

# NKR VETERINARY SPECIALISTS

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## The NKR Newsletter for Referring Clinicians

February 2010

### HEALING AND THE 'HAND OF GOD'

Dubois, the famous 16<sup>th</sup> century French surgeon had a large sign in his operating room: 'The Surgeon dresses the wound, God heals it.' Well, at least this tells us that Dubois was not one of those surgeons who regarded himself as God! More interestingly, it illustrates his awareness that the tying of the final suture is by no means the end of the road to recovery although he seemed to think that everything that could go right or wrong with the healing process was no longer in his hands.

Nowadays, surgeons take a more proactive role in achieving a successful recovery by trying to help the 'God' bit. Besides ensuring that the diagnostic work up and surgical planning have resulted in the correct part of the patient being cut, there have been gigantic advances in the way of sterile technique and theatre management to minimise complications of infection. Then there is immediate postoperative care, including patient homeostasis and pain management. At NKR each patient has a named nurse whose duty it is to monitor, medicate, manage and mollycoddle.



Opportunity for things to go wrong can arise when the patient goes home. The accurate passing on of directions for the care and medication of the patient is of paramount importance. Given that the average person apparently only retains about 30% of the information in a single verbal exchange, our discharge procedure is supported with printed instructions and advice reinforced with prompt reporting to the referring veterinary surgeon. We keep in constant touch with worried owners until the healing process is safely under way. Our Complete Support Consultation takes patient care one step further, by offering our referring vets an opportunity to seamlessly resume care of their patient.

So at NKR we do our level best to take the uncertainty out of healing by consistently applied good practices, self-evaluation, auditing and training. That is not to say that when a miraculous recovery against all odds happily occurs, there are not a few muttered words of thanks for the 'Hand of God' heading upwards!

by Mike Dale MA VetMB MRCVS CertVetAc

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## ENDOCRINE EMERGENCIES - Part 2

### Diabetes – Hyperglycaemia & Hypoglycaemia

by Sebastien Monier DrVetMed, MRCVS

Emergency and Critical Care Clinician

#### Diabetic ketoacidosis

DKA is one of the most commonly encountered endocrine emergencies in small animal practice. This is most often seen in previously undiagnosed diabetics and less commonly occurs in patients that are on inadequate amounts of insulin. In patients receiving insulin, DKA is typically seen in those with a concurrent illness leading to insulin resistance. Common disease processes in cats presenting with DKA include hepatic lipidosis, pancreatitis, chronic renal failure, and bacterial infection as well as administration of exogenous corticosteroids. In dogs, hyperadrenocorticism, pancreatitis, renal disease, urinary tract infections, otitis and neoplasia are common concurrent disease processes. Also dioestrus results in increased growth hormone levels and typically will cause DKA when the intact female diabetic dog is in season.



**Fig 1 Duodenum and right limb of the pancreas just distal to it. Pancreas is mildly hypoechoic.**

#### Images of Pancreatitis



**Fig 2 Body of the pancreas: markedly heterogeneous with irregular hypo-to-anechoic areas.**

Patients with DKA are divided into those who are “healthy” and those that appear sick. Healthy DKAs only display clinical signs typical of diabetes (pupd, polyphagia, weight loss) and present without a history of vomiting, anorexia or lethargy. These patients will have only trace-to-small amounts of ketonuria. They can be treated like an uncomplicated diabetic. Sick DKA patients have other systemic signs such as vomiting and lethargy. It is not uncommon for them to be presented semicomatose. They are true emergencies. Signs may reflect concurrent illnesses in addition.



Diagnosis of DKA is relatively straightforward, requiring only serum glucose, venous blood gas analysis and urinalysis. Patients are characterized by having hyperglycaemia, metabolic acidosis and ketosis. A minimum database (complete blood count, biochemistry, complete urinalysis and urine culture) is necessary to rule out any concurrent disorders such as pancreatitis. It may be difficult initially to assess renal function because of prerenal azotaemia and osmotic diuresis secondary to glucosuria.

