of the skin and oral mucosa. The disease is acute and the animal is systemically ill. The classic target lesions described for EM are not present. The Nikolsky sign is often positive and even routine touching or handling of the skin may result in epidermal sloughing. Any body surface may be involved but the oral mucosa and footpads are commonly affected. Other mucosal surfaces (rectal, oesophageal, conjunctival, and tracheal) may be involved. Haemorrhagic diarrhoea may be seen. Cutaneous pain is usually moderate to marked.

Differentials include burns, severe EM and vasculitis. Diagnosis is based on the progressive history, physical examination and biopsy. Histologically the key feature is full thickness devitalisation of the epidermis with minimal dermal inflammation.

TEN is associated with significant mortality (30 – 50%). Sepsis is a leading cause of death in humans. The greater the body surface area affected, the poorer to prognosis (akin to massive second degree burns).

Treatment should initially be aimed at (1) stopping any suspected drug trigger, (2) fluid and electrolyte correction, (3) prevention of infection. There is insufficient evidence for the usefulness of immunosuppression. Despite this, cyclosporine has been suggested, as has the use of intravenous immunoglobulin. Recovery is slow, taking weeks if it is does occur.

RECOGNIZING ENDOCRINE DISEASE

Remo Lobetti

Endocrine conditions are not uncommon clinical entities in small animal practice that affect both dogs and cats, with clinical signs being a combination of PuPd, polyphagia, obesity or weight loss, and alopecia. Common endocrine disorders in the dog are Cushing's disease, Addison's disease, diabetes mellitus, and hypothyroidism, whereas diabetes mellitus and hyperthyroid are common in the cat. Rare endocrinopathies include feline Cushing's disease, hyperaldosteronism, diabetes insipidus, acromegaly, hypopituitarism, and phaeochromocytoma. Other conditions that may have an underlying endocrine anomaly include hypoglycaemia (insulinoma), hypercalcaemia (hyperparathyroidism), and hypocalcaemia (hypoparathyroidism).

INTERPRETATION OF ENDOCRINE TESTS

Johan Schoeman

The interpretation of endocrine test results can be difficult. This is due to the dynamic nature of the endocrine responses to both internal and external factors that challenge homoeostasis. A wide range of results can thus be within the normal range. Since the fine line between normal and abnormal is often not so clear-cut, clinicians should view results within the context of a thorough understanding of normal endocrine physiology and a clear appreciation of the diagnostic performance properties of various endocrine tests. The paper will address aspects of diagnostic performance such as sensitivity and specificity; negative and positive predictive value and the effect of prevalence on these. In addition, various non-endocrine factors such as breed, age, time of day, drugs and concomitant non-endocrine diseases will be discussed. Next, the effect of other endocrine disease and their attendant pathophysiological changes, such as hyperlipidaemia and endogenous antibodies on the analytical validity of tests, will be mentioned. Finally, the effect of symptomatic or palliative therapy on the subsequent interpretation of endocrine tests will be covered.

EUTHYROID SICK SYNDROME REVISITED

Johan Schoeman

Non-thyroidal illness and certain drug therapies have a profound effect on circulating thyroid hormones. As a result, many dogs are erroneously diagnosed with hypothyroidism. Clinicians should be acutely aware of this potentially serious misdiagnosis, which can result in long periods of unnecessary thyroid replacement therapy and great difficulty in confirming the diagnosis after the thyroid gland has been iatrogenically suppressed by such therapy. The causes of this reduction in thyroid hormones by non-thyroidal illness, which is termed, euthyroid sick syndrome (ESS) are multifactorial. These include: glucocorticoids and circulating cytokines, such as interleukins and tumour necrosis factor. They act on the hypothalamic-hypophyseal-thyroid axis and, *inter alia*, can cause TSH suppression, decreased intrinsic thyroid hormone activity, reduce thyroid hormone protein-binding and altered peripheral hormone conversion and metabolism. This paper will address some of the common causes, recently elucidated pathomechanism and the extent and longitudinal duration of some of these changes, especially as they manifest in severe inflammatory disease.

FELINE HYPERTHYROIDISM

Joanne McLean

Feline hyperthyroidism is a metabolic disease of middle-aged to older cats that has shown a marked increase in its worldwide prevalence within the last three decades. This disorder is now recognised as the most common feline endocrinopathy in many countries. Since the first clinical reports of the disease appeared in the literature in 1979, our understanding of the disease has evolved tremendously. Initially, it was a disease that only referral clinicians treated, but is now a clinical entity that most primary clinicians routinely manage. Inclusion of the measurement of tT_4 concentration in senior wellness panels, as well as in diagnostic work-ups for sick older cats, now enables diagnosis of the condition long before patients demonstrate the classic clinical signs associated with disease. However, earlier recognition of the problem has given rise to several related questions: how to recognise the health significance of the early presentations of the disease; how early to treat the disease; whether to treat when comorbid conditions are present; and how to manage comorbid conditions such as chronic kidney disease and cardiac disease. After establishing a diagnosis, the clinician and client are then also faced with choosing the most appropriate treatment option. The choice of therapy often depends on factors such as the cat's age, comorbidities, treatment cost, availability of treatment options, and the clinician's recommendation and expertise. The goal of therapy is to restore euthyroidism, avoid hypothyroidism and minimise side effects of treatment. As a result of better awareness of the disease, routine screening tests and a variety of readily available treatment options, the hyperthyroid cat will however often live for an extended period (2-4 years) in appropriately managed cases.

UPDATE ON ADDISON'S DISEASE

Johan Schoeman

Hypoadrenocorticism (HA or Addison's disease) is an uncommon condition in dogs, characterized by a severe deficiency in adrenocortical hormone secretion. Most dogs have immune-mediated destruction of adrenocortical tissue (primary HA), yet the disorder may also develop because of dysfunction of any part of the hypothalamic-pituitary-adrenal axis (secondary and tertiary HA). Besides, at least 90% of the adrenal cortex needs to be non-functional before associated clinical signs are observed. The historical, clinical and biochemical changes in this disorder have been well-described. Lately, a number of papers have been published on the atypical form of this disease, in which destruction of part of the adrenal cortex is postulated. As a result of this, diagnostic testing had to be refined and clinicians are now required to increase their index of suspicion for this form of the disease. Moreover, and as a direct consequence of the advances in diagnostic testing and monitoring, new treatment regimens have also been developed. Therefore, this paper will focus on the recent advances in the diagnosis and treatment of Addison's disease in the dog.

