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Vol 02 | Issue 06 | December 2015

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* Cole L.K., Kwochka K.W., Kowalski J.J., Hiller A., Howshaw-Woodard S.L. – Evaluation of an ear cleanser for the treatment of otitis externa in dogs, Vet. Ther. Vol. 4(1), Spring 2003.

Editor's Note

Well, we have made it through our first year. It has been a bit of a learning curve but I am confident that our magazine is providing what we promised - concise relevant information for the small animal practitioner.

This edition focuses mainly on the adrenal gland and hyperadrenocorticism. Now that trilostane is available, many veterinarians are diagnosing and treating this disease in general practice and we hope to provide some important guidelines for decision making in diagnosis and treatment.

Also included is an article outlining, once again, the need for feeding of critically ill patients. This is such a simple intervention, but can make a huge difference in patient morbidity and mortality. The website provides loads of information on this and other topics.

I wish you all a relaxing holiday season and for those of you who are working ... this too shall end

Regards

Liesel

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A win-win situation for SAVA, Vetlink and the reader.
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VET360 aims to be a leader in the field of continuing veterinary development in Southern Africa by providing veterinary professionals from diverse disciplines with tools to help them meet the challenges of private practice. The magazine aims to make information accessible, both paper and electronic, and provide clinical, business and other veterinary information in a concise form to enable the practitioner to rapidly acquire nuggets of essential knowledge.

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Employee Relations Within A Vet Practice

Andrew Christie
BComm (Industrial Psychology)

This article is the first in a series of three which deal with maintaining positive and mutually fulfilling employee relations within a Vet Practice.

1 Beginning Recruitment and Selection

2 Middle Managing the employment relationship

3 End The end of the employment relationship

The articles are sequential in that the first deals with the start of the employment relationship (recruitment, selection and appointment), the second explores the managing of the employment relationship (performance management, the grievance procedure and the creation of an HR policy) and the third examines the end of the employment relationship (resignations and dismissals, as well as the disciplinary procedure).

1 The Importance of Getting It Right

There are a number of negative implications for the practice if you do not recruiting and selecting correctly,

Financial implications:

- The process is expensive
- Mistakes made in the recruitment process are extremely difficult to rectify
- There can be very high long-term costs when being forced retaining inappropriate staff
- The cost of dismissing inappropriate staff can be very high
- A high staff turnover rate is costly, amongst other

Andrew was born in Johannesburg and, after matriculating at St Johns, he elected to plunge straight into the business world while completing his BComm in both Business Management and Industrial Psychology through the University of Soåuth Africa. He spent two years at a leading bank in South Africa where he was involved in the implementation of the new labour legislation that was rolled out in 1997, as well as managerial training. He followed this with a stint at a computer-based training company where he assisted in the development of learning centers for major South African corporations. In 2000 he indulged his passion for books by entering into a partnership in a bookshop. Over the next five years he drove the development of that shop into a successful chain of six. In 2005 he re-entered the corporate world by forming his own consultancy, specialising in providing financial, tax and human resource services for SMME's, as well as growth management for medium corporates. The natural extension of this work into developing his clients led to Andrew opening Acacia Training, a learning and development consultancy, specialising in Financial and Human Resource training. As well as training in Europe and all over Africa, Andrew has lectured in Strategic Human Resource Development at a post-graduate level at Wits Business School. **CONTACT DETAILS: andrew.c@acahr.com**

things, as the recruitment and selection process must be repeated more often

Legal implications:
- South Africa's legal requirements and obligations give a great deal of protection to job applicants. If the recruitment and selection process is incorrect, the applicant could take the practice to court and could possibly win the case.

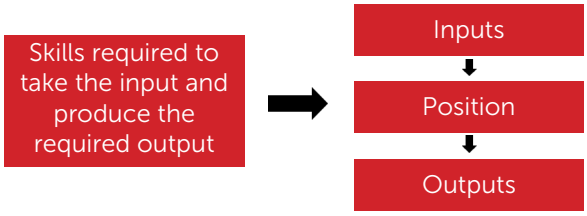
Morale implications:
- If the incorrect person is selected, it is probable that the job will not be performed optimally.
- If the incorrect person is selected, it will affect the motivation of other people in the organisation when they start questioning why a less-qualified person was appointed.

Performance implications:
- The person may not be able to perform the job
- The person may need costly retraining in order to perform the job.

Image/reputation:
- It is difficult to maintain a good practise image if employees are inefficient
- The practice cannot ensure a reputation as equal opportunity employer if poor employment standards are maintained

2 Job Analysis

Recruitment of new staff typically begins with the replacement of an employee who has left. Unfortunately, the focus of the recruitment then becomes seeking someone who has the same skills as the person who has left or, alternatively, who has the skills



that the previous job incumbent lacked. This often leads to problems as superior job applicants may be ignored and superficial decisions may be made based on the applicant's personality or even appearance.

It is suggested that the purpose of the post be examined each time someone needs to be employed. It is useful to perform a job analysis using the below simple model:

The above model shows that the job position exists purely to convert inputs into outputs. For example:
- A receptionist (position) converts customer interaction (inputs) into payments received and repeat visits (outputs)

Job analysis aims to answer the following questions:
1. Why does the job exist?
2. What physical and mental activities does the worker undertake?
3. When is the job to be performed?
4. Where is the job to be performed?
5. How does the worker do the job?
6. What qualifications are needed to perform the job?
7. What are the working conditions?
8. What machinery or equipment is used in the job?
9. What constitutes successful performance?

3 The Job Description

This is a list that a person might use for the general tasks, functions and responsibilities of a position. It may often include to whom the position reports, specifications such as the qualifications or skills needed by the person in the job, or a salary range.

A job description need not be limited to explaining the current situation, or work that is currently expected; it may also set out goals for what might be achieved in future.

A comprehensive Job Description should include:

Job Title	Job purpose
• Division/ Department	• General responsibilities: training; health and safety; equal opportunities; customer care; environment; technology
• Grade / Level	• Hours
• Reports to	• Career grade progression
• Date of issue	• Performance indicators
• Job outline	• Competencies

4 The Job Specification

The Job Specification is a statement indicating the minimum acceptable human qualities which are necessary to perform a job. Job specification translates the job description into human qualifications so that a job can be performed in a better manner

1. Experience:
- Number of years of work experience required for the selected candidate.

2. Education:
- State what degrees, training, or certifications are required for the position.

3. Required Skills, Knowledge and Characteristics:
- State the skills, knowledge, and personal characteristics of individuals who have successfully performed this job, or use the job analysis data to determine the attributes you need from your "ideal" candidate. Your recruiting planning meeting can also help determine these requirements for the job specification.
- The job specification must highlight which of the requirements are essential. This should help potential candidates de-select themselves, so be careful: you may lose otherwise good candidates who don't apply because of your 'essential' item.

In addition to this, it becomes the foundation of performance management.

5 The Selection Process

Sourcing suitable candidates is a vital part of the recruitment process because either too few applicants, or too many may be reached. One of the requirements of legally fair recruitment is that all potential applicants are advised of the vacancy.

- Adverting in newspapers and publications is the most used method. Using a newspaper reaches the most people, but using specialised publications can be extremely useful.
- Word-of mouth referrals are regarded as prejudicial UNLESS the post is adequately advertised, and the WOM candidates follow exactly the same procedure.

The biggest problem with the job applicant process is that it can very easily become very complicated. In fact, the process is quite simple and needs only contain a few basic steps, which are outlined below:

Step 1: Make position known and indicate how applicant must apply
Step 2: Collate applicant information
Step 3: Make shortlist of suitable applicants
Step 4: Interview suitable applicants
Step 5: Select best applicant for the position

Normally a CV is requested from candidates, but an alternative is to have all candidates complete a standard application form; this makes comparison easier, elicits only the relevant information and prevents a deluge of unwelcome paper.

- Compare applications against the job description and person specification outlining the skills and experience you need.
- Eliminate applicants who do not have the basic requirements for the job. Draw up a shortlist - a list of candidates to interview - based on the applicants who most closely match your needs.

Unsuitable Candidates

All unsuccessful candidates must be contacted with a reason for the unsuitability of their application. This can be done by letter or email. Telephonic interaction is not suitable due to the cost and because no record will then exist.

The reasons given should be as specific as possible and must clearly demonstrate the unsuitability of the candidate. A clause indicating that no further communication will take place must be included to prevent a correspondence developing.

Short listed Candidates

Short listed candidates should preferably be contacted by telephone.

You should mention:

- When, where and how long the interview will be
- How to get there - provide a map if necessary - and whether you will pay travel expenses
- What documents the candidate should bring with
- Who the candidate should ask for on arrival
- The names and job titles of the people conducting the interview
- If there will be a test to take, or a presentation and if so, its type and duration

6 The Interview

The interview is all about soliciting information from the job applicant. Because this is largely done through questions and answers, it is important to use the following as guidelines:

Ask relevant questions

This is perhaps the single most important piece of advice. Apart from the ice-breaker session, ALL questions must be relevant to the task – i.e. finding out about the suitability of the applicant for the post.

You may not probe into the candidate's personal interests, tastes, habits or opinions unless you can justify how the answer will tell you directly about the individual's suitability for the job.

Every question you ask of any one candidate must be capable of being asked to all candidates. It is discriminatory to ask a question of a female which you wouldn't ask a male, since to do so implies you are discriminating on the grounds of gender. Thus:

Typically this job requires some evening work. Would you have any objections to working overtime?
This is fair and relevant question, since it provides information relevant to any candidate's willingness to do the job, and can/will be asked equally of all candidates, regardless of gender.

What does your husband think of you moving if you get this job?
This is totally unacceptable, since it is irrelevant to the candidate's ability to perform the job, is personal to the candidate, and would almost certainly not be asked of a married male applicant.

Choose carefully between open and closed questions. A closed question generates a 'yes/no' response or forces the candidate to choose from two or more options, eg: *'Do you like your current job?'*

An open question encourages a wider range of responses, and is more likely to get the candidate talking, eg: *'What do you like about your current job?'*

Open questions are good for getting the candidate talking; closed questions are good for checking and clarifying what's been said, as well as for establishing facts. However over-use of closed questions can

make the interview seem like an interrogation, and can intimate the candidate.

Typically an interviewer will ask an open question, then follow up with closed questions as s/he seeks to clarify or probe the initial response, eg:

Q:'How would you feel about being in charge of other people for the first time?' (open)
A: 'A little uncomfortable'

Q: 'In what way uncomfortable?' (open)
A: 'I'd be worried about mistakes'

Q: 'Do you mean mistakes you'd make, or mis takes your staff might make?' (closed)
A: 'Mistakes I'd make'

Q: 'How worried do you feel about this?' (open)
A: 'Very'

Q: 'Enough to prevent you taking the job?' (closed)
A: 'No'

Avoid leading questions
As their name implies, leading questions lead the candidate to an answer you probably want to hear. They encourage a particular response, and thus the candidate is more likely to give an answer as/he thinks you want, rather than their own, eg:
'What do you think of the idea?'
'Do you find the job stressful?'
Instead of:
'What do you think of this stupid idea?'
'How do you handle the strain of the job?'

Treat the candidate as an equal
Avoid pompous put downs; don't be patronising; if you come over as arrogant or superior, the candidate won't open our to you, eg:
'Please tell me about your HNC, and whether it helped you in your current job'
Rather than:
'Do you think sub-degree qualifications like your HNC are of any use?'

Speak in plain English
You want the candidate to understand; don't make it difficult. Also, If you use jargon, s/he may think s/he has to reply in a like manner. On the other hand, if you speak plainly, the candidate is more likely to do the same – eg:
'Do you prefer working alone, or in a group?
Rather than:
'Are you an isolate, or do you have preference for social interaction with your peers?'

Ask one question at a time
The interview is a strain for most candidates. Don't add to the pressure by asking the candidate to hold two or three questions in his/her head. A multiple question can also sound like a trick question.
'which bit of that does he want me to answer?'
'what did you study ?
Then
'has it helped you with your current job?'
Then
'Would you recommend the course?'
Rather than:
'Tell me about the HNC – what your studied, whether you enjoyed it, what you thought of it and whether you'd recommend it to anyone else'.
Keep your questions short and simple
Long questions intimidate and confuse the candidate – and do nothing for your reputation! eg:
'What do you find most enjoyable about your current job?'
Rather than:
'Tell me about your current job. Do you enjoy it? What do you like about it most – the job itself, the people, the workplace?'

Avoid ambiguous questions
These are usually the hardest mistakes to spot, since we never intend to be ambiguous – the meaning of the question is always perfectly clear to the questioner! eg:
'Can you manage women as well as men?'

Could mean:
1. Having supervised men, would you be able to supervise women also?
2. Can you supervise a mixture of men and women, rather than just men?
3. Are you able to manage women as skillfully as you manage men?

Taking Notes
You need to have some record of the interview:
- So you can discuss/compare performance
- As proof/evidence of what was said
- As a reminder for further action
- Because memory fades

Writing as you go along can be difficult and distracting, and takes valuable time. Regular recaps and summaries are preferable; they provide suitable breaks to the flow, and show you've been listening; it also reassures the candidate that you've got the details right.

Another alternative is a pre-prepared tick sheet; these tend to be quick and easy to use, and help structure the interview. But they have two disadvantages. Firstly, candidates worry about the interviewer 'ticking' or 'crossing' sections of a sheet of paper; it distracts and can intimidate. Secondly, they need careful preparation.

Mechanical recordings can be used, but they are also intimidating to the candidate – and can break down!

