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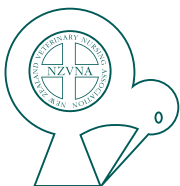
Issue 4 – 2016

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Abbreviations used in this issue

AAFP = American Association of Feline Practitioners
BCS = body condition score
BFM = body fat mass
CKD = chronic kidney disease
HFD = High fat diet
IBD = inflammatory bowel disease
PVAS = Pruritus Visual Analog Scale
RC = Royal Canin
SEC = spontaneous echocardiographic contrast



Welcome to the fourth issue of Companion Animal Research Review.

After last issue's parochial feast, we have prepared a more cosmopolitan banquet, with publications from the US, Canada, Portugal, Australia, Hungary and Greece. For each paper we have summarised the key findings, and attempted to provide some background, criticism, and relevance. It would be an overstatement to suggest that all of these are important studies that will change the way you practice, but I hope you will agree that some of it is useful, some is surprising, some even amusing, but all is at least interesting.

As always, we welcome your feedback of our selection for this issue, your comments on our commentary, and your suggestions for future issues. Until then, we hope this serves to stave off a few ennui filled moments, or at least provides an alternative to Sudoku over lunch.

Kind regards,

Dr Nick Cave

nickcave@animalhealthreview.co.nz

Research Review thanks MSD Animal Health for their sponsorship of this publication, and their support for ongoing education for animal health professionals.

Rebound hyperglycaemia in diabetic cats

Authors: Roomp K and Rand J

Summary: This retrospective analysis of 10,767 blood glucose curves from 55 cats receiving glargine under an intensive blood glucose regulation (median five blood glucose measurements/day) was conducted to determine rebound hyperglycaemic event rates. Biochemical hypoglycaemia frequently occurred, but rebound hyperglycaemia with insulin resistance occurred as four isolated events in four cats. In 14 (25%) cats, a median 1.5% of blood glucose curves were consistent with rebound hyperglycaemia without an insulin resistance (0.42% of blood glucose curves).

Comment: One of the oft discussed diabetic complications is hyperglycaemia induced by counter-regulatory responses to an excess of insulin, eponymously known as the Somogyi effect. In the study by Roomp and Rand, the authors reported the results of blood glucose curves from 55 cats, the owners of which had diligently conducted between six and 763 curves on their own cats. In a whopping 10,767 curves, 45, or 0.45%, included rebound hyperglycaemia. The authors make much of the fact that rebound hyperglycaemia was very rare, and warn against reducing the insulin dose in a poorly regulated diabetic cat as a test to see if the Somogyi effect is present. However, some points bear consideration. Firstly, it is misleading to focus on the number of curves, since well-controlled diabetics that are sampled frequently will have almost certainly biased the data. Indeed, it is the proportions of cats we treat that might experience rebound hyperglycaemia, rather than the number of potential curves in which we observe it, that we are interested in. From this data, one should consider that 25% of the cats experienced rebound hyperglycaemia at some point. Secondly, all cats were exclusively fed very low carbohydrate diets, and in all cases when the owners detected hypoglycaemia, they gave the cats a "snack" of carbohydrate. Thus, the rebound effect may not have been a counter-regulatory response at all. In our feline colony we have observed that even normal healthy cats cannot easily rebound from insulin overdose when fed a very low carbohydrate diet. So the authors were perhaps inexact to suggest the incidence of rebound hyperglycaemia based on the number of curves, but it is probably much less than 25% of cats. None-the-less, this study did emphasise that the combination of glargine insulin and avoidance of dietary digestible carbohydrate are extremely effective, and when poor control is present, the Somogyi effect is a very unlikely differential.

Reference: *J Feline Med Surg.* 2016;18(8):587-96

[Abstract](#)

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A pilot study exploring the effects of musical genres on the depth of general anaesthesia assessed by haemodynamic responses

Authors: Mira F et al.

Summary: This small study in 12 cats undergoing ovariohysterectomy examined the effect of music from different genres (pop, classical and heavy metal) on depth of anaesthesia measured through changes in arterial blood pressure and heart rate. Music exposure induced significant blood pressure changes with lower values recorded when exposed to classical music, intermediate values with pop and higher values with heavy metal.

