Companion Animal Research Review™

Making Education Easy

Issue 7 – 2019

In this issue:

Abbreviations used in this issue

ACVIM = American College of Veterinary Internal Medicine
BUN = blood urea nitrogen
CKD = chronic kidney disease
CSF = cerebrospinal fluid
GFR = glomerular filtration rate
ICU = intensive care unit
IMHA = immune-mediated haemolytic anaemia
ISCAID = International Society for Companion Animal Infectious Diseases
RCT = randomised controlled trial
SDMA = serum symmetric dimethylarginine
TSH = thyroid-stimulating hormone
UTI = urinary tract infection

Welcome to Issue 7 of Companion Animal Research Review.

After a bit of a hiatus, we're back. Once again, I've collated this buffet of publications, which is then plated up and served to you by the Research Review team. There are two reviews worth noting on IMHA and UTIs, and original studies on bladder lavage in obstructed cats, Campylobacter infections and the risk of polyradiculoneuritis, the relationship between serum lipids and survival in acutely ill dogs, the risk of lepto infection in clinic staff, conventional versus laparoscopic speys, and how the profession is addressing the relationship between pet and domestic abuse. As always, please don't hesitate to give us your feedback, and in the meantime, happy reading!

Kind regards,

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Independent commentary by Nick Cave.

Nick Cave is an Associate Professor in small animal medicine and nutrition at Massey University, NZ. He graduated from Massey University in 1990 with a BVSc, and worked in general practice for 6 years until 1997, when he returned to Massey for a residency in small animal internal medicine, attained membership in the Australasian College of Veterinary Scientists by examination, and graduated with a Masters in Veterinary Science in 2000. In 2004 he moved to the University of California, Davis, where he attained a PhD in nutrition and immunology. At the same time, he completed a residency in small animal clinical nutrition, and became a diplomate of the American College of Veterinary Nutrition by examination in 2004. In late 2005, he returned to Massey University to lecture in small animal medicine and nutrition. He is a founding member of the WSAVA Global Nutrition Committee, and a founding board member for the Massey University Working Dog Centre.

Prognostic value of dyslipidemia for sick dogs hospitalized in the intensive care unit of a veterinary teaching hospital

Authors: Viall AK et al.

Summary: This retrospective cohort study evaluated the serum concentrations of cholesterol and triglycerides of dogs when initially examined for hospitalisation in the ICU of a veterinary teaching hospital and aimed to determine whether these variables are predictive of survival to hospital discharge. Between January 1, 2012, and September 30, 2015, the medical records of 398 client-owned sick dogs and 151 healthy dogs at the same teaching hospital were reviewed. Hypocholesterolaemia (OR 1.87; 95% CI 1.04-3.34), hypertriglyceridaemia (OR 3.20; 95% CI 2.00-5.13), and concurrent hypocholesterolaemia and hypertriglyceridaemia (OR 55.7; 95% CI 3.2-959.6) at the time of initial evaluation were all identified as factors associated with increased odds of sick dogs not surviving to hospital discharge.

Comment: The exact list of analytes included in biochemistry panels may not be simply a product of arbitrary tradition, but it is certainly influenced by factors other than maximal utility. Cholesterol, for instance, is found on our biochemistry panels more because it has been of diagnostic importance in human medicine, than because of its diagnostic value in domestic species. Though it has some utility, the measurement of triglycerides would probably be more helpful both diagnostically, for prognostication, and for management decisions. The decision as to whether to restrict dietary fat following an episode of pancreatitis should, at least in part, be guided by a patient's fasting and post-prandial triacylglycerol (TAG) concentration. The analysis of TAGs has not been part of the clinical pathology canon, and as a result, our understanding of dyslipidaemias is embryonic at best. This study by Viall et al., may not lead to any diagnostic epiphanies, but it peaks behind the curtain to gaze upon the largely undiscovered land of dyslipidaemia that lies outside our normal clinical consciousness. Inflammatory cytokines have long been known to suppress hepatic lipoprotein production, and simultaneously reduce peripheral clearance of TAG, meaning that the net effect is unpredictable. Nonetheless, serum TAGs were significantly higher in sick and non-surviving dogs than in healthy dogs, although the difference was very small and not clinically or diagnostically helpful. The authors did not argue why creation of a ratio of cholesterol:TAG would be more useful than TAG alone, when there was no difference in cholesterol between groups. When one variable in a ratio changes and the other remains constant, the ratio will, of course, also change, and adds no further information. Nonetheless, when they trichotomised patients into hypo-, normo-, and hyper- categories, both hypertriglyceridaemia and hypocholesterolaemia were associated with a poorer prognosis. In their analysis, dogs that received nutritional support were significantly more likely to survive, though as always, it is unclear if that is evidence of the efficacy of forced nutrition in intensive care, or simply demonstrative that sicker patients are less likely to receive it. So for the time being, we can conclude that dyslipidaemias are common in critical illness, and related to outcome, though we are not close to having a new diagnostic tool in the box yet. That being said, I would still argue for the utility of assaying TAGs in patients with acute pancreatitis.