Finally, you can have someone in to take notes. But

The Interview Checklist

- Can I cover these questions adequately in the time available?
- Are any questions directly or indirectly discriminatory?
- Can I put this question to each candidate?
- Would I consider this question fair, if I were in their shoes?
- Would a tribunal?
- Do I know what knowledge, skill or experience area I am testing by asking this question?
- Do my questions cover the person specification? If not, how are the gaps covered (eg application form or other form of testing)?
- Are all these questions relevant? What is the purpose of each question?
- Are the questions clear?
- Are there any leading questions?
- Have I thought of follow up questions?
- Do the questions allow waffle answers, or do they probe?
- Have I some means of recording the answers?
- Have I any prejudice or bias in favour of, or against, any of the candidates?
- Can I justify every question to:
  - Each candidate
  - My line manager
- Have I left enough time for:
  - Their answers
  - Their questions

it adds to the numbers, causing further pressure on the candidate; and secondly, the quality of the record depends very much on the quality of the note taker. My own preference is for the second option – regular recap and record. It is courteous to tell the candidate that you're taking notes

After the Interview: Pre-Employment Checks
Employers have increasingly turned to pre-employment screening as a critical risk-management tool to try and avoid hiring problem employees in the first place.

Pre-employment background screening has 4 major benefits:

1. Just having background screening can discourage applicants with something to hide. A person with a criminal record or false CV will simply apply to a practice that does not pre-screen.
2. It limits uncertainty in the hiring process.
3. A screening program demonstrates that an employer has exercised due diligence, providing a great deal of legal protection in the event of a lawsuit.
4. Having a screening program encourages applicants to be especially forthcoming in their interviews.

Finally, the recruiter must remember to keep all documents pertaining to EVERY application for a period of two years. This is partly so that documentation pertaining to a potential CCMA claim is evident. Some of the key documents that must be kept could be:

- A CV
- Completed Application Form
- Certified copies of requested documents, such as ID, qualifications, etc
- Information pertaining to any credit or employment checks
- List of questions asked in the interview (including the pre-screening) and the answers

Legally, unsolicited CV's and applications need not be retained, processed or responded to.

7 The Employment Contract

Once the applicant has been selected, a job offer must be prepared and presented to the successful applicant. It is an unfortunate fact that South Africa has a high unemployment rate and job applicants may be so desperate for work that they accept relatively poor conditions of employment. Once employed, the level of desperation decreases, only to manifest itself as job dissatisfaction. This can lead to costly legal action but certainly to problems in the workplace.

So, when drafting the job offer it is important to ensure it is totally compliant with the legal requirements. The conditions of employment are set out mainly by the Basic Conditions of Employment Act (BCEA). If something is not specified by the BCEA, then common law principles apply. Similarly, if something is included in the Constitution, but is different in the BCEA, then the Constitution takes precedence.
The BCEA requires the following (as a MINIMUM) to be included in all Contracts of Employment:

1. Employer's name and address
2. Employee's name
   Employee's occupation or job description
3. The place of work
4. The date of commencement
5. The ordinary days and hours of work
6. The wage and / or the wage rate
7. The other payments to which the employee is entitled
8. The date when remuneration will be paid
9. Details of any deductions that will be made
10. The amount of leave that can and must be taken
11. The period of notice, or date of termination of contract
12. A list of any other documents which form part of the contract

Failure to give written particulars does not nullify contract

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ROYAL CANIN

Dealing With Incidental Adrenal Tumours

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An adrenal "incidentaloma" is an adrenal mass, found as an incidental finding during ultrasound or CT examination of the abdomen, where adrenal pathology is not initially suspected In humans the incidence varies from <1% to 7%, and increases with the age of the population

In canine and feline veterinary patients, most incidentally discovered adrenal masses, in otherwise healthy patients, are non-functional benign tumours or non-neoplastic lesions. Most functional adrenal masses are either cortisol secreting tumours or pheochromocytomas. In veterinary medicine there is no consensus on the best approach to an incidentaloma. Deciding upon an appropriate treatment plan requires classification of the incidentaloma as malignant or benign and functional or non-functional.

Normal adrenal gland anatomy and physiology

The adrenal gland consists of an outer cortex and an inner medulla. The latter is the site of catecholamine production. The cortex has 3 zones: from the outside to the inside these are the zona glomerulosa, the zona fasciculata and the zona reticularis. The zona glomerulosa is responsible for the production of mineralocorticoids (aldosterone). Glucocorticoids (cortisol) and androgens are synthesized in both the zona fasciculata and reticularis.

In human medicine the causes for adrenal masses include:

a. Functional masses (up to 15%): adenoma (aldosterone or cortisol); carcinoma (any adrenal hormone); pheochromocytoma; congenital adrenal hyperplasia; massive macro nodular adrenal disease; nodular variant of Cushing's disease
b. Non-functional masses: adenoma; myelolipoma; neuroblastoma; ganglioneuroma; haemangioma; carcinoma; metastasis; cyst; haemorrhage; granuloma; amyloidosis; infiltrative disease

Hormones that are released from functional masses include: cortisol or one of the precursors (Cushing's disease); aldosterone (Conn's syndrome); sex hormones and adrenalin (pheochromocytoma).

The canine patient may be completely asymptomatic, as would be expected in a non-functional, small benign adrenal adenoma. Patients with functional adrenal masses may present with signs of the underlying hormone excess. Animals with malignant tumours (functional or non-functional), may show non-specific signs such as decreased appetite, weight loss, lethargy and nausea. Should metastasis be present, clinical signs will further depend on the organ/s which have been affected.

Surgical removed of functional or malignant adrenal masses would be ideal. However, not every mass needs to be removed and surgery holds significant risks to the patient, either due to age related anaesthetic issues, co-existing morbidity or due to surgical complications. On the other hand, surgically removing a pre-metastatic adrenal malignancy may be life-saving.

General guidelines for surgical removal include masses that are larger than 3cm, show signs of malignancy, are functional or show invasion of the surrounding blood vessels. It may not be easy however, to determine the malignancy of a mass without cytological or histological evaluation. The larger the mass the greater the likelihood of it being malignant. Both CT scanning and abdominal ultrasound have been

shown to be sensitive for the detection of invasion of blood vessels.

Although ultrasound was sensitive for the detection of thrombi in the caudal vena cava, it was only 75% sensitive for detecting all forms of invasion. In one study, invasion of the surrounding blood vessel was shown to occur via the phrenico-abdominal vein as opposed to the erosion of blood vessel walls. Pheochromocytomas seem to have predilection for invasion of blood vessels. Between 36-70% of all adrenal masses (benign or malignant) are seen to invade the surrounding blood vessels.

In humans, CT scanning is an additional factor that is used to decide on adrenalectomy. Benign masses tend to have a Hounsfield unit of <10% and a >50% contrast washout. Inhomogeneous masses with an irregular border are less likely to be benign, especially if the HU is > 20. In those cases that warrant surgical removal, pre-operative investigations for metastasis should be done.

A CT scan of the thorax and abdomen has a higher sensitivity for detecting metastatic lesions than thoracic radiographs and abdominal ultrasound. As discussed previously, in human medicine, the CT density of the adrenal mass has diagnostic significance.

Masses that are <3cm in size, those that are not producing hormones and those that show no invasion of the surrounding blood vessels may be left for careful observation. The size of the adrenal mass is reassessed at 1, 2, 4 and 6 months after initial diagnosis. Those masses increasing in size should be considered for adrenalectomy. The non-enlarging, stable mass should continue to be observed at 4-6 month intervals.

Pre-operative attempts to diagnose the etiological cause for the adrenal mass are essential. Surgical excision of an undiagnosed pheochromocytoma carries a significant risk for peri-anaesthetic / operative morbidly and mortality. These can be significantly lowered by medically managing this endocrine disorder pre-operatively. Functional adrenal testing is therefore essential prior to surgical removal – especially to detect the presence of a pheochromocytoma.

Adrenal functional testing

Pheochromocytoma: Tachycardia and hypertension are the hallmarks of catecholamine release, but hormone release may be episodic. Patients may, therefore, be completely normal during the physical examination – and be normotensive. A wide variety of clinical signs may be noticed by the owner, including: collapse/weakness; panting, tachycardia/arrhythmias, restlessness, inappetence/anorexia, nonspecific lethargy, exercise intolerance, polyuria/polydipsia, and weakness.

Testing for the presence of a pheochromocytoma in dogs has centred on the measurement of adrenalin metabolites in the urine, either absolute amounts per 24hrs or as measured by a ratio to creatinine. Ideally, a first morning urine sample is collected and a small amount of acid is added to the prevent degradation of the metabolites. Metanephrine and normetanephrine to creatinine ratios are readily available to veterinarians in South Africa through use of the human clinical pathology laboratories. Pheochromocytomas should always be considered malignant in dogs, with metastasis occurring in up to 40% of affected dogs.

Hyperaldosteronism: (Conn's syndrome). This condition is suspected in patients with hypokalaemia, hypernatremia and hypertension. All findings are not consistently present in all patients. So, while identifying these electrolyte disturbances in a patient with an adrenal mass is supportive for this diagnosis, the absence of these findings do not exclude a case of Conn's syndrome.

The condition is rare in dogs and cats. Low renin and high aldosterone serum concentrations are useful in diagnosing those suspected patients with unremarkable serum electrolyte concentrations. Aldosterone levels are easily tested for, renin requires special collection practices.

Hyperadrenocorticalism: (Cushing's disease) Both the ACTH stimulation test and low dose dexamethasone may be used to diagnose Cushing's disease. Most patients should have clinical signs consistent with Cushing's disease. In some cases it may be more appropriate to exclude hyperadrenocorticism as a differential. The urine cortisol to creatinine ratio is a highly sensitively test, but not very specific. Therefore it has a high negative predictive value. Patients with a negative result are unlikely to have Cushing's; while a dog that tests positive may or may not have hyperadrenocorticism.

References

Reference available from the Author on request.

Diagnosis of Hyperadrenocorticism

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Hyperadrenocorticism (HAC) occurs as a result of hyperplasia or neoplasia (Adrenal dependent hyperadrenocorticism - ADH) of the zona fasiculata of the adrenal cortex resulting in overproduction of cortisol.

Hyperplasia of the adrenal gland results from an adenoma of the pars distalis or pars intermedia of the pituitary gland (pituitary dependent hyperadrenocorticism - PHD).

Serum cortisol has a negative feedback effect on the production of ACTH and CRH by the pituitary and the hypothalamus. ACTH also has a negative feedback effect on the hyopthalamus. In dogs the secretion of ACTH is pulsatile with 6-12 peaks in a day.[5]

Any breed can develop hyperadrenocorticism but in general middle aged to older small breed dogs develop PDH and older large breed dogs develop ADH.

The most common clinical signs associated with hyperadrenocorticism include polydipsia and polyuria, polyphagia, pattern alopecia (bilaterally symmetrical affecting flanks, ventral abdomen, neck and perineum), muscle weakness, thinning of skin, abnormal fat distribution with pendulous abdomen and hypertension. Rarer signs include calcinosis cutis and neurological signs.

Typical clinicopathological abnormalities include an increase in alkaline phosphate (ALP) activity in 85% of dogs. Synthesis of this enzyme is induced by cortisol in the dog. There may be mild hyperglycaemia. Hypercholesterolaemia and hypertriglyceridaemia are usually present.

The RCC, Hb and PCV/ Ht. may be slightly elevated Thrombocytosis may be present and the characteristic stress leukogram of mature neutrophilia, lymphopaenia and monocytosis is present.

Urine SG is <1.015 and usually in the hyposthenuric range. Proteinuria is frequently present and urinary tract infection is frequently found.

Specific diagnostic tests:

A. Urine cortisol:creatinine ratio
B. ACTH stimulation test
C. Dexamethasone suppression test
D. Endogenous ACTH

A. Urine cortisol: creatinine ratio

The urine cortisol: creatinine ratio (UCCR) has a high sensitivity for HAC but low specificity. The normal reference range is <10 (<10 x 10^{-6} – convention is to express this as 10), which effectively rules out a diagnosis of HAC. The test has a good negative predictive value.

Due to the effect of stress the test only becomes more reliable when the cut-off is set at >100 (90% probability of PDH). Thus a high ratio does not confirm HAC and further discriminating tests need to be done.

The cortisol and creatinine concentrations are measured on the same urine sample and the ratio is calculated as follows:

$$\text{Cort: Creat} = \frac{\text{urine cortisol concentration (nmol/l)}}{\text{urine creatinine concentration (µmol/l)}}$$

B. ACTH stimulation test

The method for the ACTH stimulation test in dogs is as follows:[1]

- The test can be started at any time of day
- Collect a blood sample for the basal cortisol concentration
- Inject 250µg of synthetic ACTH i.v or i.m. (125ug synthacten IM is effective)
- Collect a second sample 60 mins later.

Interpretation:
The baseline cortisol concentration is irrelevant

- A post stimulation concentration of >600nmol/l is consistent with a diagnosis of a diagnosis of HAC in a dog with typical clinical signs and showing no evidence of other clinical disease.
- This test reliably identifies approximately 84% of dogs with PDH and 51% of dogs with ADH2

Advantages:

- The biggest advantage of this test is its ability to distinguish between iatrogenic and spontaneous HAC[3]
- It can be used to monitor response to treatment
- It is quick to perform
- It provides baseline information for treatment

Disadvantage
- It does not distinguish between PDH and ADH
- It is not very sensitive in detecting HAC caused by adrenal neoplasia

C. Dexamethasone suppression tests:
Low dose Dexamethasone suppression test (LDDST)
The low dose dexamethasone suppression test is an alternative to the ACTH stimulation test for the diagnosis of HAC.

Method:
- Collect a blood sample for the basal cortisol concentration
- Inject 0.01 mg/kg dexamethasone iv
- Collect samples at 4 (-6) and 8 hours post-dexamethasone injection.

Interpretation (Figure 1)
- A normal response is suppression to below 40 nmol/l
- A complete lack of response could be consistent with PDH or ADH
- In most PDH cases, there will be suppression to below 40 nmol/l at 4-6 hours and "escape" at 8 hours with an increase in cortisol concentrations.

Advantages
- The sensitivity is higher than the ACTH stimulation test (90-95%), thus fewer cases will be missed.
- It has an almost 100% sensitive with adrenal neoplasia and 90 -95% sensitivity for PDH

Disadvantages
- The specificity however is very low if measured in sick dogs (44%- 73%) - thus a ≅ 50% chance of false positives in non-adrenal illness. [5]
- The test takes a long time (8h)
- It does not distinguish between iatrogenic and spontaneous HAC.

