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Medical Nursing Case Challenges Mini Series

Session One: Endocrine Challenges

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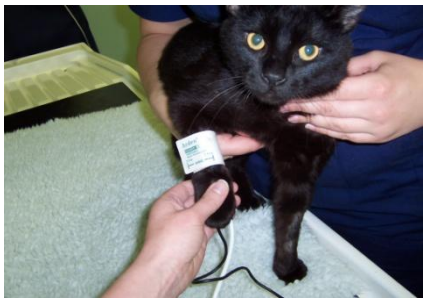


Diabetes.

Diabetes mellitus is a complex disease, with stabilisation of blood glucose levels being affected by confounding disease processes, efficacy of the primary disease control treatment, diet and exercise programme and weight control. Thus a full history of the animal, including all these factors must be taken. There are several possible causes of diabetes mellitus, including pancreatitis, obesity, drugs (glucocorticoids, progestins), concurrent illness (hyperadrenocorticism, acromegaly), genetics, immune-mediated insulitis, infections and Islet amyloidosis. Obtaining an ideal body condition score in both cats and dogs is the ideal goal in all of these cases, with obesity increasing the risk of non-insulin dependent diabetes mellitus (NIDDM) in cats by fourfold. Obese diabetic animals may have difficultly losing weight, but stabilisation of the diabetes is the initial aim, followed by a conservative weight loss programme, which does need to be carefully monitored by a veterinary professional.

Underweight animals once stabilised should be fed a modest increase in calories in order to promote repletion. Dietary therapy can only help to improve glycaemic control, but emphasis should be placed on adjustment of the insulin (or oral lypoglycemic – not commonly used due to efficacy) dosage and schedule, and control of concurrent disease.[1]

With IDDM, the beta cells within the pancreas lose their ability to secrete insulin. This can be congenital or as a result of pancreatitis, or prolonged disease to the pancreas. Exogenous insulin administration is required as the treatment. The monitoring of pancreatic specific enzymes should also occur when conducting routine monitoring of these cases, as it can be a primary cause. NIDDM is defined as insulin resistance occurring at the site of the peripheral tissues. Dysfunction of the beta cells can also be a causal factor of NIDDM, (Figure 12A). The quantity of insulin secreted by the beta cells can be increased, decreased, or can remain normal. In some texts Type 2 diabetes, which resolves is sometimes classed as transient Type 2 NIDDM. This is more commonly noted in obese cats when insulin resistance becomes established. Once the cat obtains and then maintains an ideal body condition score, NIDDM can resolve itself. If the beta cells also become exhausted; a period of exogenous insulin administration may be required. The beta cells can start secreting insulin after a period of time, and therefore is not a true NIDDM. Hyperglycaemia is toxic to beta cells and aggravates the situation by further reducing insulin secretion. This mechanism can also explain why the more obese the animal and the longer that this animal has been obese the greater the incidence of the onset of diabetes.

Diagnosis of Diabetes.

Glycated protein levels

Fructosamine and glycosylated haemoglobin

Fructosamine and glycosylated haemoglobin (GHb) are 2 glycated proteins commonly used for monitoring diabetic human patients. These 2 proteins are markers of mean glucose concentration and their amount is proportional to the blood glucose concentration. The concentration of these proteins is not affected by stress, therefore they are often used by veterinary practices to diagnose and monitor diabetic cats.

Although fructosamine and GHb are good tools for determining regulation, they will not identify an underlying problem, nor will they replace glucose curves done for therapy adjustments. Rather, they give an idea of glycaemic control over a long period: fructosamine reflects the glycaemic control for the previous 2 to 4 weeks and GHb for the prior 2 to 4 months.

Fructosamine is preferred over GHb to assess glycaemic control. It is more commonly evaluated than GHb, because simpler, less time-consuming analytical assays are available. Also, successful monitoring and regulation can be achieved with weekly or monthly measurements of serum fructosamine.

It is important to measure fructosamine when the animal is well hydrated and not acidotic. The fructosamine level can change by 100umol/l with no change in the glucose level in a 24hour period.

Advantages of measuring fructosamine

- Distinguishes hyperglycaemic, non–diabetic cats from diabetic cats with chronic hyperglycaemia.
- Not influenced by stress hyperglycaemia in cats.
- Useful in confirming diagnosis in cats.
- Helps evaluate long-term control and owner compliance with insulin treatment.