Comment: Regular readers, if a publication of this age can be said to have any, may remember discussion of an article that demonstrated the efficacy of music in reducing stress in kennelled dogs, raising the intriguing question of to what extent the dogs are consciously appreciating the music. In this present study, the authors delved into the subconscious response to music in anaesthetised cats. The authors have already published a study showing physiological responses to music under anaesthesia,¹ but in this study, they tested the difference between musical genres. Their archetypal choices were Samuel Barber's heavenly "Adagio for Strings", "Torn", by the angelic, if banal Natalie Imbruglia, and "Thunderstruck" by the somewhat less seraphic, though less prosaic AC/DC. The music was played on an MP3 player set to the same volume, delivered through headphones. Though quantitatively unimpressive, there were differences in heart rate and systolic blood pressure. If one is surprised that the musical genres had different effects, then one at least might not be surprised that the caustic crooning of AC/DC was more excitatory than the soothing string ensemble. It certainly raises an interesting question of whether thrash metal could be considered a reasonable alternative to vassopressors during surgery. Alternatively, could tranquillity in the theatre come with an increased risk of hypotension? Surprisingly perhaps, in this study only one cat became hypotensive, which was explained away as an outlier by the authors. Surprisingly, the authors did not consider the alternative explanation, which was that the cat, listening to Natalie Imbruglia at the time, might have simply been losing the will to live.

Reference: *J Feline Med Surg.* 2016;18(8):673-8

[Abstract](#)

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Animal Health publications are intended for those with a professional interest in the animal health sector.

2016 AAFP Guidelines for the Management of Feline Hyperthyroidism

Authors: Carney HC et al.

Summary: The 2016 American Association of Feline Practitioners (AAFP) Guidelines for the Management of Feline Hyperthyroidism discuss etiology and pathogenesis, recognition of health significance of early presentations, how early to treat, whether to treat in the presence of comorbid conditions, and how to manage comorbid conditions including chronic kidney disease and cardiac disease. It also makes recommendations to avoid misdiagnosis and separates the diagnosis into six clinical categories and management strategies.

Comment: Feline hyperthyroidism is a disease about which most of us feel comfortable. We are comfortable with the diagnosis and, given its incidence and predictable response to therapy, we are comfortable with the treatment. So one might ask what need is there for weighty guidelines? Is there something uncomfortable that we are hitherto ignorant of? The AAFP Guidelines are the product of eight specialist feline practitioners, and it may not be a coincidence that only one is a university academic, and that the article is far from being a vexatious read. The article starts with a whistle-stop tour of the history, clinical signs, diagnosis, and speculated causes of hyperthyroidism. Such is the brevity of the sections that one wonders why it was considered necessary at all, since no new analysis or insight was provided. However, it may be of some comfort to the reader to know that there is nothing particularly new, and that there is no need for clichéd talk of shifting paradigms. In fact, "comforting" is a reasonable term for the whole article, as it avoids unnecessarily protracted discussions and is one of the less somnambulist of its ilk. One potentially redundant contrivance is the creation of six different "groups" of hyperthyroid cats, which creates a sense of complexity where perhaps none exists. Indeed, a hallmark of the paper is the otherwise commendable clarity; and treatment of hyperthyroidism, regardless of co-morbidities, is unreservedly encouraged. The reader is repeatedly disabused of any tendency to believe that hyperthyroidism is a crutch for failing renal function, and if there was a unifying recommendation it would be to treat, with whatever modality you choose, monitor, and manage *hypothyroidism* should it occur. Perhaps that last point is new to some of us. It is now well established that hypothyroidism accelerates chronic kidney disease, but many would be excused if monitoring patients for 6 months after surgery or iodine-131 was not a staple recommendation. It is also known that hyperthyroidism elevates the serum N-terminal pro B-type natriuretic peptide (NT-proBNP) concentration, but I had not considered using the assay in treated cats when there is evidence of cardiomyopathy. The panel's recommendation to further evaluate for hypertrophic cardiomyopathy if the concentration remains elevated 3 months after treatment seems prudent, if not yet strictly founded. But the panel's pragmatic approach to selection of treatment modality was for me the most refreshing feature to this publication. The concise summaries of advantages, disadvantages, and the brief but sensible consideration of cost, were all couched on the recommendation to establish what the best modality is for every particular cat and owner. And that is a couch I found most comforting.