Reference: J Am Vet Med Assoc. 2019;254(6):699-709
Abstract

ACVIM consensus statement on the treatment of immune-mediated hemolytic anemia in dogs

Authors: Swann JW et al.

Summary: In order to address inconsistencies in the treatment of immune-mediated haemolytic anaemia (IMHA) in dogs, The American College of Veterinary Internal Medicine (ACVIM) has issued evidence-based guidelines for the management of this condition. They also reviewed emerging treatments for IMHA in dogs and highlighted areas deserving of future research. They recommend that the guidelines be implemented pragmatically, with consideration of animal, owner, and veterinary factors that may vary from case to case.

Comment: In Shakespeare's The Winter's Tale, King Leontes dispatches two lords to consult with the Oracle of Delphi, the mortal mouthpiece for the gods, to obtain a wise judgement as to the fidelity of his wife. Disliking what he learns, Leontes rejects the ruling, and pays a heavy price: the death of his son. News from learned sources is not always welcomed. I suspect I have mentioned something like this before, but whenever news of a "consensus statement" is published in the veterinary literature, a fairy in the land of Epistemology dies. Or at least self-harms a little. The intent of these statements is noble, and in the words of the ACVIM, they are an attempt "to provide the veterinary community with up-to-date information on the pathophysiology, diagnosis, and treatment of clinically important animal diseases." But isn't that what any decent review or meta-analysis is for? The suggestion is, of course, that consensus statements are required when there are too few or inadequate studies to count as true knowledge, and then the weight of learned opinion can be laid to rest on the subject, freed from the annoying shackles of evidence. I know that's a bit cynical, but we should at least be sceptical. The ACVIM has published some famously unhelpful consensus statements, including ones on microalbuminuria, dietary carbohydrate and diabetes, and perhaps even the unhelpfully parochial statement on leptospirosis. So why might I suggest that you consider reading this latest one on IMHA? Two reasons. Firstly, it's been a while since a comprehensive review of the management of IMHA has been published, and this is certainly one. Eight academics and specialists from the US and UK gave birth to this baby, and at a whopping 32 pages and 186 references, it would have certainly required the publishing equivalent of calving chains and an episiotomy to get it into the world. However, though you are unlikely to take this one to bed for a relaxing read, you would do well to have it on hand in the clinic to dip into when the need arises. You will find gems such as tips on when to transfuse, permission to use what seem to me to be eye-watering doses of prednisone, and an honest appraisal of our lack of confidence of what is actually "best" when it comes to second-line therapy if prednisone fails. They provide up to date summaries of relapse rates, recommendations for inhibition of platelet aggregation, and a cursory comment about prognosis. My second reason for recommending, is the rigor with which the authors undertook their labour. Rather than the often haphazard and highly unreliable group think approach, they used the "Delphi technique", which is based on the idea that decisions/predictions based on groups are better than based on individuals. First formally adopted in the 1950s, it is a method of reviewing and criticising data in an attempt to eliminate the often unhelpful interpersonal interactions that have undue influence in decision-making, as usually happens when groups of experts interact. In the case of this consensus statement, it has done yeoman's service. And as in the case of Leontes, we ignore the edicts from the oracles of Delphi at our peril.

Reference: J Vet Intern Med. 2019;Mar 7 [Epub ahead of print]
Abstract

International Society for Companion Animal Infectious Diseases (ISCAID) guidelines for the diagnosis and management of bacterial urinary tract infections in dogs and cats

Authors: Weese JS et al.

Summary: The International Society for Companion Animal Infectious Diseases (ISCAID) have revised and expanded the 2011 antimicrobial use guidelines for the treatment of urinary tract disease in dogs and cats. The revised guidelines provide recommendations for diagnosis and management of sporadic bacterial cystitis, recurrent bacterial cystitis, bacterial prostatitis, pyelonephritis, and subclinical bacteriuria. The guidelines also address issues pertaining to urinary catheters, prophylaxis for urological procedures, and medical dissolution of uroliths.