MANAGEMENT OF CANINE CUSHING'S DISEASE

Varaidzo Mukorera

Canine hyperadrenocorticism (HAC) is a disease resulting from overproduction of cortisol. Overproduction of cortisol in dogs is mostly via excessive production of ACTH from the pituitary gland and less commonly from excessive cortisol derived from overproduction by the adrenal glands. The management options for canine HAC include medical therapy with a variety of drugs, surgery to remove either the pituitary gland or affected adrenal gland, radiation of the pituitary gland or some combination of these. The most commonly used drugs for the management of HAC are trilostane and mitotane. The current gold standard for monitoring medical treatment is the adrenosortical stimulation test.

DIABETIC KETOACIDOSIS AND HYPERGLYCEMIC HYPEROSMOLAR SYNDROME

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Diabetic ketoacidosis **(**DKA) is characterized by hyperglycaemia, glycosuria, and ketonaemia or ketonuria with a metabolic acidosis. Hyperglycaemic, hyperosmolar syndrome (HHS) is characterized by severe hyperglycaemia (>600 mg/dl), minimal or absent urine ketones, and serum osmolality > 350 mosm/kg. Both types of crisis can develop in a diabetic animal subsequent to increases in diabetogenic hormones that occur in response to an underlying stressor, such as concurrent disease. Treatment for both crises starts with fluid therapy, which will replace vascular volume and reduce blood glucose via dilution and increased renal losses. Abnormalities in potassium, magnesium, and phosphorus are common and any deficiencies must be resolved before initiating insulin therapy. The goal of insulin therapy is to reduce blood glucose by 50-70 mg/dL/hr. Insulin is vital to reversing ketone production and acidaemia in DKA. Because of the importance of GFR on pathogenesis, insulin is less important to reverse HHS.

FLUID THERAPY

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Fluids are a double edged sword. Too much and too little are both detrimental to health. Unfortunately there is no perfect tool to assess fluid therapy and careful assessment and regular reassessment is required. We have a large choice of fluids and the perfect fluid does not exist. This talk will address the controversies of fluid therapy and attempt to give rational advice and guidance on how we should use fluids. It important that fluids are given on a goal directed approach. Fluids should be given to achieve a resuscitation end point not just a particular volume. This approach along with cardiovascular support is vital for successful management of patients

SEPSIS: THE SURVIVING SEPSIS CAMPAIGN AND RELEVANCE TO VETERINARY PATIENTS

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Sepsis is a systemic inflammatory response to infection. Sepsis can occur subsequent to severe infection with any type of organism at sites throughout the body. Early sepsis is characterized clinically by the hyperdynamic phase in dogs: fever, tachycardia, vasodilation, bounding pulses, and red mucous membranes. Cats do not display a hyperdynamic phase, instead manifesting pallor, hypotension, abdominal pain and often a relative bradycardia. The Human Surviving Sepsis Campaign Early and aggressive resuscitation is accomplished with fluids and pressors/inotropes, as needed, and should be completed as soon as possible after sepsis identification. Early antibiotic administration has been associated with better patient outcomes for people with septic shock. Use of corticosteroids is indicated only for treating septic patients that are still hypotensive despite full volume resuscitation and pressor/inotrope therapy. Additional therapy includes source control, and diligent nursing care and monitoring.

MONITORING ICU PATIENTS

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Monitoring comes from a Latin term “monere” meaning “to warn”. The aim of monitoring patients in the intensive care unit is to identify problems and correct them before they lead to irreversible changes that lead to the patient’s death. It is all about assessing oxygen delivery and ensuring that oxygen is available to every cell in the body. Oxygen is delivered by the cardiovascular system and taken by the lungs. This makes monitoring the cardiovascular and respiratory systems vital. Intensive care is usually an expensive undertaking and knowing the prognosis can assist us in our discussion with owners. It will also give us realistic expectations. This talk will discuss the principles behind monitoring patients requiring intensive care.

TREATMENT OF EQUINE WOUNDS

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ABSTRACT

The horse has a marked healing response to injury with early production of excessive granulation tissue, which is called “proud flesh” when it is elevated above the level of the skin. Proud flesh interferes with wound contraction and delays healing. Differences have been identified between the healing of limb and body wounds: epithelialisation and wound contraction occur slower, and contraction occurs to a lesser extent on the limbs. “Proud flesh” occurs predominantly in wounds below the hock and tarsus. The differences in healing are related to increased concentrations of the pro-fibrotic cytokine TGFβ-1 (Transforming Growth Factor Beta -1) in limb wounds and in excessive granulation tissue, and lower concentrations of the anti-fibrotic cytokine TGFβ-3 in excessive granulation tissue. Bandages applied to limb wounds also commonly cause “proud flesh”.
Wound healing occurs different in ponies with more production of TGFβ-1 and a rapid decay thereof. The result is a stronger inflammatory response in ponies with a resultant reduced granulation and increased contraction in the wounds. “Proud flesh” occurs rarely in ponies.
There is no question that a warm, moist environment enhances wound healing. Wound exudate is rich in healing properties including white blood cells, growth factors, metalloproteinase, and cytokines. Leukocyte function is enhanced and scar formation is minimised in a moist environment. Autolytic debridement of necrotic tissue is rapid in a moist environment. A balance in fluid production and removal must be maintained under an occlusive dressing to minimise tissue maceration. Bacterial colonisation will occur under an occlusive dressing, so preventive measures must be taken in minimising the load. It has been demonstrated that wound occlusion nearly doubles the rate of re-epithelialisation. Full- thickness wounds maintained in a moist environment, epithelialize in less than 15 days, whereas similar wounds exposed to air took twice as long. Finally, wounds exposed to air are more inflamed, painful, pruritic and maintain a thicker crust that delays the healing process.
Today there are literally thousands of wound care products on the market. Few have been looked at discretely in the horse. Although many equine practitioners are using some or all of the basic principles of moist wound healing, a plethora of new medications have evolved around the subject. Many of the newer dressings are designed to create a moist wound-healing environment, which allows the wound fluids and growth factors to remain in contact with wound, thus promoting autolytic debridement and accelerating wound healing. Presently it appears that no single material can produce the optimum microenvironment for all wounds or for all the stages of the wound healing process.
Great differences exist as to the most appropriate method of managing wounds. This is because there is little scientific evident to support the selection of one treatment over another. Recommendations are made based mainly on clinical experience, extrapolation from other species or research, or based on an educated speculation. It is very important to remember that although there are numerous products available for topical use, almost none have solid scientific support of their efficacy in the horse using controlled studies. These products may be efficacious but they do not have the evidence to back up their frequent claims.
The report provides a review of currently available dressings, their physical characteristics and describes their best use as it relates to the condition of the wound (clean, contaminated or infected) and the phases of wound healing.