Reference: *J Feline Med Surg.* 2016;18(5):400-16

[Abstract](#)

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Prevalence and clinicopathological features of triaditis in a prospective case series of symptomatic and asymptomatic cats

Authors: Fragkou FC et al.

Summary: This prospective study examined the frequency of enteritis, cholangitis and/or pancreatitis (a combination of all three is referred to as triaditis), in 20 symptomatic, 27 asymptomatic and eight normal cats. Of the 47 cats with inflammatory lesions, 13 (28%) had histopathologic inflammatory bowel disease (IBD) lesions, six (13%) had cholangitis, one (2%) had pancreatitis, while 27 had inflammation involving >1 organ. Specifically, 16 (34%) had concurrent IBD and cholangitis, three (6%) had IBD and pancreatitis, and eight (17%) had triaditis, which occurred only in symptomatic cats. A positive correlation was observed between IBD lesion severity and the number of comorbidities ($\rho = +0.367$; $p = 0.022$).

Comment: I have at times cringed so intensely when hearing the term “triaditis” that I have experienced scrotal tightening. The suggestion that anything is achieved by shoe-horning three poorly defined and frequently misdiagnosed diseases of unknown aetiology into one entity is anathema to me. An amalgam of uncertainty is unlikely to illuminate. In the study by Fragkou et al., the authors claimed to have determined the prevalence of coexistent IBD, pancreatitis, and cholangitis. For a control population, the authors collected biopsies from 39 healthy young cats presented for spaying, hence there was a marked difference in age and sex between populations, which was barely discussed. In addition, of the 39 normal cats enrolled, 11 were excluded due to occult unrelated disease, and 20 had some histological evidence of inflammation in one of the three tissues of interest. Those 20 were subsequently “diagnosed” with asymptomatic disease, including IBD, pancreatitis, and cholangitis. Added to that group, were some of the “symptomatic” cats that did not have clinical signs at the time of the biopsy. At various points the authors consider all 47 “diseased” cats together, and at others, they were considered as symptomatic and asymptomatic. All of these points make the reader shift uncomfortably, and question what a histological diagnosis of “...-itis” means in a clinically healthy young animal. The authors’ cause is not helped by the observation that the IBD histological score was not significantly different between symptomatic and asymptomatic cats, and in fact there was no significant difference between symptomatic and asymptomatic cats in the severity of histopathological lesions of any organ either. All these criticisms somewhat deflate the basis for the group allocations, and much of the discussion in this long paper seems tedious and questionable. However, the message for me in this paper was that the IBD scores were greater in cats that had concurrent pancreatic and hepatic inflammation than in those with just enteritis. The authors suggested that IBD might “play a predominant role in the syndrome.” Instead, I suggest that we consider that chronic inflammation of the intestine can “spill over” to involve others. In humans, sclerosing cholangitis occurs in conjunction with ulcerative colitis in at least 80% of cases. Recent studies have suggested that increased numbers of lymphocytes in the liver and pancreas in human IBD are derived from and primed in the inflamed intestine, and that either the liver is induced to express adhesion molecules that trap circulating gut-derived lymphocytes, or that there is inappropriate adhesion molecule expression by the endothelium in those tissues.² In either scenario, it seems likely that the dysregulation of mucosal immunity in the gut is the primary disease. If so, then we should consider concurrent cholangitis and pancreatitis as part of an inflammatory penumbra, and if we can draw one conclusion from the study by Fragkou et al., it is that it may be a sign of severity of the IBD, but not necessarily a discrete disease that requires a name.

Reference: *J Vet Intern Med.* 2016;30(4):1031-45
[Abstract](#)

Association between body condition score and cancer prognosis in dogs with lymphoma and osteosarcoma

Authors: Romano FR et al.