Limitations of fructosamine measurements

- Unable to detect short-term or transient abnormalities in the blood glucose concentration, eg, transient daily episodes of hypoglycaemia. This would require serial measurement of blood glucose concentrations.
- Hyperthyroid cats with diabetes mellitus may have decreased fructosamine concentrations despite having normal serum protein concentrations. This results from an increase in the protein turnover rate (decreased protein half-life) caused by increased thyroid hormone concentrations.
- Globulin and fructosamine concentrations are correlated in cats. Hypoglobulinemia will result in decreased fructosamine concentration—consult the laboratory performing the analysis as to whether a correction is required and whether this has been done.

Fructosamine is affected by quick metabolism: therefore it is normal to low levels in hyperthyroid cats. Diabetic and hyperthyroid: Fructosamine is not accurate in telling glycaemic control. Diabetic cats with fructosamine below 400umol/l by suspicious of hyperthyroidism.

Glycosylated hemoglobin (GHb)

GHb is produced by the non-enzymatic, irreversible binding of glucose to haemoglobin in erythrocytes. The glycation of haemoglobin is a gradual process and is not affected by acute or transient hyperglycaemia.

Use GHb concentration as a screening test for diabetes mellitus, as well as to monitor glycaemic control in treated diabetic animals.

Advantages of GHb measurements

- Unaffected by stress-related or postprandial hyperglycaemia.
- Useful in long-term monitoring of diabetic animals over the previous 2 to 3 months (2-4months in dogs).

Limitations of GHb measurements

- Test not widely available for cats.
- Not the most effective test due to the relatively long erythrocyte lifespan (~68 days in cats, ~110days in dogs).
- Less effective for short-term monitoring than fructosamine, because hyperglycaemia must be present for at least 3 weeks before increased values are detectable.
- Affected by haemoglobin concentrations—may be increased or decreased due to polycythaemia or anaemia, respectively.

Urinalysis.

Performing good urinalysis is important in both diagnosis of diabetes but also during the stabilisation period. Urine dipsticks should show:

- Bilirubin: Dogs negative to +1, cats negative.
- Blood: Negative; positive results may be caused by trauma induced by collection method.
- Glucose: Negative.
- Ketones: Negative.
- Nitrite: Test pad unreliable in cats and dogs.
- pH: 5.5-8.5
- Protein: Negative; trace to +1 in highly concentrated samples.
- Specific Gravity: Test pads unreliable in dogs and cats.
- Urobilinogen: Negative
- White cells (leukocytes): Test pad unreliable in cats and insensitive in dogs.

Urine glucose is more useful for adjusting insulin dose with glargine or determir than the intermediate-acting insulins (Lente). It isn't useful for detecting remission. This is as once at the correct dose of insulin, the pet should have no glucose in its urine (or just a trace).

Sediment analysis is also important, and it should be checked whether there is an active sediment or not. Normal findings include:

- RBCs/hpf : 0-5 (Feline and Canine)
- Casts/lpf : Occasional hyaline (Feline and Canine), no others should be seen.
- Epithelial Cells/hpf : Occasional
- Fat Droplets /hpf: Uncommon in dogs, common in cats.
- Bacteria /hpf: Negative
- Crystals/hpf: Variable

Bacteriology of a urine sample needs to be performed ideally from a cystocentesis sample. Voided sample will be contaminated, if sent to an external laboratory need to ensure that the lab knows how the sample was collected. All sample will be cultured for bacteria. Sediment analysis can give false negative very easily. Urinary tract infections can make the pet easily unstable.

Performing Serial Blood Glucose Tests.