INTRODUCTION

Wound dressings have been broadly classified as either adherent or non-adherent and absorbent or non-absorbent. Adherent dressings are frequently made from closely woven or widely open gauze, other cotton materials or wool and under most circumstances are considered passive; although a few are considered interactive. Gauze dressings are generally highly absorbent and are still used for heavily contaminated exudative wounds. Non-adherent dressings have variable absorbency and are subdivided into occlusive, semi-occlusive and biologic types. By definition, occlusive dressings are nonporous materials that have a low moisture vapour transmission. Semi-occlusive dressings are moisture and vapour permeable. Synthetic, occlusive and semi-occlusive material creates a moist

wound-healing environment and is considered interactive dressings under most circumstances. Biologic dressings can either be unprocessed natural or processed to form an acellular matrix or a plasma rich platelet gel. The biologic dressings are considered bioactive contributing not only a matrix for repair but also growth factors and cytokines to enhance the healing process.

ABSORBENT, ADHERENT AND NON-ADHERENT DRESSINGS

Many, but not all of the absorbent dressings, adhere to the wound surface affecting wound debridement. This section will focus on the types of dressing used most frequently in equine practice.

Gauze dressing

Fine and wide meshed cotton gauze has been used for many years for debridement of heavily contaminated exudative and necrotic wounds. The material allows egress of fluid and bacteria through the mesh. As the dressing is peeled off the wound fibrin, debris and necrotic tissue are removed. The material can be applied as a dry dressing when the wound fluids are copious and of a low viscosity. Application as a wet to dry dressing is most commonly used when wound fluids have a high viscosity or in the case where the wound surface is dehydrated and scabs have formed. The gauze is wetted with dilute chlorhexidine (1:40 dilution) or physiologic saline solution, excess fluid is squeezed out and the dampened dressing applied to the wound surface. When the dressing is applied wet, it is considered an interactive dressing since it has an effect of hydrating the wound surface. When the dressing dries, fibrin adheres it to the wounds surface affecting debridement. Since wound debridement with the gauze material is non-selective, the dressing is usually changed after 24 hours; the wound is lavaged with a sterile diluted antiseptic solution, which is delivered to the wound surface at a pressure of 10 - 15 PSI. If further debridement is needed another wet to dry dressing is applied. Once the wound appears clean, another dressing type is indicated. One to three applications of the wet to dry dressing are all that is needed to effectively debride most wounds. Continued use after the wound has been effectively debrided is contraindicated because of non-selective debridement and the tendency to strip off newly formed epidermis.

Hypertonic saline dressing (Curasalt™)

A hypertonic saline gauze dressing, impregnated with 20% NaCl, is commercially available (Cursalt™ Tyco Healthcare). The dressing is intended for aggressive wound debridement. The dressing is proposed to work through osmotic action to desiccate necrotic tissue and bacteria. Since the debridement is non-selective, use is often limited to the first few days of wound care. An additional benefit to the osmotic effect is that it can reduce interstitial oedema. As a result of the reduced oedema, the pressure on the capillaries in the wound bed is also reduced resulting in improved wound perfusion. The proposed best use for this dressing is for infected necrotic heavily exuding wounds. Because this type of dressing interacts with the wound surface, it is considered an interactive dressing.

Gamgee™ (G™)

G™ (3M Animal Care Products, St. Paul, MN) is a versatile product that can be used as a wound dressing, while providing protection, support and insulation. It is made of a thick layer of absorbent cotton enclosed in a non-woven cover, which makes it non-adherent. The product is soft and easily conforms to the limb and wound surface. Because it is highly absorbent, its proposed best use is for highly exudative limb wounds during the inflammatory phase of wound healing.

Antimicrobial gauze dressing (Kerlix AMD™) and poultice pad

The characteristics and proposed best uses for dressing will be covered under Antimicrobial dressings in this article.

NATURAL PARTICULATE AND FIBROUS POLYMER DRESSINGS

This group includes naturally occurring products from a range of polysaccharide materials such as dextranomes, alginates and chitin. In general these dressing are highly absorbent (hydrophilic) and best used during the inflammatory and debridement phases of wound healing.

Particulate dextranomers (PDs)

PDs come as beads (eg, Debrisan® Johnson & Johnson Products Inc), flakes (eg, Avalon® Summit Hill Laborotories,) and powders (eg, Intrasite® Smith Nephew; Intracell®, Macleod Pharmaceuticals).

Intracell® will be covered separately under the heading of Maltodextrin. Although the beads will absorb the aqueous component, including prostaglandins, from wound exudates and dissolved material materials ranging from low molecular protein and inorganic salts, their pore size precludes the direct absorption of bacteria and viruses. Microorganisms, however, are removed from the wound bed primarily by capillary action between the beads. Additionally the beads may also activate chemotactic factors that will attract polymorphonuclear and mononuclear cells.

The best use for the PDs appears to be for debridement of sloughing exuding wounds. They should be discontinued when a healthy bed of granulation tissue develops and are contraindicated in dry wounds. Since PDs are not biodegradable, they should be rinsed from the wound with saline or other sterile salt solutions before the wound dries. Doing this will avoid particulate residues and the subsequent development of a granuloma.