Summary: This retrospective case review examined whether obesity (body condition score), at the time of lymphoma ($n = 270$) or osteosarcoma ($n = 54$) predicted survival time or progression-free interval in underweight (5.5%), ideal weight (54.0%) and overweight (40.4%) dogs. Underweight dogs with lymphoma had shorter survival times ($p = 0.017$) than ideal or overweight dogs. Body condition score (BCS) was not associated with survival time for osteosarcoma, and progression-free interval did not differ between groups with either cancer type.

Comment: At a conference last year, I gave a lecture in which I suggested that we need a diagnostic test for obesity. After the derisive laughter had faded, mostly, I explained it thus: If we define obesity according to a specific body fat mass, then we are left with the difficulty of explaining why some individuals remain healthy at that body fat mass (BFM), whilst others have obesity-related diseases. In humans, it is well established that the risk of disease at any given BFM varies greatly between individuals, and if one maintains a definition of obesity as both a disease *and* a defined BFM, then one is led to the risible oxymoron “healthy obese”. If we wish to maintain that obesity is a disease state without oxymorons or talk of paradoxes, we should agree that the obese state is strongly correlated with, but is not solely determined by BFM. Thus, we should “suspect” obesity in animals as their BFM increases, but that we need markers that detect the disease, not simply the adipose. In humans, obesity is the most important risk factor for type 2 diabetes; and chronic kidney disease (CKD) and myocardial infarction are common and devastating consequences. However, in patients that do develop CKD or cardiovascular disease, the prognosis is better in obese than lean individuals.³ The incidence of several types of cancer is higher in obese than lean humans, but the effect of a higher BFM on prognosis is uncertain.⁴ In the study by Romano et al., the authors retrospectively reviewed medical records, and used the 9-point BCS scale to define BFM, and grouped dogs into three categories (under, normal and overweight). BCS category was not associated with survival of dogs with either lymphoma or osteosarcoma, and the authors were at pains to discuss why they might not have detected an effect of BCS on survival. It was surprising that the most obvious explanation – that there isn’t an effect – was not even considered. However, the authors were correct to point out that their data was unlikely to detect anything other than a huge effect. Since we don’t know at what BFM there might be an effect, the essentially arbitrary tripartite categorisation of their BFM may well have hidden it. What was not hidden, was the association between weight loss after diagnosis and a poor prognosis, though it was not discussed whether the authors thought that the association was causal, or an effect of a more aggressive tumour. So for the time being, we are only slightly wiser, but I feel no less strongly about the need for a better diagnosis of the obese state in dogs and cats. It is clear that increasing BFM increases the risk of cancer, and that weight loss after diagnosis worsens the prognosis, at least of these two neoplasms. Thus, the message remains: stay lean, and until we know more about the efficacy of nutritional intervention, be aggressive trying to prevent weight loss once a diagnosis is made.

Reference: *J Vet Intern Med.* 2016;30(4):1179-86
[Abstract](#)

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Comparative efficacy of intranasal and oral vaccines against *Bordetella bronchiseptica* in dogs

Authors: Ellis JA et al.

Summary: In a randomised controlled trial, 48 Beagle puppies were subject to experimental infection with *Bordetella bronchiseptica* 21 days after intranasal or oral vaccination or placebo. Intranasal *B. bronchiseptica* vaccination reduced rates of coughing, nasal discharge, retching and sneezing versus controls. Oral vaccination reduced coughing and retching, but nasal discharge and sneezing did not differ from controls, and orally vaccinated puppies had higher rates of all four symptoms than intranasally vaccinated puppies.

Independent commentary by Nick Cave.



Nick Cave is a Senior Lecturer in small animal medicine and nutrition at Massey University. He graduated from Massey University (NZ) 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, and 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.