Fluids are the most crucial aspect to treating DKA. Rehydration alone will lower blood glucose. It is often beneficial therefore to start patients on fluids for some hours (3-6 hours or even more) prior to giving insulin, especially in cats. Choice of replacement fluid to use in DKA is often debated; 0.9% saline is usually recommended since these patients are often hyponatraemic. However, because of the acidifying effect of 0.9% saline, a balanced isotonic electrolyte solution (Hartmann’s, LRS or plasmalyte) may be preferred. Fluid rates should be determined after assessment of the patient. Hydration status must be evaluated several times a day and rates adjusted.

Electrolyte imbalances (especially hypokalaemia, since insulin will move K<sup>+</sup> intracellularly) must be corrected. In case of refractory hypokalaemia, magnesium should be assessed to look for hypomagnesaemia and be corrected. Hypophosphataemia is a concern in DKA patients due to renal losses and is worsened when the acidosis is corrected. This may be life-threatening as haemolytic anaemia may develop. Correcting the hypoperfusion is very beneficial for correcting the acidosis. If the patient is extremely depressed and has a pH < 7.1 or a bicarbonate level < 12 mEq/L, therapy should be considered.



Insulin therapy is essential in treatment of DKA to normalize serum glucose and allow ketones to be utilized and inhibit the formation of ketones. Regular insulin is

used because it has a rapid onset and brief duration of effect. Intermittent intramuscular or constant low-dose intravenous infusion regimes can be used. In both cases the patient's blood glucose should be monitored every 2 hours. A central line or peripherally inserted central catheter (PICC) is very useful for multiple

blood samples. The goal is to keep blood glucose between 7 and 14 mmol/L, and dextrose fluids should be initiated. Once the patient is eating and stable, longer acting insulin should be used.

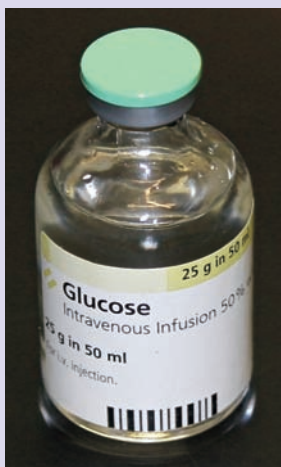
### Hyperosmolar nonketotic diabetes mellitus

This is a less common complication of DM. It has a similar pathogenesis to DKA with a relative deficiency in insulin. For the hyperosmolar syndrome to develop, some functioning  $\beta$  cells must still be producing insulin. The existence of some insulin prevents the formation of ketones.

This syndrome is characterized by severe hyperglycaemia (>33 mmol/L), hyperosmolarity (>350 mOsm/kg), dehydration and lack of ketones. These patients may still be acidotic despite the lack of ketones due to lactic acidosis (end product of anaerobic metabolism of glucose). Patients are typically quite depressed. They may be comatose upon presentation. The history of weakness may be several weeks prior to presentation.

Treatment is similar to DKA. Imbalances must be corrected very slowly. Fluid therapy is typically 0.9% saline or hypotonic 0.45% saline. Caution must be exercised to not decrease osmolarity too quickly (decreasing it by  $\frac{1}{2}$  - 1 osmol/hour). The goal should be to correct dehydration over 36 hours. While patients may still be hypokalaemic, this does not tend to be as severe as in DKA. Hypophosphataemia is less of a concern as well. Insulin therapy is the same as for DKA and these patients must be monitored very closely (urine output, electrolytes and renal values).

### Hypoglycaemia



Hypoglycaemia is defined as blood glucose < 3.3 mmol/L. Symptoms include weakness, ataxia, seizures and coma. Common causes include liver disease, excessive insulin (iatrogenic or endogenous), decreased diabetogenics such as cortisol, hypoglycaemia of toy breed puppies, sepsis. Severe hypoglycaemia is most common with insulin-secreting tumours (such as insulinoma), portosystemic shunts and in toy breed puppies.

Symptomatic patients require some form of glucose supplementation. Patients who are ataxic but still mentally alert may be fed. Seizing or comatose patients will need intravenous administration of dextrose. Caution has to be exercised in administering dextrose to insulin-secreting tumour patients. These tumours may produce more insulin after dextrose boluses, worsening the hypoglycaemia. Dogs that present seizing are a therapeutic challenge. In all cases the underlying cause should be addressed.

# NKR VETERINARY SPECIALISTS (North Kent Referrals)

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## Acupuncture

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