Calcium alginate (CA)
CA dressings are available from a variety of sources (Curasorb® Ken Vet, Greeley, CO; C-Stat® R S. Jackson Inc., Alexandria, VA, Nu-Derm® Johnson & Johnson Products Inc). They are made from salts of alginic acid obtained from algae Phaeophyceae found in seaweed. Since the dressing is hydrophilic, it can absorb up to 20 to 30 times its weight in wound fluid. This process converts the initial dry felt like material into a hydrophilic gel on the wound surface that is easily removed. The hydrophilic alginate gel forms via a calcium and sodium ion exchange, providing a moist environment conducive to wound healing. Reportedly, the dressing increases epithelialisation and granulation tissue formation. This was not found in a study done in horses.

Other attributes that may be beneficial is that the dressing improves clotting. Zinc has been added to the alginate dressing (Curasorb ZN®, Tyco Health Care Group, Mansfield, MA) to increase its haemostatic qualities. The primary haemostatic use of the dressing is in packing sinuses, fistulae and bleeding tooth sockets. Some alginate dressings have the potential to activate macrophages within a chronic wound bed and have the ability to generate a pro-inflammatory signal, which promotes granulation tissue formation[26]. Also, some alginates have the ability to kick-start the healing cascade by causing lysis of mast cells resulting in release of histamine and 5HT. CA dressings are considered bioactive.

The reported best use for this dressing is in the moderate to heavily exudative wound during the transition from debridement to repair phases of wound healing. It has also been suggested they are best used for wounds with substantial tissue loss such as degloving injuries. The dressing can be pre-moistened in preparation for application to a chronic dry wound that needs stimulation to proceed with the formation of granulation tissue. A semiocculsive non-adherent pad should be placed over the dressing, followed by secondary and tertiary bandage layers.

OCCLUSIVE SYNTHETIC DRESSINGS

Hydrogels (Polyethylene oxide occlusive dressings)
Hydrogels are a three dimensional network of hydrophilic polymers with a water content between 90% and 95%. Dressings are available in the form of sheets or gels. The sheet hydrogels currently used are believed to possess most of the properties of an ideal wound dressing (eg, BioDres® DVM Pharmaceuticals; Tegagel dressing ™ 3M, Nu-gel® Johnson & Johnson Products). When applied to a dry wound they affectively hydrate it creating an environment for moist wound healing. The amorphous hydrogel forms also have a "moisture donor" effect for necrotic wounds that require debriding. By increasing the moisture content of the necrotic tissue and increasing collagenase production, hydrogels facilitate autolytic debridement. These dressings are easily removed from the wound bed because the moist interface between dressing and the wound prevents dressing adherence.

Hydrogels containing acemannan (Carra Vet®, Veterinary Products Laboratories; Carrasorb®. Carrington Laboratories) stimulate healing over exposed bone. Acemannan is a linked acetylated mannan that has the ability to stimulate macrophages to release fibrogenic and angiogenic cytokines (Interleukin-1 and TNF- alfa), which result in a positive effect on wound healing. Additionally it appears that acemannan can bind directly to growth factors, which may prolong their stimulating affect on granulation tissue formation. Other products contain gauze impregnated with a hydrogel

(e.g. FasCure® Ken Vet, Greeley, CO, Curafil® Tyco Healthcare) and another contains 25% propylene gylcol (Solugel® Johnson and Johnson).

One study done in horses evaluating the effects of Solugel® on second intention healing of small (2.5 X 2.5 cm) full-thickness skin distal limb wounds found no beneficial effects when compared to the control saline soaked gauze dressing. Another study done on horse limb wounds, the hydrogel sheet dressing (BioDres® DVM Pharmaceuticals) resulted in an increased need to trim exuberant granulation tissue, excess exudate and prolonged wound healing by greater than two times compared to controls. The persistent formation of the exuberant granulation tissue was believed to be the result of continued application of the BioDres® during the repair phase. From this came the recommendation that the dressing should be applied within 6 hours of wounding and continued out to at least 48 hours before changing. The dressing should be discontinued at the earliest signs of the formation of granulation tissue. Additionally, before a sheet hydrogel dressing is applied, the skin around the wound should be cleaned, dried and the wound surface gently rinsed with a dilute antiseptic solution. The dressing should be cut to the appropriate size for the wound and the thin sheet on one side peeled off. The dressing is then covered with a secondary and tertiary bandage layer and should be left in place for 2 days. These dressing are best used on clean acute wounds during the inflammatory and debridement phases of wound healing.

Hydrocolloid (HC)
HC dressings consist of an inner often-adhesive layer, thick absorbing hydrocolloid "mass" and an outer, thin water resistant bacterial impervious polyurethane film. The hydrocolloid mass is either made of gelatin, pectin and carboxymethylcellulose particles suspended in polyisobutylene (Duoderm® ER Squibb, Dermaheal® Solvay Animal health) or carboxymethylcellulose particles embedded in an elastotic mesh (Comfeel® Coloplast Marietta, GA). HC dressings tend to adhere to both wet and dry tissues. Some HC have been shown to bridge the interactive and bioactive classifications by exhibiting fibrinolytic, chemotactic and angiogenic effects. Since they are able to absorb fairly large amounts of wound fluid, they are often referred to as hydroactive dressings. Ultimately, the HC dissolves at the moist surface with the wound producing a yellow-coloured fluid. Duoderm® is oxygen impermeable, which is supposed to promote the rate of epithelialization and collagen synthesis and to decrease the pH of the wound exudates, thus reducing bacterial counts. A study done in horses found that Dermaheal® or Duoderm® dressings promoted the formation of granulation tissue directly from the surface of denuded bone and on the surface of frayed tendons and ligaments. This study also found that wound infection can develop underneath these dressings; and when it does, the application should be discontinued until the wound is healthy.
The best use for these dressings in horses is during the early inflammatory phase until granulation tissue fills the wound in the early repair phase. The dressing should be applied to a clean wound, free of infection, and discontinued before the development of exuberant granulation. If infection develops, the dressing should be discontinued until the infection is controlled, then the dressing is reapplied.