Comment: Over the years of teaching at Massey, I suspect I have been inadvertently immunised against *Bordetella* on multiple occasions, as a failed attempt by a student to administer an intranasal vaccine whilst the syringe tip is firmly pressed against the mucosa results in an explosive aerosolisation into my face as I restrain the dog. Who hasn't, at some point, fought valiantly with the head of an obstreperous hound only to abandon the attempt in favour of an injectable version? These tribulations give some force to the question of whether oral delivery of the mucosal vaccine might be equally effective. An antigen delivered to one mucosal surface can stimulate secreted IgA and activated lymphocytes at other mucosal surfaces, and thus an orally delivered vaccine might result in antigen presentation within oropharyngeal lymphoid tissue, and with rehoming of activated lymphocytes to other mucosal surfaces, immunity in the respiratory tract could be sufficient. Recent research has confirmed that orally delivered vaccines produce protective immunity, and there is now at least one licenced vaccine for oral administration.⁵ Whether it is in any way different to the intranasal product is unclear and probably a proprietary secret. The first study to compare oral and nasal delivery suggested that they were equally effective, and both superior to the injectable product, although that particular study was strangely reported, and the data were inadequately analysed.⁶ This study by Ellis et al. is the first rigorous evaluation of the comparative efficacy of an intranasal vaccine with a licenced oral one, using an uncompromising challenge model. There was no difference in serum IgA and IgG induced by the two different vaccines, nor in bacterial shedding after challenge. However, serum antibody responses are not thought to be the principle determinants of immunity to *Bordetella*, and one can criticise the authors for not having specifically measured mucosally secreted IgA instead. That point was illustrated by the finding that whilst both routes significantly reduced clinical signs following challenge, clinical signs were clearly more severe in the orally vaccinated dogs than those vaccinated intranasally. It may be that the findings are specific to those particular vaccines, and since they were produced by different companies, that may well be the case. The authors duly suggested that the ideal study would compare protection using the same vaccine administered via the different routes. None-the-less, we are limited in practice by what is commercially available. So it seems that the oral vaccine may be superior to injectable versions, but for now, the struggle to deliver the vaccine nasally is still worth the effort, and the occasional accident.

Reference: *Vet J.* 2016;212:71-7

[Abstract](#)

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A randomized, double-blinded crossover trial testing the benefit of two hydrolysed poultry-based commercial diets for dogs with spontaneous pruritic chicken allergy

Authors: Bizikova P and Olivry T

Summary: This randomised, double-blinded, crossover trial in ten dogs with chicken induced cutaneous adverse food reactions was conducted to determine the clinical allergenicity of hydrolysed protein poultry feather and chicken liver diets. Median pruritus Visual Analog Scale (PVAS; owner rated) scores increased after 14 days of a chicken liver diet ($p < 0.001$) but not a hydrolysed poultry feather diet; four dogs receiving the chicken liver diet, but no dogs fed a hydrolysed poultry feather diet, were withdrawn after a flare in pruritus ($p = 0.04$). Maximal PVAS score was higher after chicken liver than hydrolysed protein poultry feather diet (median 4.7 vs 2.5; $p = 0.01$).

Comment: If a company sponsors a study that compares their product with a competitor's, what are the chances that the study will show their product to be inferior? Industry funding of research is a double-edged sword. On one side, companies need to be encouraged to support research that demonstrates the efficacy of their products, and applauded when they enlist independent researchers to do so. On the other side, there is a serious risk of bias, of which under-reporting of negative results is probably the single greatest source. In the head-to-head comparison of two different hydrolysed protein diets reported by Bizikova and Olivry, the sponsor was Royal Canin (RC). I was not surprised that they found clinical signs in dogs known to be allergic to chicken did not worsen when fed the RC hydrolysed diet, whereas 4/10 fed the Hill's hydrolysed diet did. The RC diet is more extensively hydrolysed, and it may even be that the parent proteins from feather do not contain the actual food allergens to start with. But should we still be suspicious of such a finding when the study was funded by the company? In this case, I suggest we can be as confident in the findings as for any other study. The authors (Thierry Olivry is an excellent and rigorous dermatology researcher at North Carolina) cannot be faulted in their attempts to remove any source of bias that can be removed. It was the owners that scored the pruritus, and both the owners and the researchers were blinded to which diet was fed during each trial period, and only after data analysis were they made aware of which diet was which. Thus, as much as for any other study, we can be confident in the albeit uncontroversial conclusion. And whilst I still stand by my recommendation to not use a hydrolysed diet formulated from a protein source that you suspect the patient could be allergic to, there are times when we are forced to. In those cases, based on this small study, we have some confidence to do so. For this study at least, the vested interests of the sponsor do not appear to have impeded the truth.