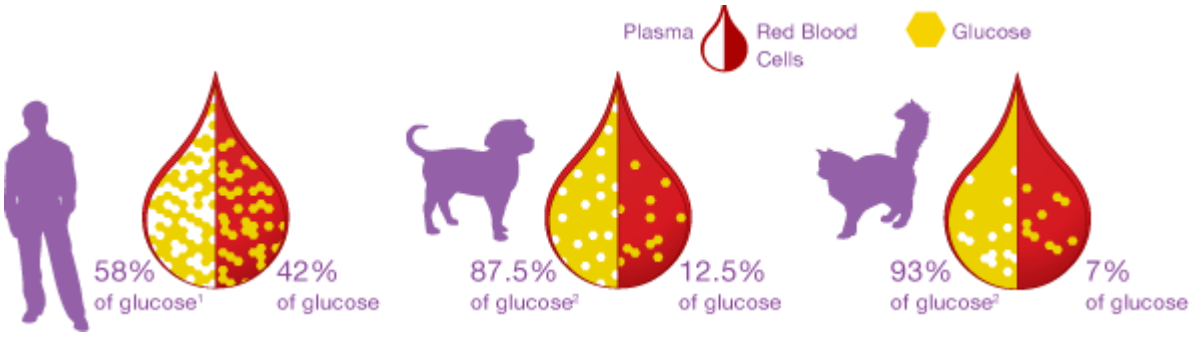
Serial blood glucose testings are useful for the monitoring of insulin therapy in dogs, and initial assessment of the response to insulin in cats. When initiating insulin therapy serial blood glucose levels should be monitored every two hours. The purpose of the monitoring is to establish whether the insulin is effective, length of effectiveness and time of nadir. There is a tendency when performing serial blood glucose tests to plot a graph and join up the dots. This can lead to misinterpretations of graphs, as if tests are two hours apart, glucose levels can be lower or higher than expected and not on the line drawn between two readings. Serial blood glucose testing is difficult in cats, and it has been shown in cats that even on consecutive days serial blood glucose testings would have caused two very different conclusions in what the veterinary surgeon would have interpreted the results and altered the insulin levels. More accurate levels can be achieved through monitor blood glucose levels at home.

Any changes in insulin should be performed slowly, and only by half a unit every 7-10days if required. It takes the body over a week to adapt to insulin level changes and therefore more rapid changes can be detrimental.

Reasons for using serial blood glucose testing:

- Determine need for insulin / Control glucose levels
- Determine the effectiveness of the treatment
    - Length of action of each injection.
    - Identify timing of nadir
    - Identify blood glucose level at nadir
- Avoid / minimise
    - Visible symptoms
    - Consequences of poor control
    - Long-term effects of diabetes (cataracts, renal and liver problems, etc.).

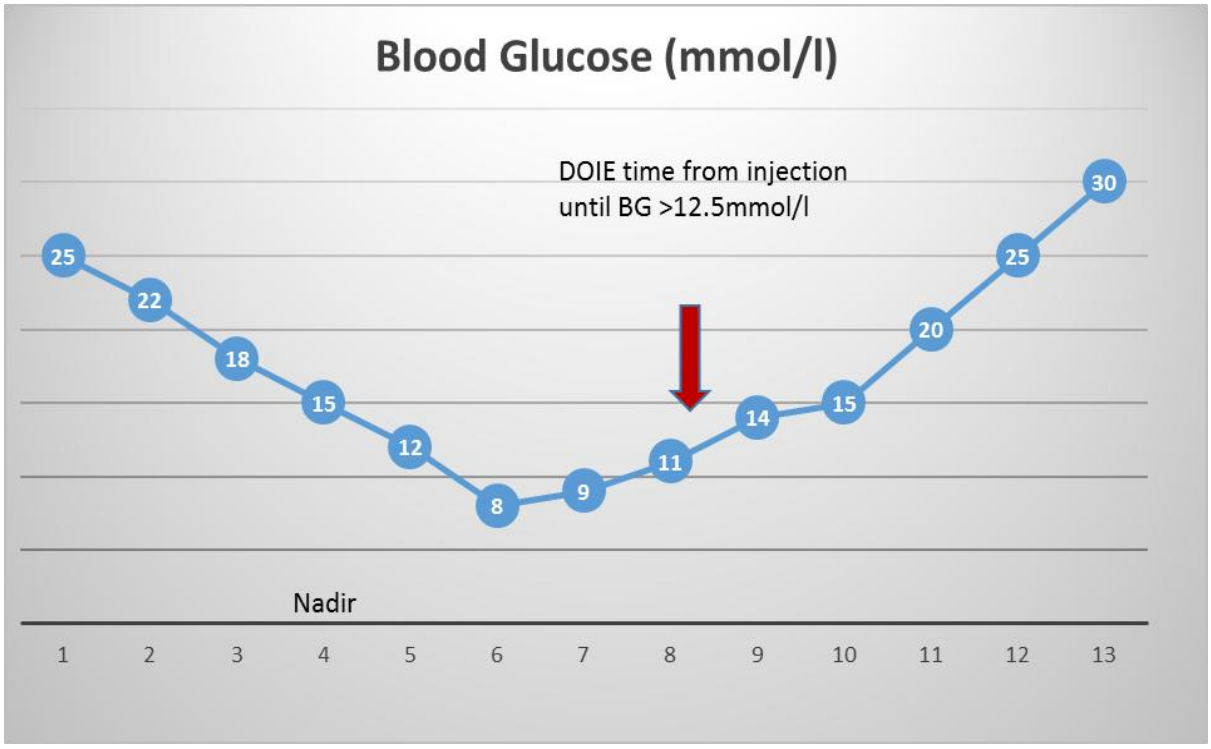
It is important to use a species specific monitor as there are large variations between species, it gives more accurate readings for the pet. Some insurance companies will allow claims for glucose monitors.