SEMIOCCLUSIVE SYNTHETIC DRESSINGS (SCD)

SCDs are commercially available in many forms; petrolatum impregnated gauze (NU Gauze sponges®, Johnson & Johnson; Vaseline Petrolatum Gauze® Tyco Healthcare; Xerofoam® Tyco Healthcare; Jelonet® Smith and Nephew); petrolatum emulsion dressing (Adaptic® Johnson and Johnson); oil emulsion knitted fabric (Curity®, Tyco Healthcare Kendall, Mansfield, MA) rayon/polyethylene fabric (Release®, Johnson and Johnson Products Inc); petrolatum impregnated gauze with 3% bismuth tribromophenate (Adaptic + Xerofoam® Johnson & Johnson Products); absorbent adhesive film (Mitraflex®, Polymedica Industries) and perforated polyester film filled with compressed cotton (Telfa®, Tyco Healthcare Kendall) Newer/advanced SCDs are also commercially available as a polyurethane sheet or foam. The latter will be covered under a separate heading.

In a study evaluating the effects of 2 semiocculsive dressings (Telfa®, Mitraflex®), a biologic dressing equine amnion and an occlusive dressing (Biodres®) on the healing of surgically created full thickness distal limb skin wounds in horses; they found that wounds dressed with Biodres® had an increased need to trim exuberant granulation tissue, excess exudate and prolonged wound healing by greater than two times compared to the control Telfa®. Wounds dressed with amnion required the least trimming of the granulation tissue and those dressed with Telfa® healed the fastest.

Polyurethane (PU)
PU dressings come as a film (eg, Op-Site® Smith Nephew, Tegaderm®, 3M, Bioclusive® Johnson and Johnson) or foam (eg, Hydrosorb®, Ken Vet, Hydrosorb® Wound Care Products, Avitar Inc, Sof-Foam® Johnson and Johnson). These dressing are designed to allow excess fluid to be lost by water vapour transmission through the membrane but prevent dehydration of the wound, thus providing an environment for moist wound healing. Although these dressings are considered non-adherent, one product Opsite® has a tendency to strip newly formed epidermis from the surface of a healing wound. The proposed best use for the sheet dressings in horses is during the repair phase; however, their unique characteristics allow them to be used during the entire healing period of a clean wound.

Polyurethane foam sponges come as sheet dressings, in situ formed foams and adhesive foams (eg, Tielle® hydropolymer adhesive, Johnson & Johnson). They are highly conforming, vapour permeable, absorptive, easy to apply, and provide an effective barrier against bacterial penetration. Moisture is absorbed into the dressing thus decreasing tissue maceration while providing a moist healing environment. The dressing is easily removed without disturbing the healing tissue. PU sponge is best used in the early inflammatory phase of wound healing, when there is considerable exudate in the wound. Under these circumstances, the bandage should be changed daily or as indicated according to the amount of fluid produced by the wound. Because of their semi-occlusive nature, they are also indicated during the repair phase of wound healing. An alternate use of the sponge is to deliver liquid medication or wetting agents to the wound by saturating the sponge before placing it on the wound. The same sponge however cannot be used for both absorption and medication delivery.

ANTIMICROBIAL DRESSINGS

Infection and bacterial colonization remain very important factors in delayed wound healing. Since the wide spread use of systemic and topical antibiotics has resulted in increasing numbers of resistant bacterial strains (eg, methicillin-resistant *Staphylococcus aureus* and Vancomycin-resistant *Enterococcus faecalis* and *Pseudomonas aeruginosa*) it has been suggested that the judicious use of antimicrobial dressings, notably those containing certain antiseptics, can be important in infection control and in promoting healing.

Iodine-containing dressing
Iodine dressing (Iodosord® Smith & Nephew, Hull, UK) is manufactured from cross-linked polymerized dextran, which contains iodine. As the dressing hydrates in the moist wound environment, elemental iodine is released to exert an antibacterial effect and to interact with macrophages to produce TNF-alfa and IL-6 that can indirectly influence wound healing. Reportedly, the best use would be for contaminated wounds early in the inflammatory phase of repair.

Another, slow release, iodine dressing (Iodoflex® Smith & Nephew) is designed to maintain adequate level of active iodine locally for at least a 48-hr period. The povidone-iodine in this product does not slow wound healing. A povidone iodine powder dressing (PRN® Wound Dressing. PRN Pharmacal) is also available. The product has 1.0% available iodine and has a broad antimicrobial spectrum, and is also fungicidal.

One study in horses documented no delay in wound healing in horses treated with 10% povidone iodine ointment compared to another antimicrobial dressing.

Antimicrobial gauze dressing (AMD)
An AMD dressing (Kerlix® Antimicrobial Dressing, Tyco Healthcare) is now available. This gauze dressing contains a polyhexamethylene biguanide (PHMB) agent that has a wide antimicrobial activity while being more biocompatible to tissues than its close relative, chlorhexidine. Kerlix® dressing has been shown to resist bacterial colonization within the dressing and to reduce bacterial penetration toward the wound site. The dressing comes packaged as a sponge or roll and the material can be applied wet or dry as described for plain mesh gauze. The proposed best use for this dressing is during the inflammatory and debridement phases of wound healing in wounds with a high concentration of bacteria, and in wounds where there is an open synovial cavity. This is also an excellent dressing for packing deep contaminated wounds associated with the body or upper limbs. The packing is pre-moistened with sterile saline, packed in the wound and kept in the wound with loosely "bow tied" large diameter sutures. The packing is changed daily with less gauze being used subsequently to pack the wound.

Poultice pad
The poultice pad (Animalintex® Poultice and Hoof pad, 3M Animal Care Products) is made of non-woven cotton pad with a plastic backing. The dressing contains boric acid (mild antiseptic) and Tragacanth, which is a natural poultice agent, and the pad is shaped to fit the sole of the foot. The dressing can be applied; hot, cold or dry. The proposed best use of the product is to apply it hot for infected hoof wounds (e.g. abscesses, dirty wounds, etc.) but it can be used as a poultice for other regions of the body. It may be used cold for sprains and strains and should be applied as a dry dressing over open clean wounds.