A disadvantage of both the ACTH stimulation test and the LDDST is that false positives may occur in animals with other chronic diseases. These include renal and hepatic disease, diabetes mellitus, neoplasia and chronic inflammation.

False positives can be reduced by selecting patients carefully. Typical clinical signs of HAC should be present in patients selected for these tests and concurrent disease should be ruled out by clinical examination, clinical pathology and imaging.

High Dose Dexamethasone Suppression Test
This is NOT a screening test. It is used for discriminating between pituitary-dependent and adrenal-dependent hyperadrenocorticism. It has become less frequently used since ultrasound is available in most practices.
Method:1,3
- Collect a blood sample for the basal cortisol concentration
- Inject 0.1 mg/kg dexamethasone iv

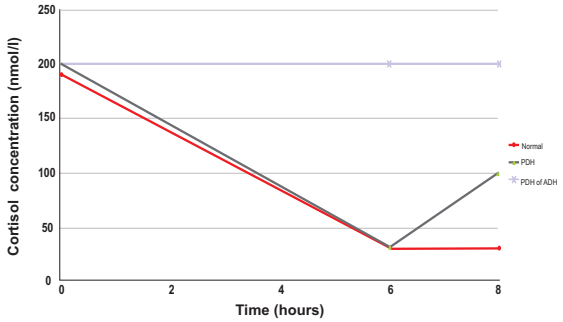

Figure 1

- Collect samples at 6 and 8 hours post-dexamethasone.

Interpretation:
- No suppression is consistent with ADH. The neoplastic cells function autonomously and are not subject to feedback suppression.
- Suppression to below 40 nmol/l is consistent with PDH.

D. Endogenous ACTH
This is the preferred test for HAC in horses but has not been used extensively in dogs.

Method:
The sampling conditions are very specific.
- The blood should be taken into chilled EDTA-aprotinin tubes, placed immediately on iced and separated immediatly refrigerated centrifuge[4]
- The plasma should be transferred into a PLASTIC tube and immediately frozen and sent to the laboratory frozen.

Interpretation[3]
- Normal dogs have an ACTH concentration of 3-10 pmol/l
- Dogs with PDH have concentration> 6.2 pmol/l
- Dogs with ADH have undetectable concentrations.

Advantages
- Single blood sample
- Easy discrimination between ADH and PDH

Disadvantages
- Specific sampling conditions
- Overlap between normal dogs and those with PDH

Summary:
- The urine cortisol: creatinine ratio is a good screening test but it cannot be used as a diagnostic test. It has a good negative predictive value
- The ACTH stimulation test and LDDST are most frequently used to diagnose HAC, but patients must be carefully selected to avoid false positives.
- The LDDST is more sensitive if an adrenal tumour is suspected.
- The high dose dexamethasone test and endogenous ACTH can be used to distinguish PDH from ADH but are rarely used.

Ultrasonographic Assessment

Of The Adrenal Glands In Dogs And Cats

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Introduction

Although ultrasound is less sensitive than computed tomography (CT) in assessing the adrenal glands it is the preferred primary method of assessment due to its accessibility in veterinary practice.

The indications to ultrasound the adrenal glands are largely to support a presumptive diagnosis of hyperadrenocorticism and to further differentiate between adrenal-dependant and pituitary-dependant hyperadrenocorticism. Further indications include investigating peritoneal or dorsal abdominal masses, hypertension and other clinical signs which may be related to phaeochromocytoma and to search for metastasis.

Normal Ultrasonographic Assessment

Patients can either be positioned in dorsal or lateral recumbency. It is essential to use a high frequency transducer to visualise the adrenal glands and a thorough knowledge of the regional vasculature is required to locate the adrenal glands. [1,2]

With the patient in right lateral recumbency, the left adrenal gland can be visualised by positioning the transducer caudal to the last rib and ventral to the lumbar muscles at the level of the left kidney. Utilising this ultrasound window, the aorta should be seen running in a cranio-caudal direction and will be the closer of the two large vessels. The left renal artery can be seen exiting the aorta directed caudally where after it immediately deviates cranially to reach the renal hilus.

The adrenal gland is located between the aorta and the left kidney, cranial to the hook made by the renal artery (Figure 2 B).[1]

With the patient positioned in left lateral recumbency, and with the transducer in a similar position as on the contralateral side, the caudal vena cava and right kidney are located as landmarks to visualise the right adrenal gland. The right adrenal gland abuts the caudal vena cava. The right adrenal gland is more challenging to find than the left gland.[1,2]

A similar approach is used when locating the adrenal glands in feline patients, however the glands are consistently more cranially located along the main abdominal vessels, lying just cranial to the cranial pole of each respective (left and right) kidney. [2]

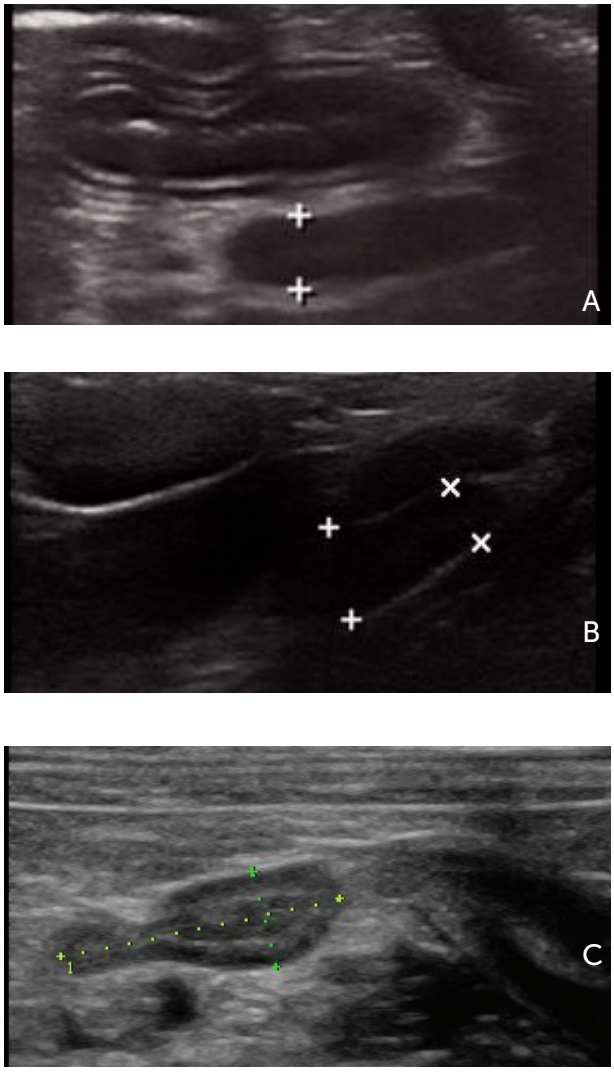

Figure 1: Normal ultrasound appearance of the left (A) and right (B) canine adrenal gland of a medium sized dog. They are slender elongated homogenously hypoechoic structures. (C) is the right adrenal gland from a Yorkshire terrier showing clear cortico-medullary distinction sometimes seen in normal adrenal glands.

The left adrenal gland is peanut shaped in smaller dogs and elongated and slender in medium to large breed dogs. (Figure 1 A and B) The right adrenal gland has been reported to have an "arrow" shape or L-shaped. The glands are typically uniformly hypoechoic how-

ever an outer hypoechoic rim and hyperechoic inner zone may be differentiated with progressive imaging techniques and equipment. This distinction represents the outer cortex and inner medulla.[2] (Figure 1 C)

Feline adrenal glands are typically more oval bilaterally and homogeneously hypoechoic. It is rare to see the cortico-medullary distinction in a cat. Mineralisation of the adrenal glands is common in cats, occurring in up to 50% of the population.[2] Such glands are hyperechoic with a distal acoustic shadow. This change does not however affect the size of the gland. Contrary, mineralisation in canine adrenal glands has a high probability of representing malignant change although rarely, it may be due to dystrophic change. [1,2,3] (Figure 3)

The cut-off for maximum adrenal gland size in the dog has been commonly referenced as 0.74mm[1,2,4] for either the cranial or caudal pole in either a sagittal or transverse plane regardless of body weight of the patient. However a more recent study (n=45), it was found that the size of the adrenal gland in patients without clinical evidence of hyperadrenocorticism varied with three weight categories[4]. The guidelines from this study are as follows:

Maximum thickness of the caudal pole of the adrenal gland in sagittal plane:

- Dogs ≤10 kg: ≤0.54cm
- Dogs 10-30kg: ≤0.68cm
- Dogs ≥ 30kg: ≤0.80cm

However the authors do acknowledge limitations to the study, such as the low study numbers and the need to further investigate at risk populations of dogs in order to fine-tune these cut-off values.[4] It has been found that the caudal pole thickness of either adrenal gland in a sagittal plane was the best dimension for evaluating adrenal gland size due to low variability, ease and reliability in measurement.[4]

Ultrasound is however, not a flawless technique and up to 25% of dogs with pituitary – dependant hyperadrenocorticism can have normal adrenal gland size on ultrasound and some healthy dogs will have adrenal glands larger than the recommended cut-off values.[1-4] Therefore, the ultrasonographic findings should be interpreted in light of the clinical signs as well as the clinic-pathological test results.

In cats, the normal adrenal glands are 10-11mm in cranio-caudal length and up to 4.3 ±0.3mm in diameter. [2]

Pathology Of The Adrenal Glands

Hyperplasia

Patients with PDH generally have bilaterally symmetrically adrenomegaly with a plump rounded appearance. This is attributable to the cortical hyperplasia secondary to pituitary disease. However in some patients with PDH there may be asymmetrical enlargement due to nodular hyperplasia. In these cases it may be difficult to distinguish the enlarged hyperplastic gland from an adrenocortical adenoma. [1,2] (Figure 2)

Another aetiopathogenesis for bilateral adrenomegaly is trilostane therapy.[2,3] This is due to cortical hypertrophy secondary to reduced cortisol production and the diminished negative feedback mechanism. Following trilostane treatment, the glands can also become heterogenous in nature or have an enhanced cortico-medullary distinction. It is therefore imperative to perform ultrasonographic assessment of the adrenal glands prior to initiating trilostane treatment.

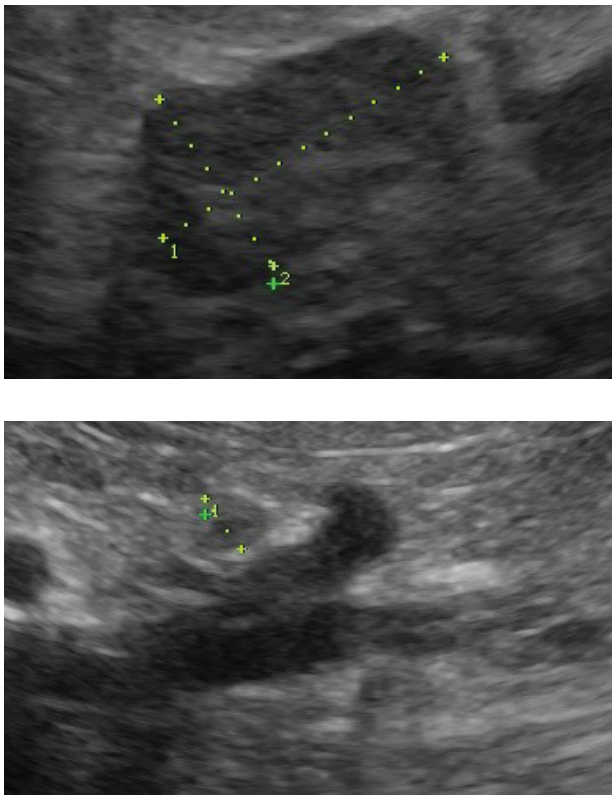

Figure 2: An enlarged right (A) and small left (B) adrenal gland in a dog with confirmed adrenal – dependant hyperadrenocorticism. The right adrenal gland measured 1.6 x 1.0cm and had an irregular margin and mildly heterogeneously hyperechoic parenchyma. The left adrenal gland was difficult to visualise due to atrophy from a functional tumour on the right and measured 0.34cm in width (between the cursors).

Neoplasia

Primary adrenal tumours are generally unilateral but bilateral tumours have been reported. In patients with clinical signs of hyperadrenocorticism and the finding of an adrenal gland nodule on ultrasound can prove a conundrum. This finding may be due to an adenoma, an adenocarcinoma or a hyperplastic nodule and none of these changes have specific ultrasonographic changes.[1-3] The following guidelines apply in such cases:

- Masses ≥ 2.0cm and/or showing mineralisation are considered less likely to represent hyperplastic

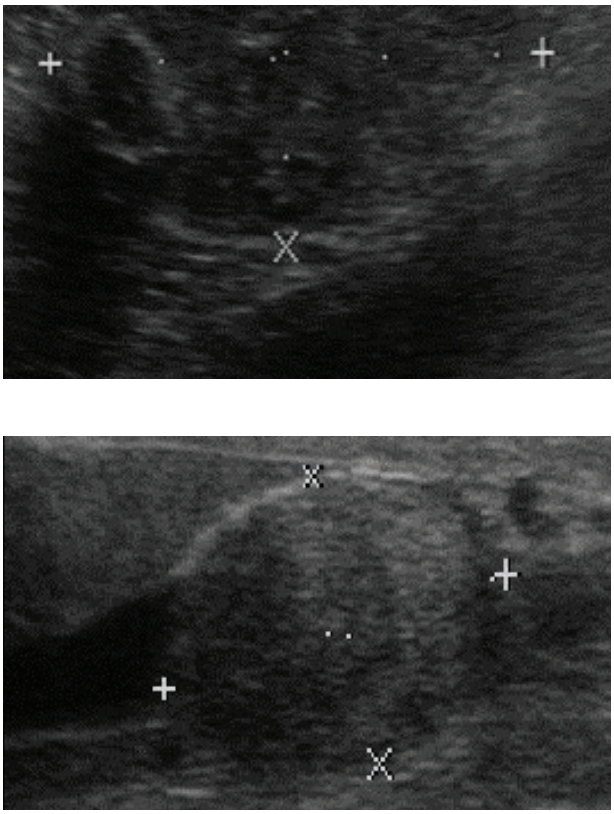

Figure 3: Markedly enlarged left adrenal gland (A) measuring 4.4 x 2.8cm in dimension and exhibiting heterogeneity and multifocal areas of mineralisation. There was an intraluminal echo within the caudal vena cava consistent with vascular invasion (B). This patient also had hyperechoic nodules in the liver which on fine needle aspirate were consistent with metastatic lesions.

change with a benign or malignant lesion more likely.