Comment: Ever wondered where the ubiquitous recommendations that antibiotic courses should last 7-10, or 10-14 days come from? If only we could answer that they were from well designed, randomised prospective clinical trials. Most "established" and "textbook" recommendations are more demonstrative of the power of the status quo than of critically evaluated distillations of research. Last year, penicillin turned 90, and whilst you shouldn't feel chagrin at not having sent birthday wishes, it is interesting to think back to its birth. Discovered by Alexander Fleming in 1928, it took several years before purification and production was sufficient to treat human subjects. The first person to be treated was a policeman in Oxford in 1941 (Gaynes R 2017). Though perhaps apocryphal, he supposedly became infected from a scratch from a rose thorn, but subsequently developed severe cellulitis and became septicaemic, and critically ill. Within 24 hours of starting penicillin injections, his fever improved, and his clinical signs started abating. His appetite returned soon after, and by day four, he was ambulatory again. And then the precious supply of penicillin ran out. Over the next few days, he deteriorated again, and died a few weeks later. From that experience, it was recommended that courses last at least 7 days. And here we find ourselves. In the publication by Weese et al, or "ISCAID" as they called themselves – a title less catchy, but no more lovely than syphilis – the authors present one of the most comprehensive reviews of antibiotic usage in UTIs available. The author list is a veritable who's who of stinky urine aficionados, and along with the IMHA review, is another tome worthy of printing and keeping in the clinic. The paper is an update from a 2011 version, and is a substantial emendation. Prominent, is the recognition of asymptomatic bacteriuria, and their strong recommendation not to culture urine from asymptomatic patients because they are thought to be "at risk" of UTI, including animals with diabetes, Cushings, and CKD, all formally on the bacto-lovers list of "must culture". They provide helpful definitions of recurrent UTIs (more than 3 in 12 months, or 2 in 6 months), and solid, if unsurprising recommendations for first-line antibiotic choices. I found I still disagreed with their stance on empirical treatment without culture and sensitivity testing for uncomplicated UTIs (I can't see the justification, unless it is financial), but they were at pains to exclude feline cystitis from empirical antibiotic treatment. There was weaker discussion, because of weaker evidence, on what to do with infection-induced struvite uroliths in dogs. Traditionally we have started with antibiotic therapy while dietary adjustment is instigated, and some have argued for extending treatment until there is complete dissolution of the stones. The expert panel suggest that antibiotics are probably not required at all, though I am still nervous of those where there is a very high urine pH from urease-producing bacteria. The conclusion on the subject was that a 7-day course might be justified if there is evidence of bacterial cystitis – presumably pyuria. But on the subject of the duration of antibiotic courses for otherwise uncomplicated UTIs, they were in full voice. Three to five days, with the shorter end being optimal. I would hope that most readers now recognise that there is no justification, and in fact never was any for the arbitrary 10-14 day course, which it seems, has its origins at the birth of antibiotics. We may have missed penicillin's birthday, but when it comes to responsible antibiotic stewardship, let's hope it is still the case that late, is better than never.

Reference: The Veterinary Journal 2019;247:8-25
Abstract

Effect of urinary bladder lavage on in-hospital recurrence of urethral obstruction and durations of urinary catheter retention and hospitalization for male cats

Authors: Dorsey TI et al.

Summary: This RCT evaluated the effect of urinary bladder lavage on in-hospital recurrence of urethral obstruction and durations of urinary catheter retention and hospitalisation for 137 male cats with urethral obstruction. The cats were randomised to one of two groups and either underwent urinary bladder lavage with saline (0.9% NaCl) solution after alleviation of the obstruction and placement of a urethral catheter (flush group; n = 69) or did not undergo the procedure (no-flush group; n = 68). The study found that urinary bladder lavage at the time of urethral catheterisation did not significantly improve in-hospital recurrence rate of the condition (13% vs 19% for the no-flush group), median duration of urinary catheter retention (37 hours vs 36 hours), nor duration of hospitalisation (3 days vs 3 days).