Silver chloride coated nylon dressing (Ag-WD)
AG - WD (Silverlon® Argentum, Lakemont GA; Acticoat® Antimicrobial Barrier, dressing, Westaim Biomedical Corp; Actisorb® Silver 220, Johnson & Johnson) are available. Silverlon has been shown to be effective in killing 5 equine pathogens in-vitro and they are antifungal. Equine wound pathogens tested included; *Escherichia coli, Klebsiella pneumoniae, Pseudomonas aeruginosa, Streptococcus epui subspecies zooepidemicus,* and *Staphylococcus aureus*. The silver that is released from the dressing over time kills the bacteria. The dressing should be moistened before application and the dressing is changed every 3 to 4 days. The product is also available in wound packing strips. When placed in a wound with depth, the strips will help facilitate wound drainage while enhancing healing. There appears to be different silver release rates for each dressing. The perceived best use for these dressings is during the inflammatory out to repair phase of wound healing.

Activated charcoal dressing
Activated charcoal dressings are available (Activate® 3M Animal Care Products and Actisorb® Johnson & Johnson). Proposed advantages of these dressings are; they provide a moist wound healing environment for autolytic debridement, they effectively absorb bacteria, they prevent the formation of exuberant granulation tissue in horses and they reduce wound odour. The reported best use for these dressings is for the heavily infected wound during the debridement phase out to the repair phase of wound healing. Anecdotally good healing has been seen in a limited number of cases though the repair phase of wound healing.

Antibiotic-impregnated collagen sponges (AICS)
AICS have been used extensively in human orthopaedic and soft tissue surgery for some time. One such product Collatamp G® (Schering Plough, Kenilworth, NJ) is made from denatured type 1 bovine collagen impregnated with gentamicin. Each 10 cm X 10 cm sponge contains 280 mg of collagen and 130 mg of gentamicin. It facilitates the prevention and treatment of infection by releasing gentamicin from the collagen matrix, initially by passive diffusion then by breakdown of the collagen by macrophages. In a study, comparing the level of gentamicin released in wound exudate after treatment with GICS or gentamicin impregnated PMMA beads; they found that on day 1, the concentration of gentamicin in GICS treated wounds was 15 times greater than the PMMA group and the concentration of gentamicin remained two times higher on the third day for the GICS group. In a clinical study done in 8 horses presenting with synovial sepsis, 7/8 horses responded favourably from implantation of the GICS sponge in the infected site. Collagen dressings have also been impregnated with amikacin.

BIOLOGICAL DRESSINGS

Biologic dressings are developed from natural products produced by the body. Reportedly they promote wound contraction and epithelialisation by retarding the formation of exuberant granulation tissue and they are considered bioactive.

Equine amnion (EA)
EA is believed to have most of the qualities of an ideal dressing. Despite its occlusive properties in the horse it resulted in less granulation tissue formation, but did not result in more rapid wound healing when compared to a synthetic semiocclusive control dressing. A study comparing amnion, a live yeast cell derivative and a non-adherent control dressing on second intention healing in horses found the percentage of epithelialisation was significantly greater and the number of days to complete healing was significantly lower for amnion covered wounds. This same study found less exuberant granulation tissue formation with amnion dressed wounds. Another study done in ponies found that amnion enhanced epithelialisation, and accelerated wound closure; in pinch-grafted wounds compared to wounds bandaged with a non-adherent wound dressing. The proposed best use for the dressing is to

apply to wounds of the distal extremities to suppress the formation of exuberant granulation tissue and accelerate epithelialisation. Bandaging over the dressing can be done but is not required.

Split thickness allogeneic skin (STS)
STS is believed to accelerate wounds healing by second intention. One study however found that wounds dressed with split thickness allogenic skin did not heal faster than similar wounds dressed with peritoneum, an acellular matrix or a synthetic dressing.

Extracellular matrix (ECM)
A significant body of work has been conducted over the past decade showing that acellular resorbable porcine ECM scaffolds derived from the small intestinal submucosa (PSIS) or from the urinary bladder basement membrane. PUBS facilitate constructive, tissue specific replacement of diverse tissue structures. The ECM scaffolds have been shown to have a profound angiogenic effect and although there is immune recognition, it occurs without rejection. The ECM's apparently have the capabilities of recruiting marrow - derived stem cells to migrate into the acellular scaffold resulting in constructive remodelling of the severely damaged or missing tissue. The healed remodelled tissue is associated with differentiated cell and tissue types including functional arteries and veins, innervated smooth muscle, cartilage and specialized epithelial structures. Additionally there is minimal scar tissue formation found in the healed wounds. There are two porcine ECM scaffolds available to veterinarians, Porcine Urinary Bladder Basement Membrane (PUBS) (ACell Vet® Scaffold; ACell, Inc; Jessup, Maryland and Porcine small intestinal submucosa (PSIS) (Vet BioSISt®; Cook Veterinary Products). Both products are considered a biologic device.

Solcoseryl® (S®)
S® is a protein-free, standardized dialysate/ultrafiltrate derived from calf blood (Solcoseryl®, Solco Basle Ltd, Birsfelden, Switzerland). In an equine study aimed at enhancing the acute inflammatory response during repair of deep wounds, they found that in the first month of repair, S® provoked a greater inflammatory response, with faster formation and contraction of granulation tissue. Subsequently S® inhibited repair by causing protracted inflammation and delayed epithelialization. The perceived best use is for deep wounds during the early inflammatory phase; treatment should be discontinued at the first signs of epithelialisation.

Platelet rich plasma (PRP)
PRP by definition is a volume of autologous plasma that has a platelet concentration well above baseline. Where the normal platelet counts in whole blood average about 200,000/µL, the platelet counts in PRP should average 1,000,000/µL in 5 mL of plasma. Reportedly lesser concentration of platelets cannot be relied upon to enhance wound healing and greater concentrations have not yet been shown to further enhance wound healing. There are at least 4 major groups of native growth factors in PRP that have the potential to enhance wound healing. PRP should only be made from anticoagulated blood since coagulation results in almost immediate release of growth factors. Within 10 minutes, it is estimated that platelets release 70% of their stored growth factors and close to 100% within the first hour. Because of this, clotting of the PRP should only be done just prior to its delivery to the surface of the wound. This is accomplished by adding thrombin to the PRP just prior to delivery. Within 30 seconds, the PRP/thrombin mixture forms a gel that adheres to the wound surface, thus forming a biologic membrane.