- Masses ≥ 4.0cm are more likely malignant than benign.

Besides a diagnoses of adrenocortical tumours, other tumours occurring in the adrenal glands include myelolipomas, phaeochromocytomas and metastatic tumours. Benign lesions such as cysts, granulomas and haematomas can also mimic neoplastic change in the adrenal glands.[1,2]

Myelolipomas are benign, endocrinologically inactive tumours. Their fatty component results in them been hyperechoic on ultrasound. [2]

Phaeochromocytomas are rare catecholamine secreting tumours. Patients present with vague clinical signs either due to the secretion of catecholamines or due to the space occupying lesion in the retroperitoneal space. These tumours are incredibly rare in feline patients. [1,2,5]

Several tumours metastasise to the adrenal glands; mammary, prostatic, gastric and pancreatic carcinomas, squamous cell carcinomas, transitional cell carcinomas, malignant histiocytosis, melanomas and haemangiosarcomas.[1] Thus if a patient is suspected of having one of these tumours, a full metastasis search should be performed with careful interrogation of the adrenal glands.

Vascular invasion

Adrenalectomy is the treatment of choice for adrenal tumours. Regional vascular invasion or tumour thrombus is reported to be as high as 82% for phaeochromocytomas and between 11% to 41% for adrenocortical tumours.[3] Vascular invasion has been reported to occur via the phrenicoabdominal vein with echogenic material reported in the phrenicoabdominal and renal veins and the caudal vena cava as a result of extension.[1-3]

Although vascular invasion is more common with tumours affecting the right adrenal gland, aggressive tumours of both glands can invade the caudal vena cava (Figure 3). Tumour thrombus has been associated with a shorter survival time. A negative finding for local vascular invasion on ultrasound is however not sufficient to exclude the possibility and it is advocated that if surgical treatment is intended, a computed tomography study is performed for optimal surgical planning.

Adrenal gland atrophy

If a patient is suspected of having hypoadrenocorticism, then a finding of small adrenal glands or the inability to find the adrenal glands supports this diagnosis. A cut-off value has been documented as ≤3.0mm thickness for the left adrenal gland and ≤3.4mm for the right adrenal gland. However, once again, ultrasound alone cannot be used to make a diagnosis of adrenal gland atrophy.[1] Other causes for non-visualisation of the adrenal glands include incorrect transducer selection/ultrasound technique, poor image quality due to gas in the GIT or patient panting and exogenous steroid administration.[2]

Conclusion

Ultrasound is the preferred first choice modality for adrenal gland assessment in patients with suspected pathology. However, there is a certain degree of overlap in the ultrasonographic appearance of healthy and diseased glands as well as non-specific pathological changes making a definitive diagnosis bases on ultrasonographic findings alone impossible.

It is therefore imperative to correlate ultrasonographic findings with clinical signs and any clinic-pathological test results in order to make a definitive diagnosis. In many cases of adrenal tumours, a definitive diagnosis will only be made at necropsy.

If a nodule or mass is found, following the necessary function tests, it advised to do follow up ultrasound studies every 3 – 6 months for monitoring purposes.

References available on www.vet360.vetlink.co.za

Treatment Options for Pituitary Dependent Hyperadrenocorticism

By Dr L L van der Merwe , BVSC (MMedVet(Med)
And by dr Marlies Bohm, BVSc DSAM MMedVet(Med) DipECVIM-CA

PDH is the most common (82%) form of canine hyperadrenocorticism. Any treatment whether surgical or medical will require life-long medication and some hormone monitoring

A. Surgical resection of the pituitary tumour (trans-sphenoidal hypophysectomy)

Surgery is the treatment of choice for humans. Bjorn Meij of Utrecht pioneered the surgery (trans-sphenoidal hypophysectomy) and has operated on > 200 dogs and cats. With surgery the cause of the problem is addressed whereas with medical management control is aimed at limiting the effects of the pituitary tumour.

If your patient is starting to show neurological signs and the CT/MRI indicate that the tumour is still operable then surgery is really your best choice. If your patient is relatively young you could also consider surgery – because if he is expected to survive for many years assuming his Cushing's is controlled then it means there are many years for the pituitary tumour to grow and ultimatly causes neurological disease.

There are some pituitary tumours that are too large to be removed. The bigger the tumour the greater the risk associated with surgery and the lower the chance that the tumour can be completely removed.

Dr Meij is prepared to fly out from Utrecht and perform the surgery at Onderstepoort. The post op period is tricky and requires constant supervision in ICU. You will need to cover the costs of Dr Meij's flights, his professional fee for the surgery and Onderstepoort's fee. The last case cost the owners approx. R50 000. (Feb 2014). Contact Dr Marlies Bohm (marlies@wol.co.za) if you are considering this option - she organised this for a patient recently.

Prognosis:

92% of a group of 150 dogs survived surgery and the immediate post-operative period and the survival rates are expected to be even higher in resent cases, as the technique was refined. In 9/150 (6%) dogs the surgeon wasn't able to remove all the tumour.

- In 25% of dogs signs of Cushing's recurred 6 weeks – 56 months (median 18 months) post operatively.
- 75% of patients stayed free of signs of Cushing's and died of unrelated diseases. These dogs lived for an average of 28 months (range 2-87 months) after surgery. This doesn't sound terribly long, but Cushing's typically affects middle aged to elderly dogs that have a limited life expectancy in any case.

Post-operative treatment:

All dogs will need thyroid hormone supplements and hydrocortisone post operatively. The hydrocortisone dose is slowly weaned down to a fairly standard lowest effective dose.

- The thyroid hormone dose needs to be adjusted to the individual dog.
- The hydrocortisone dose is increased during times of stress (illness, travel, kennelling, hunting).
- Dogs may need DDAVP (a synthetic form of antidiuretic hormone, vasopressin) transiently in the immediate post-operative period.

Based on 150 operated dogs: 47% of dogs can stop taking DDAVP within 2 weeks of surgery and an additional 31% could stop eventually. The remaining 22% needed it for life.

B. Treatment with mitotane (Lysodren = o'pDDD)

Mitotane, the first effective treatment for Cushing's, destroys the cortisol producing cells in the adrenal gland. Lysodren is not licensed for use in SA and a section 21 application is required.

Induction :

Lysodren dose - 50mg/kg oid

- Start induction on a Thursday / Friday so that the chances of drama on a weekend are diminished as the process generally takes 5 – 10 days.
- The pills are given in the morning after breakfast has been eaten.
- If the dog fails to wolf down his breakfast as nor-

mal - then withhold the medication for that day and perform an ACTH stim test (on the same day if possible). Do not administer any further lysodren until you have your results.
- The target of induction is a post stim cortisol of 25 – 125nmol/L. Unless the dog is showing clinical signs of addisons disease - don't panic - you have stopped the treatment and the decrease will flatten out. If the cortisol is <25nmol/L you may supplement with 1mg/kg prednisilone for a few days. I generally wait 1 week after this sample and then start the weekly maintenance regimen. If the cortisol was <25nmol/L you may want to repeat the ACTH stim to make sure it is in the target range before starting the maintenance therapy - this may take a few weeks in some cases.

Maintenance: Lysodren 50mg/kg, 1 – 2 x/week

- Once on maintenance therapy repeat the ACTH stimulation test after 4-8 weeks to see where the maintenance dose is keeping the cortisol. The target once again is 25 – 125nmol/L. I personally find this a bit low - especially if sampled just prior to next dose (trough level) and am happier with a level between 50 – 150nmol/L. (vdM)
- Note timing of sampling is important: peak effect (i.e. lowest cortisol) a day or so after dosing, lowest effect (i.e. highest cortisol) just before next dose. Make a note of this for proper comparison of cortisol levels
- If your patient is ill /otherwise stressed (e.g. kennelled, going hunting) you may need to supplement low doses of cortisol (prednisone) on those days.

Complications:

- Approximately 25% of dogs show one or more adverse effects during induction and approximately 33% develop a sign of overdose at some time during maintenance treatment
- Overdosing: despite all instructions and counselling, 6-10% of dogs receive excess mitotane and become transiently or permanently Addisonian at some stage in their treatment
- Gastric irritation: vomiting / diarrhoea may be caused by problems other than the treatment. Giving the drug with food reduces the likelihood this side effect.
- Lack of response to treatment: a few dogs have needed > 60 d of daily treatment before their cortisol production decreased. These may do better on trilostane.
- Sudden expansion of a pituitary mass: Sometimes the pituitary tumour can enlarge and start causing neurological deficits. In some dogs this happens during the induction phase.
- Liver toxicity: has been reported in some very rare cases.

Response to treatment:

- The pu/pd and polyphagia usually improves dramatically during the induction phase and normalises in most, not all cases.
- Weight loss will take some months obviously.
- It will take some months for the serum chemistry values to normalise.
- The skin may take up to 6 month to recover. It may actually get worse for 1-2 months before starting to improve.

Advantages:

- This is the only medical treatment option if your dog has calcinosis cutis (calcium deposits in the skin) or myotonia (muscle stiffness).
- You only need to medicate 1-2 x a week
- It is more cost effective than trilostane

C. Treatment with trilostane

Trilostane inhibits conversion of pregnelone to progesterone, a precursor for cortisol and aldosterone – thus both hormone levels are decreased, aldosterone to a lesser degree though. It is licensed in the UK, Europe and USA as a drug called Vetoryl®, which is not available in SA. Several compounding pharmacies produce a trilostane capsules in a variety of sizes. Around 90% of dogs with pituitary dependent Cushing's can be well controlled with this drug.

Pharmacokinetics

It is important to understand some of the pharmacokinetics of this compound as it affects amounts dosed, treatment frequency and interpretation of tests monitoring treatment efficacy.

- Trilostane has low water solubility and an inconsistent absorption which is affected by the formulation. If your patient isn't responding to trilostane or needs very high doses it MAY be because the drug is not being absorbed properly from the compounded version. If this is the case you could switch to imported Vetoryl and do a Section 21 application.
- Absorption increases if dosed with meals.
- The duration of effect varies considerably between patients. Peak plasma levels are reached after 1 .5 - 2 hours and back to baseline at 10 – 18 hours. The drugs is generally active for about 13 hours.
- Results of the ACTH stimulation test used for monitoring response to therapy varies depending on time after dosing of the (post pill).
    - The peak drug levels will result in peak effect and maximum cortisol decrease at about 2 hours post pilling.
    - Cortisol levels were higher when the ACTH stimulation test is performed 4 hours versus at 2 hours post pilling. A current standard is to collect samples 4-6 hours post pilling.
- Due to a loss of negative feedback during trilostane treatment there is an increase in ACTH secretion, which in turn leads to increase size of adrenal glands in treated dogs

- Adverse effects are self-limiting mild GI signs and rarely Addisonian type signs , requiring drug withdrawal and, possibly treatment .The increased endogenous ACTH secretion, especially at the higher starting doses initially recommended, caused adrenal necrosis. This side effect is much reduced with the lower effective doses utilised these days.

Treatment and Monitoring:

Initially the recommendations were 2-5mg/kg/day. This resulted in side effects and currently lower doses are achieving good results without the side effects.

- 1.5mg/kg *oid* or divided *bid* in the morning with food. In larger dogs (>20kg) give slightly less than 1 mg/kg.
- An early 2-week check-up is to make sure the post stim cortisol level is not dropping too low. No dosage increases are made at this time.
- *BID* treatment has been shown to require a lower overall drug dose. Studies show improved survival in dogs treated *bid vs* those treated *oid*. Median survival of 900d for *bid vs* 662 d for oid treatment. Which makes sense if, at best, the drug is only in the system for 13 hrs .
- In dogs where clinical signs are still present and suppression is in the target range on *oid* treatment, *bid* medication is required as the effect of the drug is too short.
- In dogs with concurrent diabetes mellitus - trilostane must be used bid from the beginning to reduce insulin resistance.
- Repeat ACTH stimulation test at 4-6 weeks post initiation of treatment to evaluate the effect of the drug on cortisol secretion. Dosage adjustments can now be made if required.
- Once the target post ACTH stimulation cortisol range of 50 – 150nmol/L, collected at 4-6 hours post pilling, is reached, monitoring can be done every 4-6 months.
- If the post stim cortisol value is suppressed to <50 nmol/L then the medication is stopped for a week and restarted without performing and ACTH stim test.
- If the dog was showing any signs of inappetance, tremors and lethargy concurrently with a low post stim cortisol value then an ACTH stim test is performed prior to re-initiating treatment and treatment is only started again once cortisol levels rise into the target range. For some reason - even though the drug is just an enzyme inhibitor and has a short half-life – its effects can be quite lasting in some animals.

References available on www.vet360.vetlink.co.za

Hypertension and Proteinuria in Canine Cushings Syndrome

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Hypertension and proteinuria are 2 complications of cushings disease that should be tested for an managed. Up to 85% of dogs may have hypertension and 44 – 46% may have proteinuria and these conditions may persist despite successful management of the cushings disease. Because both proteinuria and hypertension are major factors in the development and progression of chronic kidney disease these conditions need to be managed in addition to the cushings.