Comment: Like Lennon and McCartney, bacon and eggs, pigeons and chlamydia; a common cold is frequently accompanied by a hot lemon and honey drink. Well at least it is with me. I am well aware that, like sprinkles of holy water, it does no harm, but it does no good either, and yet, I cannot evade the placebo effect. That's fine for benign self-treatment, but what about my clinical decisions and actions? I still vividly remember the first demonstration of how to vigorously lavage a cat's bladder immediately following urethral catheterisation by one of my first employers. The anonymous doyen filled the bladder to near capacity, held it in his hand and tossed it up and down with the same sort of action Shane Bond might use on a cricket ball as he starts his run up. All wrist. Then the bladder was expressed with a surprising vehemence that often resulted in the dual action of forcefully evacuating the bladder of its gritty contents, and the spritzing of the nurse holding the kidney dish. I was sold. What could make more sense than to eliminate the incriminated sediment and reduce the risk of reobstruction in the near future? So is the study by Dorsey et al., the definitive thumb to the nose of that apparent sense? Perhaps. The study design, appropriate powering, and reporting are excellent examples of how prospective research should be conducted, and the results are compelling. There were no hints of significant differences between the groups after randomisation prior to treatment, and care was made to exclude cats with confounding problems. The test group was to be lavaged with 500 mL of saline, and then clinical decisions were made by the attending clinician. Reobstruction occurred in 13% of lavaged cats, and 19% of control cats, which was not remotely significant. Catheters were removed after 3 (accidental) to 172 hours, though there was no difference between groups. It is implausible that substantial crystalline debris reformed in that time, so it is more likely that reobstruction occurs from the mucus, blood, and urethral swelling that subsequently develops. However, not all cats received 500 mL of lavage, with the average receiving 250 mL. In addition, there was no description or standardisation of the method of lavage or, notably, expression. So we are left uncertain as to how much of the sediment remained. Those of us who practice bladder lavage know that simple lavage and expression fails to remove the bulk of the sediment, and it requires the wrists of Shane Bond to encourage that which sinks, to be that which suspends, and thence evacuated. The authors understandably conclude from their data that bladder lavage appears to lack efficacy, and go as far as to warn against the technique, because it increases the anaesthetic time. That last bit of pudding seems a bit over-egged, given that it doesn't take more than a few minutes, and in comparison with the time taken to unblock some cats, is negligible. So I'm not sure I buy the warning to abandon the technique. But is this study enough to dissuade me? Or, dear bladder-jiggling readers, you? I think I'll make myself a nice hot lemon and honey and ruminate on that for a bit.

Reference: J Am Vet Med Assoc. 2019;254(4):483-6
Abstract

Evaluation of serum symmetric dimethylarginine concentration as a marker for masked chronic kidney disease in cats with hyperthyroidism

Authors: Peterson ME et al.

Summary: This prospective study in 262 hyperthyroid cats and 206 aged-matched, clinically normal cats was undertaken to investigate serum symmetric dimethylarginine (SDMA) concentration as a potential marker of masked azotaemia chronic kidney disease (CKD) in untreated hyperthyroid cats. Creatinine, urea nitrogen, SDMA, T4, and TSH concentrations were measured before and 1, 3, and 6 months after treatment with radioiodine (I-131). I-131-treated cats were classified as azotaemic or non-azotaemic based on persistent, post-treatment creatinine concentrations >2.1 mg/dL; none of the hyperthyroid cats were azotaemic before treatment. At a median of 6 months from the start of I-131 treatment, 42 (16%) cats became azotaemic (14 of these cats had high SDMA concentrations before treatment). SDMA showed a sensitivity of 33.3% and specificity of 97.7% as a diagnostic test for pre-azotaemic (masked) CKD in untreated hyperthyroid cats. The authors concluded that finding a high serum SDMA levels in a hyperthyroid cat could help predict development of azotemia after treatment.