Reference: *Vet Dermatol.* 2016;27(4):289-e70

[Abstract](#)

Neural mechanisms for lexical processing in dogs

Authors: Andics A et al.

Summary: In a functional MRI study in dogs, researchers analysed whether dogs can segregate and integrate lexical and intonational information. There was a left-hemisphere bias for meaningful word processing that was independent of intonation, while the right auditory brain region distinguished intonationally marked and unmarked words. An increase in activity in primary reward regions of the brain occurred only when both lexical and intonational information were consistent with praise.

Comment: The typically hyperbolic and misleading headlines in the popular press that heralded this article recently were almost enough to put me off reading this paper. "Dogs understand both words and intonation", and "Dogs really do understand human language", were typical fare. But what can be concluded from this trial, where dogs were trained to lie in a MRI scanner while they listened to a series of commands? The study used a 2-by-2 design, where positive phrases they were familiar with (e.g. "well done") were spoken in a positive intonation and a neutral one, and then meaningless neutral phrases they had not been trained with (e.g. "as if such yet") were spoken in the two intonations. It would have been fascinating to have completed the "set" by using negative words, and an admonishing intonation. What would certainly not have been fascinating, was surely the hours of frustration training the dogs to lie still in an MRI scanner. It was imperative they avoided drugging the dogs, but one wonders if they avoided drugging themselves. However, the researchers were principally interested in whether dogs require both lexical and intonational information to correctly mentally process a verbal reward. The first finding was that, independent of intonation, dogs process the sounds of human language more within the left than the right hemisphere when they are familiar with the words, but when they hear unfamiliar words there is no lateralising bias. The second finding was that when the word was delivered with a positive intonation - irrespective of familiarity with the words - it led to activation in the right auditory brain region. However, for activation of the reward pathways, both a familiar reward phrase and positive intonation were required. Since human language processing occurs more in the left hemisphere, some commentators have interpreted this finding as "understanding", having confused "recognition" with "lexical comprehension". There are many other examples of left lateral processing of conspecific sounds in species as diverse as macaques and zebra finches. However, the particular combination of words and intonation could activate neural reward circuits, whereas positive intonation alone could not. It is easy to be distracted by philosophical discussions of how these findings describe the extent to which dogs understand meaning in words. Admittedly, the article in *Science* is typically brief and short on discussion, but I suspect that the authors were more interested in what the study says about humans, than what it says about dogs. Their findings further emphasised that regional processing of "sounds with meaning" is not uniquely human, and did not evolve with the development of complex language. To quote the authors' closing sentence, "What makes lexical items uniquely human is thus not the neural capacity to process them, but the invention of using them."

Reference: *Science* 2016;Aug 30 [Epub ahead of print]

[Abstract](#)

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Retrospective evaluation of the incidence and prognostic significance of spontaneous echocardiographic contrast in relation to cardiac disease and congestive heart failure in cats: 725 cases (2006-2011)

Authors: Peck CM et al.

Summary: This retrospective study during 2006-11 analysed data from 725 cats undergoing evaluation for cardiomyopathy, to evaluate whether spontaneous echocardiographic contrast (SEC) was associated with increased mortality. Cardiac abnormalities identified on echocardiography with SEC increased the risk of death versus those without SEC. Dilated, unclassified, and hypertrophic cardiomyopathy were more likely to have SEC than other types of cardiac disease.