Angiotensin converting enzyme inhibitors (ACEi) re used to decrease intra-glomerular hypertension as well as reduce mesangial proliferation and glomerular remodelling. When managing proteinuria, note that ACE Inhibitors such as enalapril and benazepril have special warnings for use in patients, they can cause an (initially reversible) azotaemia. You should have a baseline urea and creatinine. Recheck levels 2 weeks after initiating treatment in any patient , even a non-proteinuric patient. Re-check biannually as well. An initial dosages of 0.5 mg/kg q24h (for benazepril) and 0.5 mg/kg q12h (for enalapril) are appropriate; the dosage of both drugs can be increased if required, by giving q12h for benazepril and up to 1.0 mg/kg q12h for enalapril. These drugs can also be used as second- or third-line agents for controlling hypertension.

Amlodipine (0.2 – 0.4 mg/kg q24-12h) is the best agent for primary control of systemic blood pressure (SBP) >160 mmHg; for 140-160 mmHg, rather use an ACE inhibitor or angiotensin receptor blocker (ARB).

Never start a patient on anti-hypertensive agents on the basis of a single measurement, measurements in a very stressed patient or for SBP <140 mmHg. Rather re-evaluate in 5 – 14 days (sooner, for more hypertensive patients).

For example, in an asymptomatic cat with an initial SBP of 220mmHg, I would recheck in 3 – 5 days, ensuring a calm environment. In a symptomatic patient (cat with retinal detachment, gallop rhythm and a proteinuria, with a SBP of 190/130 mmHg), immediate antihypertensive therapy and hospitalisation is more appropriate.

When amlodipine is used, recheck the patient every 5 – 14 days, depending on the severity of the hypertension, and the UPC every 14 – 30 days, to chart response to therapy. Your target should be a UPC <0.2 and SBP 120-140mmHg with diastolic blood pressure (DBP) of 70 – 90 mmHg, no lower. Diastolic hypotension is as damaging to organs as hypertension.

CPD Questions

AC/1429/15

Articles to read for answering the questions

1. Incidentalomas. Dr Frank Kettner
2. Ultrasound of the Adrenal Gland. Dr Nicky Cassell
3. Diagnosing Hyperadrenocorticism. Dr Sandy May
4. Treatment and Monitoring of treatment for hyperadrenocoreticism. Dr L van der Merwe & Dr Marlies Bohm
5. Proteinuria and Hypertension in HAC. Dr A Zambelli

1. Which one of the hormones listed below is NOT produced by the adrenal gland.
   a. Cortisol or one of the precursors
   b. Aldosterone
   c. Sex hormones
   d. Growth Hormone
   e. Adrenalin

2. Which one of the following statements is NOT one of the guidelines used to decide to surgical remove an adrenal masses?
   a. Masses that are larger than 3cm. .
   b. Masses which show ultrasonographic signs of malignancy.
   c. Masses which are functional.
   d. Masses which show invasion of the surrounding blood vessels.
   e. Masses which show metastases to the liver.

3. Which one of the following statements regarding ultrasonographic evaluation of the adrenal glands is INCORRECT?
   a. The glands are typically uniformly hypoechoic. An outer hypoechoic rim and hyperechoic inner zone may be differentiated.
   b. The cut-off for maximum adrenal gland size in the dog is 0.74mm for either the cranial or caudal pole and is highly sensitive and specific for a diagnosis of HAC.
   c. Up to 25% of dogs with PDHcan have normal adrenal gland size on ultrasound and some healthy dogs will have adrenal glands larger than the recommended cutoff values.
   d. In some patients with PDH there may be asymmetrical enlargement due to nodular hyperplasia and it may be difficult to distinguish from an adrenocortical adenoma.
   e. Masses ≥ 2.0cm and/or showing mineralisation are considered likely to represent a malignant lesion.

4. Which one of the following statements regarding the ACTH stimulation test in dogs is INCORRECT?
   a. The baseline cortisol concentration is irrelevant.
   b. A post stimulation concentration of >600nmol/l is considered highly specific and diagnostic of hyperadrenocorticism in the dog.
   c. A big advantage of this test is in distinguishing between iatrogenic and spontaneous cushings disease.
   d. It is the only test used for monitoring response to treatment.
   e. It does not distinguish between PDH and ADH.

5. Which one of the following statements regarding the Low dose Dexamethasone suppression test (LDDST) is INCORRECT?
   a. The LDDST is a test used to differentiate ADH from PDH .
   b. The LDDST is more sensitive than the ACTH stim test overall.
   c. The LDDST is more sensitive than the ACTH stim test diagnosing PDH.
   d. The LDDST is more sensitive than the ACTH stim test for the diagnosis of functional adrenal neoplasia.
   e. The LDDST is a screening test for hyperadrenocorticism.

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6. Which one of the following statements regarding the medical management of HAC is TRUE
   a. Trilostane therapy is 100% safe.
   b. Trilostane needs to be administered *bid*.
   c. Lysodren causes long-term irreversible adrenal gland suppression.
   d. Both medications have resulted in addisons and death in patients as a side effect.
   e. Induction of cortisol suppression with lysodrenal always occurs within 10-14 days days.

7. Which one of the following statements is TRUE
   a. The target range for successful therapy using trilostane is a basal cortisol of <50nmol/L.
   b. The target range for successful therapy using trilostane is a basal cortisol of >50nmol/L.
   c. The target range for successful therapy using trilostane is a basal cortisol of 50nmol/L – 150nmol/L.
   d. The target range for successful therapy using trilostane is a post stim cortisol <50nmol/L.
   e. The target range for successful therapy using trilostane is a post stim cortisol of 50 -150nmol/L.

8. Which one of the following statements is TRUE?
   a. There is no effect of timing on test results when performing the ACTH stim test to monitor treatment efficacy in HAC.
   b. The ACTH stim test ,when monitoring efficacy of trilostane should be performed just prior to the next dose (trough effect)
   c. The ACTH stim test, when monitoring efficacy of trilostane should be performed just 4 hours after the pill is given.
   d. The ACTH stim tes,t when monitoring efficacy of Lysodren should be performed just prior to the next dose (trough effect)
   e. The ACTH stim test, when monitoring efficacy of Lysodren should be performed 4 hours after the pill is given.

9. Which one of the following statements is INCORRECT?
   a. Neurological signs may develop due to effects of uncontrolled cortisol production, thrombocytosis and hypercoagulability
   b. Neurological signs may develop due to effects of pituitary mass which keeps on growing
   c. Proteinuria may persist despite treatment
   d. Hypertension may persist despite treatment
   e. Clinical signs of overdose of medications occurs in about 10% of patients

10. A 10 year old intact fox terrier male presented with a history of waxing and waning inappetance . The abdomen is moderately distended. The dog is less active than normal and the owners complain it is breathing faster than normal. It is also PU/PD according to them. A serum chemistry profile shows a moderately elevated ALT, mildly elevated ALT and slight lipaemia. Which one of the tests listed below is NOT initially indicated for the patient described above?.
    a. Blood smear
    b. Urinalysis
    c. ACTH stim test
    d. Abdominal ultrasound
    e. Thoracic radiographs

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Fipronil Toxicosis in Rabbits

By Laura A. Stern, DVM, ASPCA
Animal Poison Control Center staff
ASPCA APCC

Exposure to these veterinary products can cause life-threatening signs.

Fipronil is a phenylpyrazole insecticide used to control a variety of insects such as ants, beetles, cockroaches, fleas, ticks and termites. It comes in a variety of formulations: topical, spray, dust and bait. This article focuses on the topical spot-on product labeled for use in dogs and cats.

When this product initially debuted, published, extralabel dosing recommendations for fipronil were used successfully in rabbits,[1,2] but subsequently, extralabel administration in rabbits has become contraindicated due to toxicity concerns.[2,3]

Pharmacokinetics and metabolism

Dermal absorption of topically applied fipronil in rabbits is low at 0.07%. Oral absorption is higher at 30% to 50% of the ingested dose and is possible if the rabbit licks the product off after topical application.[4] Fipronil is detected on the hair and superficial skin layers but not in the dermis or hypodermis.[5] It accumulates in the sebaceous gland and then is released by the follicular ducts.

Mechanism of action

Fipronil blocks GABA receptors in the central nervous system, which leads to the prevention of chloride ion uptake and results in excessive central nervous system stimulation and death. Fipronil exhibits a greater affinity for binding insect GABA receptors than for binding mammalian GABA receptors, resulting in a wide margin of safety for most mammals while still causing death to insects.[4]

Toxicity

The no observed adverse effect level (NOAEL) for rabbits is 5 mg/kg/day when applied topically.[4] Topically applied fipronil is classified as moderately toxic for rabbits.[6] The dermal LD50 for rabbits is 354 mg/kg.[6]

In one study, 10 mg/kg/day of fipronil applied topically to rabbits for 21 days caused decreases in mean body weight, weight gain and food consumption.[4] Other studies have shown that dermal dosing in rabbits causes hypersalivation, tremors, hyperactivity, diarrhoea, emaciation and death.[6] Delays in the appearance of signs were noted in these studies.

Seizures were not seen until three to nine days after exposure, and death often occurred 11 to 14 days after exposure.[6] Young rabbits have been reported to be more sensitive to the effects of fipronil than older rabbits are.[3,7]

ASPCA Animal Poison Control Center data

A review of the ASPCA Animal Poison Control Center's toxicology database from 2003 to 2014 yielded 77 fipronil toxicosis cases involving rabbits.[8] These cases involved exposure to a single agent (fipronil-containing spot-on products used extralabel, inappropriately or erroneously) and were assessed as medium- or high-suspect cases based on the history of exposure and clinical signs. Of the 77 rabbits, follow-up was not available for 49 (64%), treatment was still continuing in four (5%), a full recovery was noted in three (4%), and death or euthanasia was observed in 21 (27%).[8]

The most commonly reported clinical signs observed in this review were seizures, anorexia, lethargy, hypothermia, tremors, adipsia, ileus, agitation and hypersalivation (Table 1). The gastrointestinal signs and depression often, but not always, preceded the tremors and seizures.[8]

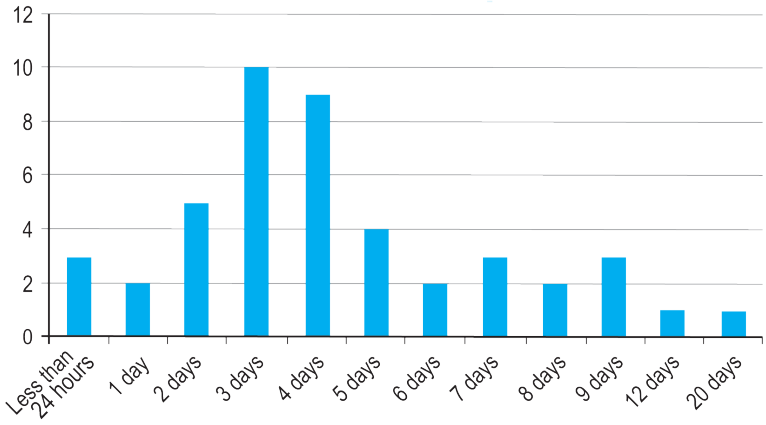
The onset of seizures was often markedly delayed from the exposure, starting as soon as a couple hours after exposure to as long as 20 days later (fig 1). In one case, mild seizures lasted several weeks.[8]

Table 1: Commonly reported clinical signs in rabbits exposed to fipronil*

Clinical Signs	Number of rabbits (n=77) affected
Seizures	45 (58%)
Anorexia	44 (57%)
Lethargy	36 (47%)
Hypothermia	7 (9%)
Tremors	5 (6%)
Adipsia	5 (6%)
Lieus	2 (3%)
Agitation	2 (3%)
Hypersalivation	2 (3%)

**Source AnTox Database Urbana, Illnois: ASPCA Animal Poison Control Center, 2003-2014*

Fig 1: Graph representing seizure onset and frequency in affected rabbits



Monitoring

Monitor the patient's food and water intake, as anorexia and adipsia are common. Also monitor the patient for changes in body temperature, tremors and seizure activity.

Treatment

Bathing the rabbit with liquid dishwashing detergent within 48 hours of the exposure will easily remove the fipronil.[9] After 48 hours, bathing is probably minimally effective. Special care should be taken to keep the rabbit warm during and after bathing until its fur is fully dry.

Benzodiazepines, such as diazepam (1 to 3 mg/kg intramuscularly or intravenously) or midazolam (1 to 2 mg/kg intramuscularly or intravenously), can be given to treat seizure-like activity.[10] For patients with seizures lasting longer than 48 hours, levetiracetam therapy can be initiated (20 mg/kg orally t.i.d., potentially for a few weeks).[11]

Maintenance fluids (100 to 120 ml/kg/day) should be administered to maintain the patient's hydration.[11]

Nutritional support, such as Critical Care (Oxbow Animal Health; 10 to 15 ml/kg orally *b.i.d.* to *t.i.d.*), may be indicated in anorectic rabbits.[11] Often, inappetant rabbits may consume this voluntarily, but force feeding with a syringe may be indicated when continued feed refusal is present. If the rabbit is hypothermic, provide an external heat source, such as a heating pad or warming blanket.

Conclusion

Fipronil has a narrow margin of safety in rabbits. Its administration is contraindicated because of the potential for life-threatening signs and the availability of safer alternative spot-on products for external parasite control.

Seizures, anorexia, adipsia and lethargy are common clinical signs in rabbits exposed to topical fipronil products. The onset of seizures may be greatly delayed in these patients, and at-home monitoring for the development of seizures for several weeks after exposure is warranted. Mild seizures may last for several weeks. The prognosis is guarded for all rabbits exhibiting seizures.[9]

References will be available on www.vet360.vetlink.co.za

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Current Concepts in the Diagnosis and Management of Pyothorax

Stillion JR, Letendre J. A clinical review of the pathophysiology, diagnosis, and treatment of pyothorax in dogs and cats. J Vet Emerg Crit Care 2015;259(1):113-129.