Comment: Since feline hyperthyroidism was first described in 1980 by Mark Peterson, we have moved through, like the phases of grief, different phases of treating it. The first phase was that we would treat cats with carbimazole (or methimazole) and if they became even a teeny bit azotaemic, then we would withdraw treatment, and I-131 was deemed contraindicated in those cases. The idea was that the increased blood flow from the hypertensive hyperthyroid state was "supporting the kidneys". Then someone pointed out that was bollocks. Glomerular hypertension and hyperfiltration leads to progressive sclerosis, proteinuria, and the acceleration of tubular disease. Phase 2 followed, and we blissfully treated every cat, and didn't worry about much. Not even hypothyroidism. Then someone pointed out that hypothyroid cats with CKD die even quicker, and now we find ourselves in phase 3, where we treat every cat, but monitor for hypothyroidism, and when identified, either decrease the dose, or in a post I-131 cat we supplement to achieve euthyroidism. Now that the SDMA assay is available, we have a new hammer, and every problem looks like a nail. In Mark Peterson's latest foray into the thyroid, the esteemed list of authors pose the question of whether SDMA might be a more sensitive marker of decreased GFR prior to treatment with I-131. The answer – not really, since the sensitivity (or predictive power) is too low to be clinically useful as a screening test. However, let's suppose it is: what would follow from that? The change from "occult status" to "clinical status" may simply be a matter of laboratory values, the result of a predictable decrease in GFR once T4 is normalised, and, according to our phase 3 evidence and thinking, improves the prognosis for CKD progression. The authors argue that an elevated pre-treatment SDMA is highly specific, but insensitive for predicting post-treatment "CKD" (my quotes). I would suggest that the CKD was already present, and it is simply the increase in BUN, creatinine, and further increase in SDMA that have resulted. The conclusion that a) identifying these cats may allow treatment of CKD earlier, and b) identified cats should be more closely monitored, don't quite hit the spot for me. All cats should be monitored after treatment, and the authors' own data confirmed that hypothyroid cats declined faster than euthyroid, with some not becoming hypothyroid until 12 months after I-131 treatment. As for allowing earlier treatment (a.k.a renal diet), I ask: earlier than what? Two or 4 weeks after initiating treatment? Even if we believed that would make a difference, the sensitivity of the SDMA, along with their predictive regression equation, is too low to make any conclusion other than "I should monitor this cat". Which is what we should do for *every* cat.

Reference: J Vet Intern Med. 2018;32(1):295-304
Abstract

An aged canid with behavioral deficits exhibits blood and cerebrospinal fluid amyloid beta oligomers

Authors: Rusbridge C et al.

Summary: These authors detected soluble amyloid β (Aβ) oligomers in the blood and CSF of a 12-year-old Samoyed dog with cognitive dysfunction. They also showed that the dog's Aβ oligomers affected the survival of human-derived neuron-like cell lines and had a direct effect on the aggregation kinetics of human synthetic Aβ peptides.

Comment: Old dogs can become demented. Doolally. Bonkers. Nuttier than a fruit cake. Fruitier than a nut cake. For as long as the beasts have been our besties, it has been recognised that cognitive decline occurs in aged dogs. Amyloid plaques were described in the histology of aged dogs brains in the 1960s, and medical literature considered the similarities to human Alzheimer's, with doctors being the first to coin the term "canine cognitive dysfunction" in the late 1980s. It wasn't until a decade later, that the term and the similarities to human cognitive decline were widely accepted by veterinarians. A surprising amount of observational and histological research had been conducted outside the veterinary academy before the first veterinary review was published in 2001, by the serendipitously named Dr Head (Head E 2001). And to this day, antiquated Airedales, or in this case, senescent Samoyeds, remain a font for exciting developments. I'll admit, that of all the papers I picked for this edition, this one is the least likely to be actually read by you. That's fine, because for us as clinicians, I think there is a simple take-home message that is easily distilled in the summary, plus, it is one of the more frustrating and impenetrable reads I've had in a while. Recently, it has been shown that small fragments (oligomers) of the Aβ-peptide that accumulates in Alzheimer's, can be found intracellularly, extracellularly, and in both CSF and serum of patients. It is now thought that these are not simply markers of the disease, but the soluble Aβ oligomers are the major neurotoxins responsible for the progressive neurodegeneration (Jeong S 2017). The paper by Rusbridge et al., describes a single senile Samoyed, with clinical signs of cognitive dysfunction, MRI and histological changes consistent with Alzheimer's and, for the first time, the presence of the toxic Aβ oligomers in both serum and CSF. In itself, it is an interesting finding, and another piece of evidence in support of canine age-associated cognitive decline as a close, perhaps equivalent disease to Alzheimer's. However, it also raises the possibility that we may soon have a relatively simple assay (ELISA) to use on either serum or CSF as a specific diagnostic test for the disease in dogs.

Reference: Front Aging Neurosci. 2018;10:7
Abstract

Investigation of the role of Campylobacter infection in suspected acute polyradiculo-neuritis in dogs

Authors: Martinez-Anton L et al.