Comment: Of the very limited number of echocardiographic features that I can reliably identify in cats, left atrial “smoke” is one. The hypnotically slowly swirling acoustic patterns seen in the dilated atria of cats with advanced cardiomyopathy are thought to occur as a result of red cell rouleaux or other aggregates within the abnormal eddies of the atrium. A previous study suggested an association between the ultrasonographic appearance and both hypercoagulability and risk of arterial thromboembolism, although that particular study was not really designed to test the strength of the association, or the predictive power of the finding.⁷ In the study by Peck et al., the records of a large number of cats were reviewed to specifically ascertain the diagnostic value of the finding. The appearance of “smoke” was given the unlovely and less evocative term “spontaneous echocardiographic contrast”, or SEC. A cursory read of the paper, and certainly the abstract, suggest that SEC has a strong negative prognostic value, where 76% of cats without SEC were alive at 12 months, whereas only 49% with SEC were. Disappointingly, the description of the statistical analysis were in themselves cursory, and it is not clear what factors were considered as confounding factors, which is a shame, because the study had the power to truly quantify the utility of SEC as a prognostic finding. The authors report that the multivariate analysis revealed that the presence of congestive signs, an increased left atrium to aortic ratio, and age, were all stronger predictors of mortality than the presence of SEC. Thus, the authors could well be accused of over egging their pudding by suggesting the importance of SEC, and much of the discussion ignores the relative importance of the other factors’ association with mortality. For those who do wield the acoustic wand over the chests of cats with cardiac disease, this paper serves to emphasise the importance of those previously established prognostic indicators. However, despite the optimistic wording of the abstract, left atrial smoke is more visually interesting than it is prognostically helpful.

Reference: *J Vet Emerg Crit Care (San Antonio) 2016;26(5):704-12*

[Abstract](#)

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Insulin access to skeletal muscle is impaired during the early stages of diet-induced obesity

Authors: Broussard JL et al.

Summary: This study in male mongrel dogs receiving a control diet (n = 16), or a short- (n = 8) or long-term high-fat diet (HFD; n = 8), used intravenous glucose tolerance tests (testing insulin sensitivity) and hyperinsulinaemic euglycaemic clamp, to examine the access of insulin to skeletal muscle, the major site for insulin-mediated glucose uptake. Both short- and long-term HFDs induced obesity and lowered insulin sensitivity. Insulin concentrations in lymph fluid (representing interstitial space to measure insulin access to muscle) were approximately 50% lower than plasma insulin concentrations under control conditions. A long-term HFD caused fasting plasma hyperinsulinaemia, but lymph fluid insulin concentrations were not altered, suggesting impaired insulin access to muscle.

Comment: This is one of those studies that you are unlikely to ever read. Buried within the human obesity literature, the research is intentionally written as an experimental model for human obesity and, perhaps understandably, absolutely no consideration is given to the implications for canine obesity. HFDs have for some time been of interest to researchers in human nutrition, both as a supposed cause of obesity, but also as a cause of other diseases independent of the obese state. In rats fed HFDs, especially those high in saturated fat, there is an inflammatory response in several tissues, including the brain. Changes to the intestinal microflora, disturbances in intestinal permeability, and increased absorption to bacterial components such as lipopolysaccharide have all been credited. The inflammatory response is, in part, responsible for the more well-known association between HFDs and insulin resistance. The study by Broussard et al. extends our understanding by elucidating other mechanisms for HFD-induced insulin resistance. By feeding a diet with 52% of calories as fat to a group of large cross-bred dogs and allowing them to become overweight, they demonstrated that within 6 weeks, peripheral insulin sensitivity was decreased. A large component of the resistance was the result of decreased insulin transport across the vascular endothelium, in addition to the defect in insulin signalling within muscle. Although the paper was frustratingly written, the findings are compelling, and have significant implications for humans. But what are the implications for dogs? Is a diet of 52% fat (on an energy basis) an unhealthy diet for dogs? The authors did not appear to consider that neither people nor rodents have evolved to cope with high fat consumption, but dogs have. Sled dogs in Antarctica have traditionally been fed diets of >60% fat, and diabetes is not a feature there. Additionally, the authors did not differentiate between the effects of weight gain over HFDs. What happens when lean dogs are fed HFDs and remain lean? If this study shows what I suspect it shows, it is that even a small increase in body fat, perhaps to as little as BCS 6, produces metabolic derangements in dogs. Whether there is any problem with dietary fat independent of body condition remains untested in this regard. Until more canine-specific data is available, we should worry less about macronutrient proportions than about maintaining a lean body condition, and remind our medical colleagues that mice, men, and mutts are not all the same.

Reference: *Obesity (Silver Spring) 2016;24(9):1922-8*

[Abstract](#)

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