CONCLUSION

The selection of wound dressing for treatment of wounds destined to heal by second intention or be treated by delayed closure can be important to the outcome. Different dressings have been shown to promote healing during different phases of the wound healing process. Generally speaking, clean acute wounds are best dressed with an occlusive dressing until a healthy bed of granulation tissue develops. During the transition from the debridement to granulation tissue phase alginate dressings are recommended. Once granulation develops a semiocclusive dressing is recommended. Heavily contaminated or infected wounds are best treated with adherent dressings or particulate dextranomers or antimicrobial dressings until a healthy bed of granulation tissue develops, at this time a semiocclusive dressing is selected for the repair phase. Although the reports on biologic bioactive dressings are limited and in some cases conflicting, they represent an important category of dressings that will undoubtedly realise more use in the future.

RECOMMENDED READING

1. Ted Stashak, Ellis Farsvedt and Ashlee Otic. Update on Wound Dressings: Indications and Best Use. Clinical Techniques in Equine Practice 3, 148 – 163, 2004.

2. Ellis Farstvedt, Ted Stashak and Ashlee Othic. Update on Topical Wound Medications. Clinical Techniques in Equine Practice 3, 164 – 172, 2004.

PRACTICAL APPLICATION OF SPECIAL RADIOGRAPHIC VIEWS IN EQUINE PRACTICE

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ABSTRACT

Special radiographic views can be invaluable in making or confirming a diagnosis in equine lameness cases. Special views of the carpus include dorsal75°lateral-palmaromedial, dorsal75°medial-palmarolateral and flexed dorsoproximal-dorsodistal oblique views. Special views of the metacarpo/-tarsophalangeal joints include amongst others dorsodistal-palmaroproximal-125°, flexed dorso35°disto-palmaroproximal, flexed dorsoproximal-dorsodistal, lateral45°proximal-mediodistal, dorso30°proximal70°lateral-plantarodistal medial, dorso45°proximal45°lateral-plantarodistal medial and palmaroproximal-palmarodistal oblique views. The two special views for the tarsus are a flexed lateromedial and a flexed dorsoplantar view of the calcaneus. The only special view obtained of the stifle joint is the flexed cranioproximal-craniodistal oblique view. These views are used to evaluate structures and areas of the articular surface that can't be fully appreciated in the standard radiographic views and aids in providing a diagnosis, prognosis and appropriate treatment plan.

INTRODUCTION

Special radiographic views are often overlooked or forgotten in equine practice. Although not applicable in every case there are instances where they may be invaluable in making or confirming a diagnosis. This paper serves as a review and a summary of special radiographic views, the anatomy that they highlight and how they are obtained for the most commonly radiographed equine joints.

GENERAL COMMENTS

The standard radiographic nomenclature as proposed by the American College of Veterinary Radiologists is used to describe the radiographic views[3]. Some of these views require manipulation and unnatural positioning which may be difficult if the area being examined is very painful[2]. Local or intra-articular anaesthesia and/or sedation might be necessary to obtain the appropriate view. Radiographic safety must always be adhered to according to the ALARA (As Low As Reasonably Achievable) principle.

THE CARPUS

The carpus comprises the antebrachiocarpal, middle carpal and carpometacarpal joints. Intercarpal joints are formed by the articulation of the carpal bones in each row. To identify early changes associated with degenerative joint disease (DJD) a dorsal75°lateral-palmaromedial oblique and a dorsal75°medial-palmarolateral oblique view can be included[1]. This angle exposes more of the joint margins ensuring complete assessment. When sagittal or parasagittal fractures are suspected flexed dorsoproximal-dorsodistal oblique views are necessary[1]. The beam is angled at 85° to highlight the distal aspect of the radius[1]. A 55° angle is used to project the proximal row of carpal bones and a 35° angle exposes the distal row of carpal bones[1].

THE FETLOCK (METACARPO/-TARSOPHALANGEAL) JOINT

The metacarpophalangeal joint is formed by the third metacarpal bone (Mc III), the proximal phalanx (P1) and the proximal sesamoid bones (PSBs). In the metatarsophalangeal joint the third metatarsal bone (Mt III) forms the proximal articulation. These will mainly be referred to the metacarpophalangeal joint in the text but the same views may be applied to the metatarsophalangeal joint.

To fully evaluate the distal articular surface of Mc III several views at different angles are necessary[1]. The dorsodistal-palmaroproximal-125° oblique or 125°DPa view is used to evaluate the palmar articular aspect of distal Mc III[2]. The limb is extended and placed on a positioning block so that the dorsal aspect of Mc III forms a 125° angle with the horizontal plane[1]. The primary beam is directed

horizontally and centered to the fetlock joint[1]. Approximately one quarter to one third of the PSBs are projected below the joint space[1]. This view does however cause distortion and magnification[1]. An alternative method of obtaining an image of the same area is to take a flexed dorso35°disto-palmaroproximal oblique view[1]. The fetlock joint is semi-flexed and the beam is angled 35° distal from the dorsal aspect of Mc III[1]. The angle can be adjusted to examine different sections of the distal articular aspect of Mc III[1]. The axial margins of the PSBs can easily be evaluated, as they are displaced proximally[1]. On both these views the palmar articular aspects of Mc III condyles are evaluated for comminuted fractures as well as for osteochondral lesions[2].

The flexed dorsoproximal-dorsodistal oblique view of the metacarpophalangeal joint is used to examine the dorsodistal articular aspect of Mc III, which includes the sagittal ridge[2]. The fetlock is flexed and the toe may be rested on a positioning block[1]. The primary beam is angled proximally at 45-70° on the dorsal aspect of the limb and centered on the metacarpophalangeal joint[1]. The image receptor is placed on the dorsal aspect, distal to the joint and parallel to the horizontal plane[1]. Articular involvement of lesions on the dorsodistal aspect and sagittal ridge can be determined and osteochondral lesions of the sagittal ridge can be further examined[2].