Summary: This case-control study involving 27 client-owned dogs suffering from suspected acute polyradiculoneuritis (APN) and 47 healthy dogs (client-owned or owned by staff members) was undertaken in order to estimate the association between *Campylobacter* species. infection and APN. APN cases were 9.4-fold more likely to be positive for *Campylobacter* spp. compared to control dogs ($p < 0.001$) in cases in which the faecal sample was collected within 7 days from onset of clinical signs. Furthermore, a significant association was detected between dogs affected by APN and the consumption of raw chicken (96% of APN cases; 26% of control dogs). *Campylobacter upsaliensis* was the most common *Campylobacter* spp. detected.

Comment: In 1859 a French physician, Octave Landry, described a 43-year-old man that experienced fever, malaise, and pain in all limbs. Despite initial recovery, the patient subsequently developed an ascending paralysis starting with sensory loss, followed by ataxia, paralysis, and after 3 weeks, death from respiratory failure (Pearce J 1997). The syndrome was more fully described by Guillain, Barre, and Strohl in 1916, giving rise to the eponymous diagnosis, Guillain-Barre syndrome (GBS), and robbing Landry of fame. GBS is an immune-mediated polyneuropathy that is usually preceded by a gastrointestinal or respiratory illness 1-6 weeks prior to presentation. Many infections agents have been causally linked to GBS, however the most common and best described precedent infection is *Campylobacter jejuni* enteritis. Following the 2016 floods in Havelock North, up to 40% of the population developed acute enteritis, the majority of which were attributed to intestinal infection with *Campylobacter* spp. By the time the waters had subsided, there were over 1000 notified cases of *C. jejuni* infection, 45 hospitalisations, and four deaths. (McElnay C 2017). Two of the hospitalisations were due to the development of GBS, which tragically claimed the life of one woman. Humans are famously susceptible to disease from *Campylobacter* spp. infection, and even very small oral doses are infective and pathogenic. Yet dogs and cats are far more resistant to disease following intestinal colonisation, and in a study of healthy dogs at Massey, *Campylobacter* spp. were part of the microflora in 36% (Bojanić K et al. 2017). Colonisation is relatively common across feeding practices, however raw food feeding is a consistent risk for increased carriage, not surprisingly in NZ, where 31% of non-poultry raw foods, and 73% of raw chicken pet foods contain *Campylobacter* spp. In the study by Martinez-Anton et al., from Melbourne, the authors present the first convincing evidence for a causal link between Campylobacter colonisation in dogs, and the development of an acute onset polyneuropathy. The prevalence of carriage in controls was similar to the Massey study (23%). The prevalence in affected dogs was 48%, although when they tightened up the case definition, the difference increased, to cases being 9-fold more likely to be a carrier. This finding does not prove causality, however, it is compelling, especially with the association with feeding raw chicken, although it would have been nice to have had histories of any precedent enteritis. Molecular mimicry is likely at play, since Campylobacter lipopolysaccharide has been shown to immunologically cross-react with gangliosides in mammalian periperhal nerves. In the Massey University Veterinary Teaching Hospital, the causes of acute peripheral neuropathies are almost always undetermined, and after ruling out the usual suspects (congenital forms, ticks, toxoplasma, neospora, organophosphate, lead, degenerative, neoplastic, myasthenia, etc.), they are given the moniker "acute idiopathic polydradiculoneuritis" or something similar, and managed with a supportive, fingers-crossed approach. Now we have evidence that enteric colonisation may, in some cases, be a causal factor. It is not suggested that treatment for *Campylobacter* spp. colonisation would be useful, but prevention is always better than cure. As if we needed another reason to question the practice of feeding raw food diets.

Reference: J Vet Intern Med. 2018;32(1):352-60
Abstract

Despite high-risk exposures, no evidence of zoonotic transmission during a canine outbreak of leptospirosis

Authors: Guagliardo SAJ et al.

Summary: This US study examined the zoonotic potential of a canine leptospirosis among dog owners and animal care providers in the Phoenix metro area, with the Maricopa County Department of Public Health, Arizona Department of Health Services and Centers for Disease Control and Prevention conducting both active and passive surveillance. Passive surveillance identified seven symptomatic persons previously exposed to canine leptospirosis, of whom six underwent testing for serological evidence of *Leptospira* spp. infection (microscopic agglutination test MAT) and all were found to be negative; four were also negative by PCR. A serosurvey conducted in nine dog owners and 109 animal care providers found all were seronegative. Infection control practices were inconsistent, 44.6% of participants used gloves when working with canine leptospirosis urine, and 54.5% when working with blood.