There are different types of human glucometers and some owner can't or won't want to buy a species specific glucometer. Human whole blood calibrated monitors are approximately 20-25% lower than species specific (~1-2mmol/l). Human plasma equivalent monitors tend to read 10-15% lower. If owners are using human glucometers, find out type and adjust for the glucose concentration.

Definitions:

- Nadir: Lowest point of the blood glucose.
- DOIE: Duration of Insulin Effect, this is defined as the time from insulin injection until the blood glucose exceeds 12.5mmol/l.
- SBG: Serial blood glucose.
- SBGC: Serial blood glucose curve.
- NPH Insulin: *Neutral Protamine Hagedorn* Insulin.
- PZI: Protamine Zinc Insulin.



Types of Insulin.

Caninsulin manufactured by MSD, is a veterinary porcine lente intermediate-acting insulin, made up of 30% semilente (short-acting) and 70% ultralente (long acting) insulins. ProZinc from Boehringer – Protamine Zinc insulin (long acting insulin). Long acting insulins such as Glargine and Determir are both not licenced for the veterinary market. Likewise Neutral (Soluble) insulin – solely used in cases of diabetic ketoacidosis.

Caninsulin.

Caninsulin is structurally identical to canine insulin, and is licensed for both canine and feline use. The licenced starting insulin dose range is:

- 0.25- 0.5IU/kg **twice** daily - larger bodyweight dogs started at the lower end of the range.

Dose can also be dependent on initial blood glucose concentrations. Remember to round the pet's bodyweight down to the nearest whole kilogram and the calculated dose down to the nearest whole unit.

- Dosage Blood glucose concentration

Starting insulin dose

<20mmol/l (360mg/dL) = 0.25IU/kg

>20mmol/l (360mg/dL) = 0.5IU/kg

ProZinc.

ProZinc (PZI insulin) has a long duration of effect, with the glucose nadir normally 9hours post administration. It is a recombinant human insulin in a protamine zinc combination. Only four amino acids differ from feline insulin.

Stabilising the Diabetic Patient.

Our initial aims are to control clinical signs, (PD/PU, polyphagia), and to maintain the blood glucose between 5.5 to 14mmol/l. Performing the serial blood glucose testing will give you guidance. The first thing to look at is does the blood glucose level drop when insulin is administered. If it does then good. If it doesn't and the insulin dose is <1iu/kg bwt then consider insulin under dosage. If insulin dose is >1 to 1.5iu/kg bwt then consider insulin ineffectiveness or insulin resistance. If the fructosamine level is <450µmol/l consider Somoygi over swing, poor injection technique or stress hyperglycaemia.

The ideal blood glucose level is between 5.5 and 7mmol/l, (though there are a lot of different reference ranges). If the nadir was >8.5mmol/l, then the insulin dose can be increased by 10%, and reassessed in 7-10days time. If the nadir was <5.5mmol/l, then the insulin dose can be decreased by 10-20% and reassessed in 7-10days time. Based on using Caninsulin.

If using Glargine or Determir if pre-insulin >10mmol/l, and the nadir >8mmol/l, increase the insulin by 0.5-1IU. If the pre-insulin is >10mmol/l and the nadir 5-8mmol/l. Keep the insulin at the same levels for several weeks, then aim to reduce the nadir to 4-7mmol/l. If the nadir <4.5mmol/l and the pre-insulin <10mmol/l decrease by 0.5-1iu/injection.

There are three phases of treatment with Glargine therapy.

1.) Increasing the dose every 5-7days by 0.25-1iu depending on current dose of insulin and degree of hyperglycaemia. Aiming to increase the dose until all blood glucose concentrations are within 4-11 throughout the day.