Summurised by Jennifer L. Garcia, DVM, DACVIM
VETERINARY MEDICINE

Why they did it

A lack of consensus exists on the appropriate approach to managing pyothorax in dogs and cats. Despite the many treatment options, the optimal therapeutic approach is unclear, and the prognosis for successful outcome is variable.

What they did

The authors present an overview of the current literature with respect to the pathophysiology, diagnosis and treatment of pyothorax in dogs and cats. They provide instruction for the placement of a small-gauge thoracic drain.

What they found

The origin of pyothorax often remains unknown in dogs and cats, but recent evidence suggests that parapneumonic spread—pleural infection arising as a result of pneumonia or lung abscess—may be the most common route of infection in cats. In dogs on the other hand, the cause of pyothorax may depend more on geographic region (e.g. migration of grass awns in endemic areas). The authors note that infection with feline leukemia virus or feline immunodeficiency virus has not been associated with pyothorax in cats.

Diagnosis:

The introduction of bacteria and inflammatory cells into the pleural space and disruption of Starling's forces and lymphatic drainage that govern pleural fluid drainage lead to fluid accumulation. The authors discuss the need for cytologic analysis as well as aerobic and anaerobic bacterial culture of pleural fluid to provide a definitive diagnosis. Polymicrobial infections are common. Common aerobic organisms isolated from feline and canine pyothorax patients include *Escherichia coli* and *Pasteurella*, *Actinomyces*, *Nocardia*, *Streptococcus, Staphylococcus* and *Corynebacterium* species. Common anaerobic organisms include *Peptostreptococcus anaerobius* and *Fusobacterium, Bacteroides, Prevotella* and *Porphyromonas* species.

Sepsis may occur in up to 40% of cats with pyothorax, and cats may present with bradycardia and hypothermia. In addition to thoracic radiographic and ultrasonographic examinations, thoracic computed tomography may be beneficial in assessing patients with pyothorax and identifying the need for surgical intervention (e.g. foreign body, pulmonary abscess).

Blood and pleural N-terminal pro-B-type natiuretic peptide (NT-proBNP) concentrations may be useful in differentiating cardiac from noncardiac causes of pleural effusion in cats, but the authors noted that overlapping values between these two groups limit the diagnostic utility of this test in cases of pyothorax.

Treatment:

Antimicrobial therapy and thoracic drainage are considered the cornerstone of therapy for patients with pyothorax. Antibiotic selection should be based on culture and sensitivity data when at all possible.

Broad-spectrum therapy to address both aerobic and anaerobic pathogens is recommended. Empiric therapy for dogs may include a potentiated penicillin in combination with a fluoroquinolone; monotherapy with a potentiated penicillin may be sufficient in cats.

Intravenous administration should be considered until the patient is stable and eating; however, the optimal duration of therapy is unknown and is commonly two weeks past radiographic resolution of effusion.

Mechanical removal of infected pleural fluid is also integral to successful management, but there are no data in veterinary medicine as to which method is superior—needle thoracocentesis versus chest tube placement. Similarly, there is controversy regarding the utility of thoracic lavage or use of intrapleural medications. For patients with thoracostomy tubes, there is a lack of consensus on whether intermittent versus continuous drainage is best.

Thoracotomy or video-assisted thorascopic surgery may be considered for patients in which there is an indication for surgery (e.g. foreign body) or for patients that do not appear to be responding to aggressive medical management. The ideal type and timing of surgical interventions, however, is unclear.

Take-home message

No evidence-based recommendations or consensus of opinion has been established on the best way to treat dogs and cats with pyothorax. The prognosis is variable, but good outcomes may be achieved with appropriate care. Patients that present with evidence of sepsis or respiratory decompensation have a worse prognosis.

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Cutaneous Adverse Food Reactions: What's New?

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CAFR is defined as an aberrant immunological response, most often to a food protein.[11] Currently it is believed that Type I (immediate, IgE mediated) hypersensitivity plays an important role in CAFR, although this is still unproven. Type III (delayed, immune complex) and type IV (delayed, cell-mediated) hypersensitivity reactions have also been proposed.[13] Gastrointestinal signs are only seen in 10 – 15 % of dogs with CAFR.[17]

Non-immunologic adverse food reactions (AFRs) include food intolerance and food intoxication which usually result in gastrointestinal signs.[1, 13] Food intolerance may be due to digestive enzyme deficiencies or idiosyncratic reactions to food additives such as colourants and preservatives and food intoxications due to the ingestion of bacterial or fungal toxins.[13]

Pathogenesis

There are currently over 6000 recognised dietary allergens, which include glycoproteins, lipopolysaccharides and carbohydrates. Glycoproteins (molecular weight of 10 – 70kDa, heat and acid stabile) are most commonly associated with an allergic response.[1] The most important allergens implicated in CAFR cases are beef, dairy products and wheat, followed by lamb, chicken, egg, corn, fish, rice and soy.[11] Food additives, such as preservatives are very rarely documented to cause CAFR.[11]

In most cases the offending food source is one that is fed often and can be part of the diet, a treat or flavoured medication.[16] Multiple sensitivities are common – 64% of cases.[6, 13] Clinical signs of antigen sensitization usually follow a long duration of exposure – up to 2 years.[15]

- Dogs that are allergic to beef are usually not sensitive to milk, but they should avoid liver, veal and gelatin.
- Dogs allergic to egg are usually allergic to the proteins in the egg white. These dogs can usually tolerate chicken meat, but not vaccines grown on egg.[1]

The gastrointestinal tract is exposed to a large number of potential allergens every day without manifesting hypersensitivity reactions clinically.

- Ingested proteins are usually digested to amino acids and small peptides which are poorly immunogenic, but proteins that are heat-stabile and poorly digestible retain allergenic features.
- A protective mucous layer containing IgA and carbohydrate particles limit interaction between the allergen and the intestinal microvilli. This layer may be disrupted by diseases of the gastrointestinal tract (e.g. inflammatory conditions or infectious diseases).
- Potential allergens also have to go through an epithelial barrier where after they encounter gut-associated lymphoid tissue (GALT). Oral tolerance to allergens develops with controlled exposure of the GALT to food allergens. Where this mechanism is dysfunctional, sensitization may occur.[13]

Prevalence

Approximately 10-15% of all allergic skin conditions are caused by CAFR. It is the third most common allergic skin disorder after flea allergic dermatitis and canine atopic dermatitis.[11]

Breeds

Dog breeds that are predisposed to CAFR include the American Cocker Spaniel, Boxer, Chinese Sharpei, Collie, Dachshund, Dalmatian, English Springer Spaniel, German Shepherd, Golden Retriever, Lhasa Apso, Miniature Schnauzer, Poodle, Pug, Rhodesian Ridgeback, and West Highland White Terrier.[13]

The first clinical signs usually occur before 1 year of age in 48% of CAFR dogs compared to 16% with atopic dermatitis.[11]

Clinical features

Although no age or sex predilections have been documented, many cases occur at an early age (less than 1 year).

- Non-seasonal pruritus is the most important clinical sign. The severity of the pruritus varies16. Pruritus without visible skin lesions often precedes the development of skin lesions in most (47%) affected dogs.[13]
  Most dogs and cats with CAFR have permanent pruritus that is not obviously related to ingestion of the offending diet.[2]
- If there is a seasonal worsening of the pruritus, concurrent atopic dermatitis is possible and should be addressed once the CAFR is controlled[11], or the dog eats a varied diet and is intermittently exposed to the allergen(s).[1]

The distribution of the clinical signs is exactly the same as in canine atopic dermatitis with the face, periocular areas, ears, axillae, inguinal region, ventral abdomen, perianal area and palmar and/or plantar and dorsal interdigital skin most commonly involved.[4]

In a study of 843 atopic dogs minor clinical differences were found between CAFR and CAD cases but were not sufficient to differentiate between CAFR and CAD. The only way to differentiate between them is by doing an elimination diet trial. A partial response suggests both food and environmental triggers.

Otitis externa commonly occurs in dogs with CAFR: occurring in 24% as the only clinical signs and in 80% as part of the clinical picture.11The otitis externa is usually bilateral but can also be unilateral.[13] Secondary infections often complicate the otitis externa. Malassezia pachydermatis is usually the predominant organism early in the course, while Staphylococcus spp. and gram negative organism infections often develop in chronic cases.[13]

In 10-31% of the cases very subtle concurrent gastrointestinal signs e.g. intermittent vomiting, loose stool, chronic diarrhea, flatulence, increased frequency of defecation and borborygmus were identified in dogs with CAFR.[11,13]

CAFR dogs can also present with other dermatological conditions, such as seborrhoea, superficial pyoderma, Malassezia dermatitis, vasculitis, urticaria, erythema multiforme and symmetric lupoid onychodystrophy.

Diagnosis

Before considering CAFR or CAD other causes of pruritic skin disease such as parasitic infestations (e.g. sarcoptic mange, demodicosis, cheyletiellosis, fleas), allergies to parasites (e.g. flea allergic dermatitis), metabolic disorders (e.g. hypothyroidism with deep pyoderma) or in rare cases dermatophytosis need to be ruled out. Bacterial or Malassezia infections should also be identified and treated appropriately since they often worsen pruritus.

- ### The elimination diet trial

  The gold standard to diagnose CAFR is the resolution of clinical signs when an elimination diet is fed exclusively for a minimum of 6 – 8 weeks followed by a relatively immediate recurrence of clinical signs after a challenge with previously fed food items. The diagnosis is confirmed after resolution of signs when the elimination diet is fed again.[11]

  It is usually easiest to perform the challenge with a small amount of previously fed dog food. In dogs with CAFR clinical signs generally manifest within 2 weeks of challenge although, very often already within 2-48 hours. If the dog does not relapse with "dog food" then other previously fed treats should be introduced one by one.[7] A "new" dietary component should be added every 2 weeks.

  Other treatments are often instituted along with the start of the elimination diet trial such as antibiotic or antifungal therapy for concurrent microbial infections, treatment of otitis externa and temporary anti-pruritic medications. It is important to continue with the diet after this treatment has finished in order to determine whether the clinical improvement is sustained or merely as a result of the antimicrobial and other treatment itself.

  Most dogs with CAFR will have at least a partial response to an elimination diet in 6 - 8 weeks. In most cases clinical cutaneous symptoms improve after 4 – 6 weeks on the diet, whereas gastrointestinal signs, where present, usually improve within 2 weeks.[11]

  Maximum improvement may take as long as 10 – 13 weeks. In some patients clinical symptoms will only partially improve. These patients either suffer from a combination of CAFR and CAD, have uncontrolled secondary bacterial or yeast infections, parasitic infestation or had diet indiscretions.[11]

- ### How to improve owner compliance when performing an elimination diet trial

  Lack of owner compliance is generally the biggest reason for the failure of dietary trials. Often, poor compliance is due to inadequate client education by the veterinarian and therefore it is very important that owners are informed comprehensively about CAFR.

  Many owners find it difficult to believe that the pruritus that their dog is experiencing, is food related.

Reasons for this are that dogs often have been eating the same diet for years without any problem, the dog does not show any gastrointestinal problems, the pruritus is not related to feeding time, they are feeding a good quality, premium diet, they have switched between commercial diets and the pruritus persists.[1]

It is a good idea to provide a hand out with all the necessary information as well as the "rules" of the elimination diet trial because it can be challenging for pet owners to carry out a successful elimination diet trial.

Owner compliance is usually better when commercial diets are chosen.

1. Disallow all treats, table scraps, rawhide treats, flavored medications and toothpaste, any medication or food supplements in gelatin capsules (may contain cattle or swine protein),
2. if there are small children in the household to prevent them from spilling food or feeding the dog from the table
3. Prevent access to cat dishes and cat litter boxes
4. Prevent the patient from licking the dishes in the dish washing machine and from eating from other pet's food bowls.[11]
5. If there are other animals in the household, it is better if they all eat the elimination diet and no one receives any treats.
6. The other option is strict separate feeding and to wash all dishes well after feeding times.

"Legal" treats can be prepared for the dog once the protein and carbohydrate sources have been decided on. Tinned food can be dried into rusks in the oven. Sometimes dogs will refuse initially to eat the elimination diet. Usually, if the owner is prepared to persevere, the dog will become hungry after a day or 2 and start to eat the diet.

Warming up the food or adding salt may sometimes help. Some dogs are "vacuum cleaners" and may scoop up anything during their daily walk. The best way to prevent this is by putting on a muzzle for the duration of the walk, but this is not acceptable to all owners.

Owners should return the dog for a follow up appointment four weeks into the diet trial. During this visit, any questions regarding the diet trial can be addressed and the regression of concurrent infections can be accessed. It is good to weigh the dog before and during the diet trial. Although the diets that are designed for elimination diet trials are nutritionally complete, occasionally a rapid gain or loss of weight can be seen.[7]

- **Selecting the appropriate elimination diet**

A proper elimination diet should consist of a novel protein and carbohydrate source, which the affected patient has not eaten previously. One of the most important features of an elimination diet is selecting a diet that the dog will eat willingly for the entire trial period.[1] There are three categories of elimination diets: home cooked novel protein diets, commercial novel protein diets and hydrolysed protein diets. Each of these has its advantages and disadvantages.

Home cooked novel protein diets

Where an owner is willing to prepare a single protein source, single carbohydrate source diet for several weeks, home cooked diets are an option.