Comment: I have a confession to make, regarding my attitude to "pathopanic". It's a word about which I am fairly confident I coined, and about which I am even more confident no-one else will use. It describes the reaction to the discovery of a putative pathogen that is relatively common but usually occult, and once "found", there is a disproportionate reaction to it. The response to discovering a healthy animal is carrying MRSA, is often disproportionate to the concern of antibiotic misuse that has led to the prevalence of MRSA in the first place. The finding of Campylobacter in the faeces of a hospitalised dog leads to barrier nursing, without consideration of the other 36% of cases that are happily shedding the bacteria undiagnosed. If it was rational to barrier nurse all animals that carry putative pathogens, then shouldn't we screen all animals to determine if they have those pathogens? Or do we simply deal with those we serendipitously happen upon? Or as Angelo argues in Measure for Measure, "The jewel that we find, we stoop and take't because we see it; but what we do not see we tread upon, and never think of it." My confession then, is that when I test a hospitalised dog for leptospirosis, but have a low index of suspicion, I have at times not mentioned it to staff unless we subsequently confirm infection, lest the animal be subjected to the altered care of isolation and barrier nursing, and the owner to the expense. That attitude is diametrically opposed to official hospital policy, and I cannot defend the charge that I am playing the odds with the health of staff. So what are those odds with *Leptospira* spp infection in dogs? The study by Guagliardo et al. is perhaps the first to have provided us with an answer. During 2017, there was a spike in clinical leptospirosis cases in dogs iin Arizona - more than 70 in 18 months - and the authors had the foresight to spring into action and follow cases and those who cared for them in 22 clinics. Previous serosurveys of veterinarians have only included, or have been biased by large animal practitioners, and this was the first study of companion animal clinical staff, with a particular focus on nurses and techs. Glove use was low amongst staff, though the authors comment that there are actually no good studies that have determined what adequate, let alone ideal personal protective equipment would be for nursing leptospirosis patients. Many staff reported direct skin contact with blood or urine, nonetheless, none of the 121 people followed developed antibodies. So, a small, but well conducted study of actual hospital practices, and thankfully, no one was infected. It would be an egregious interpretation to infer that caution is not therefore needed when suspected or confirmed cases of leptospirosis are in hospital, but while it is not a cause for complacency, this study can provide some comfort when a diagnosis is made after exposure of staff. And people like me, need to continue to fight against a natural tendency to diagnose pathopanic, when it's actually appropriate health and safety practice, and to ensure that our standard hygiene procedures are followed, in all patients.

Reference: Zoonoses Public Health 2019;66(2):223-31
Abstract

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Considering the relationship between domestic violence and pet abuse and its significance in the veterinary clinical and educational contexts

Authors: Newland X et al.

Summary: This systematic review and thematic analysis examined the relationship between domestic violence and pet abuse in the veterinary clinical and educational contexts. The analysis identified pet abuse as a risk factor for domestic violence, and domestic violence perpetrators may use pet abuse to exert physical, psychological or emotional control of their partner. Veterinarians therefore may act as frontline professionals in the recognition of the link between domestic violence and pet abuse and should assess individual cases for diagnostic indicators of non-accidental injury. Despite legal and ethical obligations of veterinarians, they are often uncertain and unprepared to address domestic violence and pet abuse in the clinical context. Many factors may contribute to a lack of veterinary intervention, including animal welfare concerns, confusion about reporting processes and uncertainty identifying accidental versus non-accidental pet injuries.