2.) Holding the dose, once within 4-11 throughout the day.

3.) Decrease the dose if pre-insulin <10mmol/l OR nadir <4mmol/l.

If near remission, keep to the trigger points (pre-insulin and nadir levels). Keep decreasing insulin by 0.5iu/cat bid through to 1/2iu/cat SID. If the pre-insulin is 10mmol/l on 0.5-1iu SID (per injection) withhold the insulin and check for remission. Need to overcome the glucose toxicity for the beta cells to recover and go into remission.

Pancreatitis.

Diagnosis can be difficult in these cases, but using Species specific pancreatic Lipase tests alongside clinical signs is important. The statistics are quite high with 50% of diabetic cats, and 13% of diabetic dogs having pancreatitis. The inflammation can be fluctuating and therefore the insulin requirements can also vary. Regular testing should occur for those that have proven to have pancreatitis; and be instigated for those where there are issues with stabilisation.

Nursing the Diabetic In-Patient.

Hypoglycaemia.

Clinical signs of hypoglycaemia should be explained to pet owners at the time of the initial diagnosis of diabetes mellitus; though the signs will differ from patient to patient.

Signs can include:

- Lethargy
- Ataxia
- Muscle twitching
- Severe seizures
- Disorientation
- Lack of responsiveness
- Coma

Peripheral blood sampling can be very difficult in a hypoglycaemic animal, with signs of shock being displayed and peripheral vasoconstriction. Treatment of the clinical signs is urgently required, followed by close monitoring and investigation of the cause of the hypoglycaemia. Administration of glucose followed by a meal of highly digestible food is required.

Care is required when administering intravenous glucose as it is an irritant when administered in high concentrations. All glucose solutions should be diluted with sterile saline or water for injection, to make a 10-20% glucose solution for administration via a peripheral vein. Higher levels can be given if a central line is utilised. Glucose supplementation is more effective when a loading dose is administered followed by a continuous rate infusion (CRIs). As with all CRIs, they aid to remove the peaks and troughs of repeated boluses.

It is important to actively encourage the animal to eat after glucose administration. The initial cause of the lower blood glucose level can still be ongoing, and therefore a more sustained supply of glucose can be required. A highly digestible diet should be fed as this will provide simple carbohydrates to the animal a lot quicker, than a high fat diet. Dextrose powder can be mixed into the diet to increase the simple sugar contribution. High fat diets need to be avoided as this will cases delayed gastric

empting. The ingested food needs to reach the small intestine in order for the simple sugars to be absorbed. A low fat, low fibre, highly/easily digestible diet should be used in order to get the ingested simple sugars into the small intestine as quickly as possible.

After a diabetic has experience a period over low blood glucose levels, the body produces other hormones that will cause an increase in blood glucose level. These Somorgyi over swings are primarily due to too much insulin being administered, (maybe as the overall requirement is decreased through many different reasons). Performing a glucose curve after a hypoglycaemic episode will show an increased blood glucose due to these compensatory mechanisms.

Diabetic Ketoacidoisis.

Can be seen in those animals that are previously thought to be healthy, or those that have been previously diagnosed with diabetes mellitus. Cats can undergo waxing waning levels of insulin (and thus requirements). If a diabetic dog (that has been previously stable) presents with DKA, we need to do further investigations to what mechanisms of insulin resistance are present.

Clinical Signs can include:

- Polyuria/polydipsia
- Anorexia
- Vomiting and/or Diarrhoea
- Depression
- Weakness or Collapse
- Poor body condition
- Hepatomegaly
- Acetone smell on breath
- Deeper more rapid respiration reflecting metabolic acidosis.

Diabetic Ketoacidosis (DKA) can arise in an animal with previously diagnosed diabetes mellitus (DM) or it can appear to occur suddenly in an animal that the owner thought to be healthy. For DKA to develop there is usually a triggering condition, as shown in the table below. This triggering condition then causes the glucagon:insulin ratio to increase. Glycagon promotes glycogenesis and the

formation of ketoacids. Insulin is also required for metabolism of ketones to carbon dioxide and water. When there is a low level of ketone production in uncomplicated DM, the ketones can be metabolised and do not build up to the point of which they cause clinical signs.