- Freshly prepared food is free of food additives.
- Protein sources that are acceptable include rabbit, venison, lamb, goat, squid, ostrich, crocodile, duck and some fish (trout).[1]
- If potatoes and white rice were the previous carbohydrate sources, oatmeal, sweet potatoes, pumpkin or tapioca may be used.[1]
- The protein : carbohydrate ratio should be 1 part meat : 2-3 parts carbohydrate and about 400 gram per 5 kg body weight should be fed per day.[1,11]
- It is important to select a protein and a carbohydrate source that is readily available, especially for the duration of the elimination diet trial.
- Home cooked diets may be unbalanced but since these diets are generally fed for 6 to 8 weeks only, no deficiencies have been reported.[7]
- Home cooked diets should not be used at all in growing dogs for more than four weeks without being nutritionally balanced by an experienced animal nutritionist. Skeletal or organ pathology can result from a poorly balanced diet in these dogs.[4]
- A balanced recipe formulated by an animal nutritionist should be used for owners who choose to home cook long term for the patient.[16]
- Tap water should also be substituted with bottled water.[1]

There is no evidence that raw foods are superior to cooked foods as an elimination diet. When proteins are heated they become less allergenic. In addition raw food diets place the dog and owner at increased risk of exposure to pathogenic *Salmonella spp.*, *E. coli*, and other microorganisms.[9]

Commercial novel protein diets

Commercial novel protein diets contain whole proteins which are not commonly found in dog foods. Truly "novel" protein is becoming more difficult to find as the regular diets available increasingly contain more varied and exotic ingredients.
The choice of diet should be made on the basis of the protein content and should be one which has not routinely been fed to the pet in the past, and ideally not at all.[7] The "novel" proteins used vary in different parts of the world, mainly due to availability and may include venison, lamb, rabbit, duck, various fish, moose, goat, ostrich, wild boar, and emu, among others.

It is very important that there should only be one single protein source whenever possible. Carbohydrate sources are less often described as causes of CAFR, but the ideal diet should include a novel carbohydrate source as well.[13]

Hydrolised protein diets

In these diets, the protein source – usually a common parent protein such as chicken or soy - is broken down into peptides and amino acids by a process called hydrolysis. The proteins are reduced in size below 10kDa, which is too small to initiate an IgE-mediated reaction. The rationale behind the development of these diets assumes that most CAFR reactions in dogs are IgE mediated.[7] If this was the case, then in theory it should not be possible for any dog to be allergic to these diets. Some dogs, although very few, still are.[1] This is most likely because not all CAFR cases are IgE mediated, but whether this is the case is currently unknown.

A number of studies have evaluated the performance of hydrolysed diets in clinical settings and found them to be well tolerated and effective in most cases.[5,12,14]

There is a concern about the response to these diets by dogs known to be hypersensitive to the parent protein. A systematic review of 11 studies that reported on hydrolysed protein diets concluded that they should be avoided when the parent protein is suspected as an allergen, because some dogs still display signs of CAFR when fed the hydrolysed diets derived from known allergens.[12] Hydrolysed diets tend to be more costly and are sometimes less palatable.[4]

Other tests for diagnosing CAFR

Other tests that have been used to diagnose CAFR include intradermal skin tests, serologic tests and gastroscopic and colonoscopic provocation tests. These have been shown to be either of limited diagnostic use or are not routinely available.

Many studies have shown that in vitro (laboratory) serologic tests and intradermal skin tests do not correlate well with results of dietary elimination trials.[3,7,8,10]

Despite these findings, a number of companies offer in vitro testing of blood or serum for IgE antibodies to foods. These tests often report false positive reactions to food ingredients and often identify proteins that the dog is commonly fed and not necessarily proteins that cause hypersensitivity reactions.[1] Serologic tests are sensitive indicators of food-specific IgE antibodies, but are poor predictors of clinical reactivity. Because of the serious discrepancy between test results and clinical response, serologic tests should not be used to diagnose or choose a food for treatment of CAFR.[10]

Tests that are not routinely available include intestinal permeability, gastroscopic provocation and colonoscopic provocation tests.

Management of the CAFR dog

A CAFR dog may be maintained on the diet which was used in the elimination diet trial. Alternatively, and if the offending allergens have been identified, an alternative diet which is "allergen" free for that dog can be utilised. Some dogs with CAFR can develop a hypersensitivity to the new diet after months to years on this diet. In these cases a new diet needs to be found. Young dogs may also progress to develop concurrent hypersensitivities to environmental allergens7.

Conclusions

- CAFR is common in the dog and is mostly indistinguishable from atopic dermatitis based on clinical features alone.
- A 6 – 8 week elimination diet trial utilizing a novel protein or hydrolysed protein diet is necessary for diagnosis.
- Once the offending food or ingredients have been identified, the novel protein or hydrolysed diet may be used as a maintenance diet, or the owner may prefer to experiment with new foods.
- Where the allergens have been identified and can be avoided, the prognosis for dogs with CAFR is excellent.

References will be available on www.vet360.vetlink.co.za

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Nutritional Support for Critically Ill Patients

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Edited: Dr L.L. van der Merwe

The five standardised vital signs which are now considered to make up part of the clinical examination now include **temperature, pulse, respiration, pain assessment as well as nutritional assessment.**

Detailed descriptions of nutritional assessment guidelines are available as part of a nutrition toolkit published on the WSAVA website (www.wsava.org/nutrition) by the WSAVA Global Nutrition Community.

Apart from obtaining a thorough nutrition history, animals should be weighed on a daily basis and additional parameters such as body condition score, muscle condition score, serum proteins and serum electrolytes are assessed depending on the case.[10] The nutritional plan should be evaluated daily and adjusted according to the patient's changing needs. A very useful WSAVA monitoring sheet is available for adaptation / use in the hospital.[10]

Assisted feeding using feeding tubes can improve outcome and decrease duration of hospitalisation in critically ill dogs and cats.

Providing resting energy requirements (RER) will prevent the deleterious effects of malnutrition. It is easy to implement and needs to be considered as part of the therapeutic plan in small animal patients. Yet , despite the benefits of nutritional support of ill animals, this aspect is often ignored or delayed. The veterinarian is focused on the medical or surgical condition alone rather than the patient as a whole.

Rationale for implementing nutrition in a critically ill patient

It is widely accepted and makes clinical sense that feeding patients with critical illnesses will be of benefit to them and aid in their recovery. Documented effects of malnutrition include alterations in energy and substrate metabolism, compromised immune function and delayed wound healing.[3] A 2010 study concluded that energy supply, even when only supplying levels close to resting requirements, was positively associated with recovery.[2]

It has been demonstrated that healthy animals which suffer malnutrition mainly metabolise and lose fat. This is known as "simple starvation".[3] Injured or sick animals will mainly metabolise lean body (muscle) mass if malnourished and this is known as "stressed starvation" and results from an elevation in catecholamines, glucocorticoids and glucagon as well as peripheral insulin resistance.[3] Stressed starvation is the reason why critical care clinicians advocate early enteral nutrition.

The Nutritional Plan

When patients are not consuming adequate calories to supply the body with energy, the clinician will have to make a decision as to whether or not to feed the patient

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Fig 1: A cat with a nasoenteric tube placed. A collar is placed to prevent the patient from dislodging the tube. (Photograph Courtesy Dr van Schoor. Onderstepoort Veterinary Academic Hospital)

by artificial means, when to commence feeding, how much food to supply, how often to feed, the type of food to feed, which route of nutrient supply to use, and how to monitor the nutritional status of the patient. This all forms part of the nutritional plan.

1. ASSESS nutritional status

A subjective assessment is made in order to decide on the nutrition of a critically ill patient.[4] This includes a history of a loss of appetite, weight loss observed by the owner and the presence of vomiting or diarrhoea.[3,4]

Clinical assessment of the body condition, weight of the patient, muscle condition score, quality of the hair coat and mental status can guide the clinician in the nutritional assessment.[3,4,6,10]

In many canine diseases *nil per os* for the first 24 – 72 hours was recommended in the past. However, studies in general hospital populations of critically ill small animals have shown that nutritional supplementation, even in moderation and providing close to RER, was positively associated with discharge from hospital.[2,6]

The exact timing of initiation of early enteral nutrition varies in both human and animal literature.[5,6] In some experimental animal studies, nutrition was initiated within 2 hours. Human studies recommend feeding as early as 6 hours after admission.[5]

A recent veterinary study defined "early" nutrition as nutritional supplementation within 24 hours after admission.[6]

2. TYPE of diet required

Sick animals will mainly be using fat and protein for energy. Carbohydrates will generally be more poorly utilised due to peripheral insulin resistance. Diagnostic testing will identify specific diseases such as renal failure, heart failure, liver failure, pancreatitis, neoplasia and others that may influence the nutritional management plan.[3,4]

3. CALORIC needs

Critically ill patients should start receiving RER as soon as they are haemodynamically stable.[3,4] Previously RER was multiplied by an illness factor to provide higher energy levels.[7] This practice was discontinued, as it was shown that it is not necessary to feed patients more than RER and may in fact be detrimental.[2,3,4]

Should it become evident that a patient requires more nutrition than what is being provided – weight changes, ongoing losses (vomiting and diarrhoea) - the number of calories can be increased by up to 25% at a time.[3]

The resting requirements are calculated using one of the following formulae[7]:

- For animals > 2kg <30kg body weight (BW) – linear formula:
  - [BW x 30] +100 = kcal/24hours
- For all animals <2kg and >30kg – allometric formula
  - 100 x (BW)0.75 = kcal/24 hours

Alternatively, the WSAVA RER chart can be used to determine the RER of the patient based on its body weight.

4. ROUTE of administation

The preferred route of nutrition is always to use the gut, and use parenteral nutrition as a last resort. Enteral nutrition is the safest, simplest, most cost effective and most physiological route.

Appetite stimulants:

Used in cats with partial anorexia. Commonly used drugs include diazepam, cyproheptadine and mirtazapine. Diazepam should be avoided in cats with liver disease because of its potential hepatic side effects.

Hand feeding/Force feeding:

This is of limited use as especially in cats it causes food aversion, and rarely are sufficient calories ingested. If patients eat willingly off a spoon / syringe - then it is an option to consider in recent illness where problem will probably be short-term.

Naso-oesophageal / nasogastric tubes

- These tubes are CONTRA-INDICATED in patients without a gag reflex or which are comatose or vomiting
- Use polyurethane or silicone feeding tubes - stay supple when exposed to gastric acid.
  - The silicone tubes are softer and more flexible - which can cause kinking . They also have a narrower internal diameter. The French unit (F) refers to the outer diameter.
  - Polyurethane Ryles feeding tubes are commonly used.
- Naso-enteric tubes (Fig 1, 2) are usually preferred because no general anaesthesia is required for their placement. The choice between nasoesophageal (NE) – place to 7th intercostal space and

nasogastric (NG) feeding largely depends on the preference of the attending clinician.[11] Previously it was thought that NG feeding tubes were associated with more complications than NE tubes. These potential complications include regurgitation, aspiration pneumonia, gastroesophageal reflux and reflux esophagitis.[11] Arguments for NG tube placement include the fact that gastric decompression can be achieved if the end of the tube is situated within the stomach lumen and that gastric residual volumes can be measured regularly.[11] It was shown in a study that complication rates with naso-enteric tubes are quite low and complication rates between NG and NE tubes are not significantly different.[11]

- Disadvantage:
  - Small tube diameter, thus food has to be very liquid. Volume can limit amount of calories given.
  - The tube may also cause discomfort in the nasal passages.

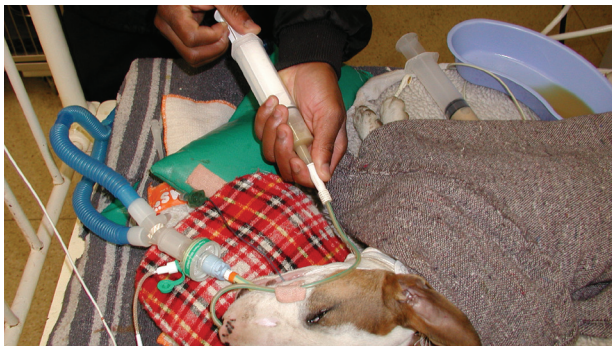

Fig 2: A nasoenteric tube placed in an anaesthetised bull terrier. Placement of a cuffed ET tube and elevation of the patients head and thorax while feeding reduces the risk of aspiration. (Photograph Courtesy Dr van Schoor. Onderstepoort Veterinary Academic Hospital)

Oesophagostomy tubes

In patients where severe facial injuries prohibit the use of naso-enteric tubes or where tube feeding will be required for more than a week, an oesophagostomy tube should be used.[9]

The tubes are easily inserted although GA is required. Advantages are a wider tube diameter as well as placement on the neck - which causes less irritation and interference with normal function. Even if pulled out accidentally before the 7- 10 days required for stomaformation, the incision will close up without complications.

Gastrostomy tubes

These are placed surgically or endoscopically. Once again a relatively large diameter catheter can be placed. The stomach wall must be fixed to the abdominal wall if doing surgical placement as this limits the risk of peritonitis if the tube is accidentally removed before a stoma has developed (<10 days post op).

Fig 3: Oesophagostomy tube placed in a puppy. (Photograph Courtesy Dr van Schoor. Onderstepoort Veterinary Academic Hospital)

5. AMOUNT and method

Daily food volumes are calculated and divided into approximately 6 meals.[7] Food is warmed to body temperature prior to feeding. Feed relatively slowly - over a few minutes to avoid oesophageal spasm and discomfort. Make sure the animal is sitting and not in lateral recumbency when feeding.