A lateral45°proximal-mediodistal oblique view is used to evaluate the abaxial surface of medial PSB[1]. While the horse is bearing weight the beam is angled 45° proximally on the lateral aspect of the metacarpophalangeal joint with the image receptor placed vertically on the medial aspect of the limb[1]. The medial PSB is projected proximal to the lateral PSB. A medial45°proximal-laterodistal oblique view is used to evaluate the abaxial surface of the lateral PSB[1]. These views are used to determine articular involvement of an abaxial fracture of the respective PSB[2].

The lesions identified with the following sets of views are more commonly encountered in the pelvic limb, therefore reference is made to the plantar surface and metatarsus III (Mt III)[2]. The dorso30°proximal70°lateral-plantarodistomedial oblique view is used to evaluate the proximoplanar articular aspects of the lateral plantar eminence of P1[1].The lateral PSB is projected distal to the medial PSB. The medial plantar aspect of the joint can be highlighted by use of the dorso30°proximal70°medial-plantarodistolateral oblique view[1]. These views are used to localise and determine the origin of proximo-plantar fragments, separate centres of ossification and osteochondritic lesions of P1[1].

On the dorso45°proximal45°lateral-plantarodistomedial oblique view the lateral condyle of Mt III is projected distal to the medial condyle and it highlights its plantar aspect[1]. It is useful for identifying stress related changes in the lateral condyle of Mt III[2]. The dorso45°proximal45°medial-plantarodistolateral oblique view is used to evaluate the plantar aspect of the medial condyle of Mt III. In these views there is also separation of certain areas of the PSBs allowing evaluation without superimposition of the surrounding bony structures.

To aid in the evaluation of the axial and abaxial margins of the PSBs a palmaroproximal-palmarodistal oblique view can be obtained[1]. The horse stands with the limb under examination as far caudally as possible and the foot is placed on a cassette tunnel containing the image receptor[1]. The primary beam is angled at an angle of approximately 85°[2]. Lateral and medial angulation of 15° will accentuate the abaxial surface of the medial and lateral PSB respectively[1]. Osteolytic lesions and fractures of the PSBs can be further elucidated using these views[2].

THE TARSUS

The tarsus comprises the tarsocrural, proximal and distal intertarsal and the tarsometatarsal joints. A flexed lateromedial view is considered one of the special views of the tarsus. This view allows good visualisation of the proximoplantar aspects of the trochlea of the talus, the *Tuber calcanei* and the caudodistal aspect of the tibia[1]. The tarsus is manually flexed at an angle of approximately 50° avoiding abduction of the limb[1]. The primary beam is centered on the talus and the image receptor is placed on the medial aspect of the joint[1]. This view is useful to highlight any pathology involving the proximalplantar surface of the medial trochlear ridge of the talus including fragmentation, fractures or osteochondrosis[2].

The flexed dorsoplantar view of the calcaneus is used to demonstrate the *Sustentaculum tali*, *Tuber calcanei*, the tarsal groove and the medial trochlear ridge of the talus[1;2]. The tarsus is flexed manually,

the image receptor is held parallel to the plantar aspect of the calcaneus and the primary beam is directed as perpendicular to it as possible[1]. Evaluating and localising osteitis and osteomyelitis of the calcaneus and *Sustentaculum tali* can be done on this view[2]. Calcaneal and proximoplantar fractures of the medial trochlear ridge of the talus can also be confirmed and further classified[2].

THE STIFLE

The stifle is composed of three joints: the femoropatellar, medial and lateral femorotibial joints. The only special view obtained of the stifle joint is the flexed cranioproximal-craniodistal oblique view. The patella, femoral intertrochlear groove and troclear ridges are well visualised using this view[1]. The stifle is manually flexed, the image receptor is placed parallel to the cranial aspect of the tibia and the primary beam is directed as perpendicular to it as possible[1]. This view is necessary for diagnosis of sagittal fractures of the patella and is indicated in cases with trauma or lameness associated with the patellar region.

CONCLUSION

These special radiographic views can be used to confirm a suspected diagnosis on standard radiographic views and could potentially provide a diagnosis when no abnormalities are detected on the standard views[2]. Information gained from the special views can also aid in providing a prognosis and appropriate treatment plan[2].

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STANDARD EQUINE RADIOGRAPHIC VIEWS, THEIR WORTH & BASIC RADIOLOGICAL ANATOMY

Nicolene Hoepner

No paper submitted

CURRENT DEWORMING PROTOCOLS FOR HORSES DENTISTRY FOR DUMMIES

Ingrid Cilliers

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"BACK TO SCHOOL ARRHYTHMIC" DRUG & INFUSION CALCULATIONS

Dr Lynette Bester

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There is an increasing demand on veterinary nurses to take more and more responsibility for drugs and drug protocols. The ability to do accurate and quick drug calculations is an essential skill for nurses.

This lecture aims to explain calculations in a step by step approach for routine drugs used during anaesthesia, but also include fluid rates and constant rate infusions. There are quite a few calculators available online, but it is still important to understand how these calculations are done and to develop a "gut feel" if a dose or dose rate seem to be wrong.

Additional reading:

https://www.vetnurse.co.uk/nursing/w/vet-nurse-revision_1/calculation-of-drug-doses-key-notes.aspx – Dosage Calculations for Veterinary Nurses & Techniciansby Terry LakePublished March 15th 2004 by Butterworth-Heinemann

http://www.vasg.org/drug_delivery_calculators.htm

BRUCELLA ABORTUS – A FRUSTRATING HERD DISEASE

Ken Pettey

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K Labuschagne

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THREE TECHNIQUES CONFIRMING SEPARATE SPECIES STATUS WITHIN THE *CULICOIDES BRUCEI* SPECIES GROUP

K Labuschagne

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KRUGER NATIONAL PARK ANTI-RHINO POACHING K9 UNITS

Johan de Beer

No paper submitted

HEADING HOME, THE BEST WAY TO RECOVERY

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Client non-compliance is one of the most frustrating aspects of any veterinary practice. The veterinary nurse plays a vital role in the link between the veterinarian and the owner/client. By getting the veterinary nurse to talking to the owners, educating them about their pets disease process and understanding their personal circumstances and adapting treatments/instructions to better suit them, compliance will be improved immensely.