Comment: In this provocative review from the Department of Veterinary Sciences at Melbourne University, the authors cite evidence from several countries to amass a substantial case for the widespread association between domestic violence and pet abuse. We are presented with sobering figures of the numbers of veterinarians who report having been exposed to suspected or confirmed cases of pet abuse, a realisation that is softened slightly by the very high proportion of veterinarians that agree we have a moral responsibility to act. The authors suggest that there is discordance between our recognition of responsibility, and our uncertainty as to what the best course of action is. On that point at least, I don't see the discordance, and the two positions seem clearly compatible. And despite the high proportion of vets in several countries that report having seen pet abuse, the authors present a considerable amount of information describing the clinical and historical signs that should alert clinical staff to the possibility. They do not present any data to suggest it is undiagnosed, yet it is framed as if it poses a peculiar diagnostic challenge. But the reason this paper has unsettled me is that the authors suggest that inadequate education of veterinary undergraduates is to blame for professional lack of action, and that increased education is a solution. The authors lament the dearth of material in veterinary curriculae across the world, but as I write this, I'm still not sure I agree. As to the association between pet abuse and domestic violence, how much is required to be taught other than that the association exists? And I am unconvinced that the diagnosis of abuse presents a special challenge. But it is on the question of teaching an appropriate response that I shift most uncomfortably in my seat. What do *you* think I wonder? That there is a desire for us to be omnicompetent professionals is undeniable, and if we can act appropriately, then we should. But what is needed before education, is knowledge as to the best method of intervention. We cannot teach what we do not know. Appropriate intervention is surely culture and context specific, and might differ between individual veterinarians. It seems implausible that one approach would suit all circumstances, but where is the certainty that any approach is better than another? And even if we did know what approach works, why is it necessarily the case that it should be taught to undergraduates? Why could what is taught, not be simply the number to call for help? Not everything has to be, or should be taught at undergrad level, and some things are considered post-graduate level. Most of us were young when we graduated, had little life experience outside typically supportive and abuse-free families, and were hardly the demographic for confidently addressing suspicions of domestic abuse on our first day in practice. The authors point out that data on the efficacy of undergraduate education are lacking. Perhaps more importantly, data on the effectiveness and consequences of any intervention are lacking. This is most certainly a subject where unproven approaches might be severely misguided, so what should be done? John Stuart Mill said in 1867: "Bad men need nothing more to compass their ends, than that good men should look on and do nothing." That sentiment is shared by these authors, and by me, and few would disagree. But I am left wondering what the action should be, and if the lecture theatres of veterinary schools are the best places for the good men and women of our profession to learn that.

Reference: N Z Vet J. 67(2):55-65
Abstract

A comparison of the rates of postoperative complications between dogs undergoing laparoscopic and open ovariectomy

Authors: Charlesworth TM and Sanchez FT

Summary: This retrospective study (2013-18) investigated whether laparoscopic (n = 154) or open surgical (n = 106) ovariectomy had a lower overall and wound-healing complication rate in dogs. Clavien-Dindo grading indicated that, 44% of dogs undergoing open ovariectomy developed a complication, 28.3% had wound healing complications, 5% had superficial site infections, 4% had incisional herniations and 1% had a deep surgical site infection. Among dogs undergoing laparoscopic ovariectomy 20% developed a complication, 11% developed wound-healing complications, 3% developed superficial surgical site infections, and there were no deep surgical site infections.

Comment: Over the past 10-15 years, the orthodox approach to sterilising bitches has changed. In 2006, a review from Utrecht collated evidence to prefer ovariectomy over ovariohysterectomy, though I suspect it is still uncommonly practiced in New Zealand (van Goethem 2006). The decreased surgical morbidity is the main argument for ovariectomy, combined with the absence of significant increases in risk when the uterus is left in place. With the preference for ovariectomy, the argument for a laparoscopic approach became stronger. Initially, there was the preposterous suggestion that bitch speys might become a referral surgical procedure, but now, there has been a slow and steady increase in the availability of laparoscopes in non-specialist clinics. I couldn't find data on how common laparoscopic speys are around the world, but it is far from rare. As is often the case, the claims of decreased risk preceded the evidence of it by several years, and the surgeons at Eastcott Referrals, Swindon, UK, claim that this paper is the first to document complication rates for both techniques in one hospital. The study was retrospective, and the technique applied to each dog was determined by the owner, although they did not offer laparoscopy to owners of dogs 5 kg or smaller. The statistical analysis was rudimentary to say the least, and no attempt was made to determine if there were systematic differences between the two populations that might account for any differences in surgical complications other than the technique. The laparoscopically operated group had a median weight of 19.6 kg, compared with 9.6 kg in the open technique group, but no real attempt was made to discuss if this might have been influential. Other details of the surgical techniques, anaesthetic regimes, surgery duration, different theatres, different operating times, or even a different distribution of surgical staff between the two techniques were either scant or completely absent. So, the first paper to have compared the two techniques, comes from a private referral hospital with surely a vested interest in the outcome, and is a bare-bones retrospective. I doubt this will be the last word on the subject, but as the first word, it might not make you rush out to buy that shiny scope just yet.

Reference: J Small Anim Pract. 2019;60(4):218-22
Abstract