The feeding tube is flushed with warm water (5 - 20ml depending on patient size and tube volume) at each meal prior to feeding and immediately after feeding to avoid obstruction of the tube. Maximum tolerable feeding volumes in debilitated dogs and cats can be estimated as 5 – 10 ml/kg body weight initially.[7] Patients that do not tolerate large volumes can be fed very small volumes more often and in certain patients continuous rate infusion (CRI) feeding may be preferred.[7]

Attempts to unblock a blocked tube with warm water flushing followed by suctioning are often not successful.[8] Sodium bicarbonate added to the water may be more successful. Unfortunately it may be necessary to replace a blocked tube.[8]

Metabolic complications resulting from too much food too soon are referred to as "refeeding syndrome".[7,8,9] It causes amongst others, hypophosphataemia, hypokalaemia and hypomagnesaemia.[8,9] Clinical manifestations of refeeding syndrome include muscle weakness, cardiac arrhythmias, respiratory depression, intestinal ileus and haemolysis. Refeeding syndrome can be avoided by initially feeding 25% -50% of RER and gradually increasing the energy supply over a period of a few days until 100% RER is being fed.[7]

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Calorie Needs for an Average Healthy Adult Cat in Ideal Body Condition*

Weight (kg)	Weight (lb)	Kilocalories/ day
1.0	2.2	100
1.5	3.3	130
2.0	4.4	160
2.5	5.5	180
3.0	6.6	210
3.5	7.7	230
4.0	8.8	250
4.5	9.9	270
5.0	11.0	290
5.5	12.1	310
6.0	13.2	330
6.5	14.3	350
7.0	15.4	370

Note: These recommendations are for guidance only.
Cats *are individuals and some may have higher or lower requirements in order to maintain an ideal, trim body condition.*
Dogs *are individuals and some may have higher or lower caloric requirements in order to maintain an ideal, trim body condition.*

**If the cat is overweight, these estimates may be too high and further calorie restriction will be required.*
***If the dog is overweight, these estimates may be too high and further calorie restriction will be required.*

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Calorie Needs for an Average Healthy Adult Dog in Ideal Body Condition**

Weight (kg)	Weight (lb)	Kilocalories/ day	Weight (kg)	Weight (lb)	Kilocalories/ day
2	4.4	140	26	57.2	970
3	6.6	190	27	59.4	1000
4	8.8	240	28	61.6	1020
5	11	280	29	63.8	1050
6	13.2	320	30	66	1080
7	15.4	360	31	68.2	1100
8	17.6	400	32	70.4	1130
9	19.8	440	33	72.6	1160
10	22	470	34	74.8	1180
11	24.2	510	35	77	1210
12	26.4	540	36	79.2	1240
13	28.6	580	37	81.4	1260
14	30.8	610	38	83.6	1290
15	33	640	39	85.8	1310
16	35.2	670	40	88	1340
17	37.4	700	41	90.2	1360
18	39.6	730	42	92.4	1390
19	41.8	760	43	94.6	1410
20	44	790	44	96.8	1440
21	46.2	820	45	99	1460
22	48.4	850	46	101.2	1480
23	50.6	880	47	103.4	1510
24	52.8	910	48	105.6	1530
25	55	940	49	107.8	1560

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Corticosteroid and Nonsteroidal Anti-inflammatory Drug Interactions

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Both corticosteroids and nonsteroidal antiinflammatory drugs (NSAIDs) are known to cause adverse effects in dogs and cats. The theoretical risk for toxicity is increased when these drugs are used concurrently and this practice is generally contraindicated.

However, corticosteroids and NSAID combinations may have additive therapeutic benefits when treating some disease conditions because they sequentially block the arachidonic acid cascade production of prostaglandins (PGs). This effect may be adverse, though, as PGs also benefit and protect the gastrointestinal (GI) tract, haemostasis, and renal function.

Pathophysiology

The mucousa of the GI tract is regularly exposed to a wide range of potentially damaging substances, including those that are ingested (bones, drugs, etc) and endogenous secretions (gastric acid, bile salts).The GI mucousa is not only able to resist damage by these substances for the most part, but it has tremendous reparative capacity when damage does occur.

Systemically, cyclooxygenase (COX)-derived PGs are very important in modulating GI mucousa defense and repair, with COX–1-derived PGs supporting mucousal blood flow, mucus and bicarbonate secretion, and normal platelet aggregation, while COX–2-derived PGs modulate the inflammatory response so that normal healing can occur.

Haemostasis is highly influenced by PGs, with platelet aggregation and vasoconstriction promoted by platelet-derived thromboxane-A2 and inhibition of platelet aggregation and vasodilation promoted by endothelium-derived prostacyclin. Renal PGs help maintain normal blood flow and glomerular filtration rate (GFR).

There are significant interspecies differences in the presence and distribution of COX isoforms. In dogs, both COX isoforms have major roles in normal renal function, with COX-1 expressed at high levels in the collecting ducts and renal vasculature, and basal levels of COX-2 present in the maculae densa, thick ascending limbs, and papillary interstitial cells. COX-2 expression is markedly increased in volume-depleted dogs.[1]

COX-derived PGs are very important in modulating GI mucosa defense and repair, with COX–1-derived PGs supporting mucousal blood flow, mucus and bicarbonate secretion, and normal platelet aggregation, while COX–2-derived PGs modulate the inflammatory response so that normal healing can occur.

Currently, there is no published information regarding the distribution of COX isoforms in the cat, but clinical experience suggests that they are more susceptible to drug-induced nephrotoxicity than dogs.

Gastrointestinal Complications

Corticosteroids

- Corticosteroids are known to induce GI ulceration and haemorrhage in dogs.[2-5] It is thought that ulceration is caused by blockage of PG synthesis at the level of phospholipase A.
- Corticosteroids also inhibit ulcer healing by altering gastric mucus composition, decreasing the rate of mucousal cell turnover, reducing capillary and fibroblast proliferation, and enhancing collagen breakdown.

NSAIDs

- Through PG inhibition, NSAIDs reduce mucus and bicarbonate secretion, increase gastric acid secretion, and reduce mucousal blood flow.

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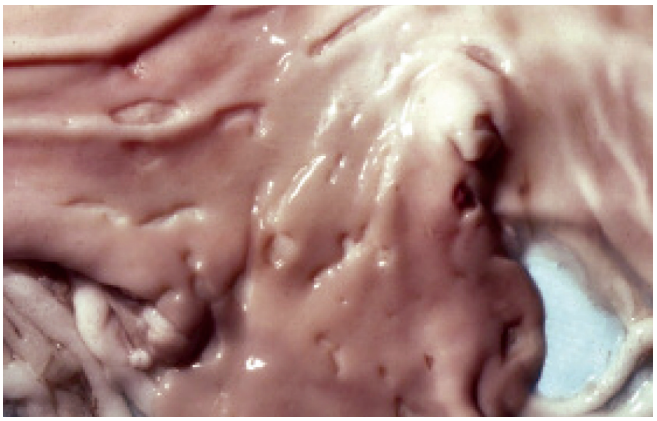

Postmortem stomach specimen from a Great Dane that was administered 2 doses of naproxen shows multiple NSAID-induced mucosal erosions. Courtesy Dr. Michael Schaer

- NSAIDs also trigger an increase in adhesion of leukocytes to the vascular endothelium in the GI microcirculation, which is an early and critical event in the pathogenesis of NSAID-induced mucousal ulceration.[6]
- NSAID-induced GI bleeding is increased by NSAIDs that have COX-1 activity, which reduces platelet aggregation.

Corticosteroids + NSAIDs

- Retrospective studies have associated the concurrent use of corticosteroids and NSAIDs with GI ulceration and perforation in dogs and cats.[7,8] Concurrent administration of an immunosuppressive dose of prednisone and ultralow-dose aspirin in healthy dogs increased the frequency of mild, self-limiting diarrhoea but did not increase the severity of GI lesions compared with prednisone alone or placebo.[5]
- In a study comparing concurrent administration of ketoprofen (a COX-1 selective NSAID) and prednisolone with meloxicam (a COX-2 selective NSAID) and prednisolone, the ketoprofen-treated dogs had significantly more severe GI lesions compared with the meloxicam-treated dogs or untreated controls. All ketoprofen-treated dogs had positive fecal occult blood tests, but only one meloxicam treated dog had a positive test.[9]
- In a comparison of the effects of flunixin and flunixin plus prednisone on the GI tract of dogs, dogs given prednisone and flunixin developed GI lesions more rapidly and lesions were more severe than those in dogs given flunixin alone.[10]

Complications of GI ulceration include intraluminal haemorrhage, ulcer perforation, and stenosis of affected areas. Perforation and septic peritonitis are the most common life-threatening sequelae of GI ulcers in dogs and cats.[7]

Haemostatic & Renal Complications

Little is known about the adverse hemostatic or renal effects of corticosteroid/NSAID combination therapy.

- Any dog or cat that becomes anorectic or vomits while on combination NSAID and corticosteroid therapy should be promptly evaluated by a veterinarian.
- Concurrent therapy with corticosteroid/NSAID combinations should only be done when medically necessary and with close and careful patient monitoring.
- Prophylactic therapy may be warranted in patients that are at risk for GI ulcers and receiving both types of drugs.

- In dogs, the combination of ketoprofen and prednisolone caused significant decreases in GFR and renal plasma flow, hyperalbuminuria, an increased urine albumin:creatinine ratio, and enzymuria with exfoliation of renal tubular epithelial cells.
- Some abnormal enzymuria and exfoliation of renal tubular epithelial cells were seen in dogs receiving meloxicam and prednisolone, but no other indications of renal dysfunction were documented.
- The combination of meloxicam and prednisolone did not cause demonstrable changes in bleeding times, while the ketoprofen/prednisolone combination caused greatly prolonged mucousal and cuticular bleeding times with no effect on measures of secondary hemostasis (prothrombin time, activated partial thromboplastin time, fibrinogen concentration).[9]

Prevention Of Complications

Currently there is insufficient evidence to recommend prophylactic gastroprotective drugs in all dogs or cats receiving NSAIDs and/or corticosteroids, but prophylactic therapy may be warranted in patients that are at risk for GI ulcers and receiving both types of drugs.

- Prophylactic administration of misoprostol (a PG analogue), histamine-2 receptor antagonists (eg, cimetidine, ranitidine, or famotidine) and proton pump inhibitors (eg, omeprazole, pantoprazole) has been evaluated in humans and dogs.
- In a study of healthy dogs, famotidine, pantoprazole, and omeprazole significantly suppressed gastric acid secretion, but only twice daily administration of a suspension of omeprazole met the criteria for therapeutic efficacy for acid-related disease as assessed in human patients.[11]
- Misoprostol has shown efficacy in preventing aspirin-induced gastric mucosal ulceration in dogs.[12] However, in dogs with intervertebral disc disease

treated with dexamethasone, neither omeprazole nor misoprostol was effective in healing or preventing the further development of gastric ulcers.[13]
- In dogs undergoing spinal surgery that received methylprednisolone sodium succinate, neither cimetidine, sucralfate, nor misoprostol reduced postoperative GI bleeding.[3,4]

Treatment of Complications

Treatment of GI toxicity is intensive and mainly symptomatic. Anorexia and/or vomiting are frequently the first indication of GI ulceration and perforation. Any dog or cat that becomes anorectic or vomits while on combination NSAID and corticosteroid therapy should be promptly evaluated by a veterinarian.

- Vomiting & Diarrhoea: Fluid and electrolyte losses from vomiting or diarrhea are managed with commercially available intravenous fluids.
- Haemostasis Complications: Blood transfusions are necessary in patients with haemostatic complications.
- Hypoproteinaemia: Hypoproteinaemia that results from loss of plasma proteins into the ulcerated GI tract can be corrected with intravenous infusions of plasma.
- Renal Failure: The general principles of managing drug-induced renal failure include treatment of life-threatening features, such as shock, respiratory failure, hyperkalaemia, pulmonary oedema, metabolic acidosis, and sepsis. Further administration of nephrotoxic drugs should be avoided and drug dosages should be adjusted as appropriate for the patient's GFR.
- Peritonitis & Bacterial Septicaemia: Broadspectrum antimicrobials are indicated when there is evidence of peritonitis or bacterial septicaemia.
- Pain: Pain must be managed with opioid analgesics.
- GI Perforation: If GI perforation is suspected or diagnosed based on abdominal fluid cytology, abdominal radiography or ultrasound, or endoscopy, prompt surgical exploration and correction are necessary. Open abdominal drainage may be necessary as well.[7] Even with prompt surgical correction, GI perforation has a high mortality rate.
- GI Ulcers: Antiulcer medications may be beneficial and speed healing of GI ulcers.
  - The clinical "efficacy" of antiulcer drugs is typically evaluated by comparing endoscopically detectable mucosal damage and ulcers in treated patients control animals. However, a true ulcer is deep enough to reach or penetrate the muscularis mucosa, which cannot be measured endoscopically.
  - The NSAID-induced endoscopic "ulcers" seen in these studies may not be relevant, as clinically significant GI hemorrhage is rarely seen in humans or dogs with these lesions.[14]
  - Currently, there are limited efficacy data on these drugs in dogs and none of the antiulcer drugs has been evaluated in cats.
  - In humans, proton pump inhibitors are preferred for healing and prevention of GI ulcers in patients taking both traditional NSAIDs and selective coxibs, and who have risk factors associated with more frequent or severe GI complications, including patients with previous ulcers, the elderly, and those receiving concomitant corticosteroids or anticoagulants.[15]
  - Omeprazole, sucralfate, and misoprostol are limited to oral formulations, which are not feasible in actively vomiting patients or if GI perforation is likely.
  - H2-blockers and pantoprazole are available in injectable formulations.

Summary

It is very difficult to predict which corticosteroid/NSAID combinations (drug formulation, dose, dosing frequency, treatment duration) will result in adverse effects in any individual patient. Therefore, concurrent therapy should only be done when medically necessary and with close and careful patient monitoring. Further evaluation in dogs and cats is needed, but proton pump inhibitor drugs, such as omeprazole, appear to be the best choice for pharmacotherapy of GI ulceration because their effects are therapeutic as well as prophylactic. Management of adverse haemostatic and renal effects is mainly supportive.

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Reference: 1. Wernham BGJ, Trumpatori B, Hash J, et al. Dose reduction of meloxicam in dogs with osteoarthritis – associated pain and impaired mobility. J Vet Intern Med 2011; 25:1298-1305. S3 METACAM® oral suspension. Veterinary medicine. Each ml contains 1,5 mg meloxicam. Reg. No. 94/3.1.2.1/15 (Act 101 of 1965). Namibian Reg. No. V11/3.1.2.3/1154 (Act 13 of 2003). For full prescribing information refer to the package insert approved by the Medicines Control Council. Ingelheim Pharmaceuticals (Pty) Ltd, Animal Health Division, 407 Pine Ave, Randburg, 2125. Tel: +27 (011) 348-2400. Email: salesAH@boehringer-ingelheim.com. Cpy. Reg. No. 1966/008618/07. BI Ref. No. V30/2015 (May).

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