Bacterial Infections

Any significant infected focus but especially:

- Urinary tract infection – upper and lower
- Prostatitis
- Pneumonia
- Pyoderma
- Otitis externa
- Severe gingivitis/oral abscesses from tooth root infections

Inflammatory Disease

Pancreatitis

Endocrinopathies or physiological endocrine changes

Hyperadrenocorticism (cats and dogs)

Acromegaly (cats and dogs, though different mechanisms)

Hyperthyroidism (cats)

Hypothyroidism (dogs)

Phaeochromocytoma

Dioestrus phase of oestrus cycle

Iatrogenic causes

Steroid therapy – including intra-aural or ocular

Table: Conditions that can trigger DKA through insulin resistance.

Initial Assessment of DKA Patients.

The initial assessment of these patients needs to completed as rapidly as possible. Parameters to be measured include:

- Packed cell volume (PCV) alongside total solids/proteins
- Electrolytes – sodium, chloride, potassium (and phosphorus)
- Renal function assessment – urea, creatinine and phosphorus
- Blood gas analysis
- Blood ketone levels
- Urine analysis
    - Specific Gravity
    - Dipstick analysis – assess for ketones
    - Sediment examination
    - Submit for culture

Blood serum ketones should be tested for, but many practices don't have the facilities in order to do this. Urine dipsticks can be used to measure ketone levels, but do not measure the most predominant ketone body (β- hydroxybutyrate), therefore false negatives can be seen. The addition of hydrogen peroxide to the sample to oxidise the β-hydroxybutyrate to acetoacetate will enable the urine dipstick to measure all of the ketone bodies.

Small handheld ketone meters are becoming more readily available, and are fairly inexpensive.

Reference ranges for ketones are often quoted as:

- Fed State: 0.1mmol/l
- Overnight fast: 0.3-0.7mmol/l
- Metabolic foods: 1-3mmol/l
- Diabetic Ketoacidosis: >15mmol/l

The overnight fast has a higher ketone level than those fed a normal diet, as the body is using stored body fats as an energy source. Metabolic diets includes Purina DM and Hills m/d diets.

Management of DKA Patients.

The initial management of DKA patients is to provide insulin in order to reduce the hyperglycaemia and promote ketone metabolism whilst correcting the intravascular volume, correcting hydration levels and any electrolyte imbalances.

Fluid Therapy.

Fluid therapy is a vital aspect of the initial emergency treatment of DKA. The aims of fluid therapy are to:

- Address dehydration issues.
- Correction of Acid base balance.
- Create a means in which to deliver medications, glucose, electrolytes.

Current recommendations are to address dehydration and correct potassium prior to administration of insulin.

In order to administer fluids we need to ensure that we have correctly calculated the correct requirement for the patient. The percentage of dehydration and the calculated deficit needs to added to the maintenance requirements of the patient.

Percentage of dehydration	Clinical Signs
<5%	No obvious outward signs Concentrated urine
5-8%	Slightly prolonged CRT Slight tenting of the skin Mucous membranes feel tacky Third eyelid visible
8-10%	Sunken eyes Prolonged CRT Obvious tenting of the skin
10-12%	Oliguria Tented skin remains in place Clinical shock can be experienced
>12%	Progressive shock Coma and death.

The level of hydration of the animal needs to be accessed. This can be completed through clinical examination, looking at haematology parameters such as PCV and Total Protein levels. Once the fluid requirements have been calculated, then need to determine if a shock rate bolus is required. Need to also ascertain whether additional IV access lines are needed. Especially if potassium and insulin are going to be administered intravenously.

When using PCV to access whether the animal is dehydrated it needs to be done in conjunction with Total Protein levels. TP can be measured on a urine refractometer. Depending on levels will help guide with assessment with fluid requirements.

- Increase in PCV and TP = dehydration
- Decrease in PCV and TP = aggressive IVFT, haemorrhage.
- Decreased PCV and normal TP = possible increased destruction of RBCs.
- Increased PCV and decreased TP = dehydration with protein loss, e.g. gastro enteritis.

Shock rate for isotonic crystalloids will vary greatly. The values below are guidelines and are per hour. A bolus for a defined period of time is then administered, for example 15mins. The animal is then examined again and a discussion made to whether another bolus given whether the animal is moved onto more maintenance requirements/rates.

- Shock dose = 60-90ml/kg (dog) or 40-60ml/kg (cat)
- Moderate hypoperfusion = 30-50ml/kg (dog) or 10-20ml/kg (cat).
- Mild = 10-20ml/kg (dog) or 5-7ml/kg (cat).

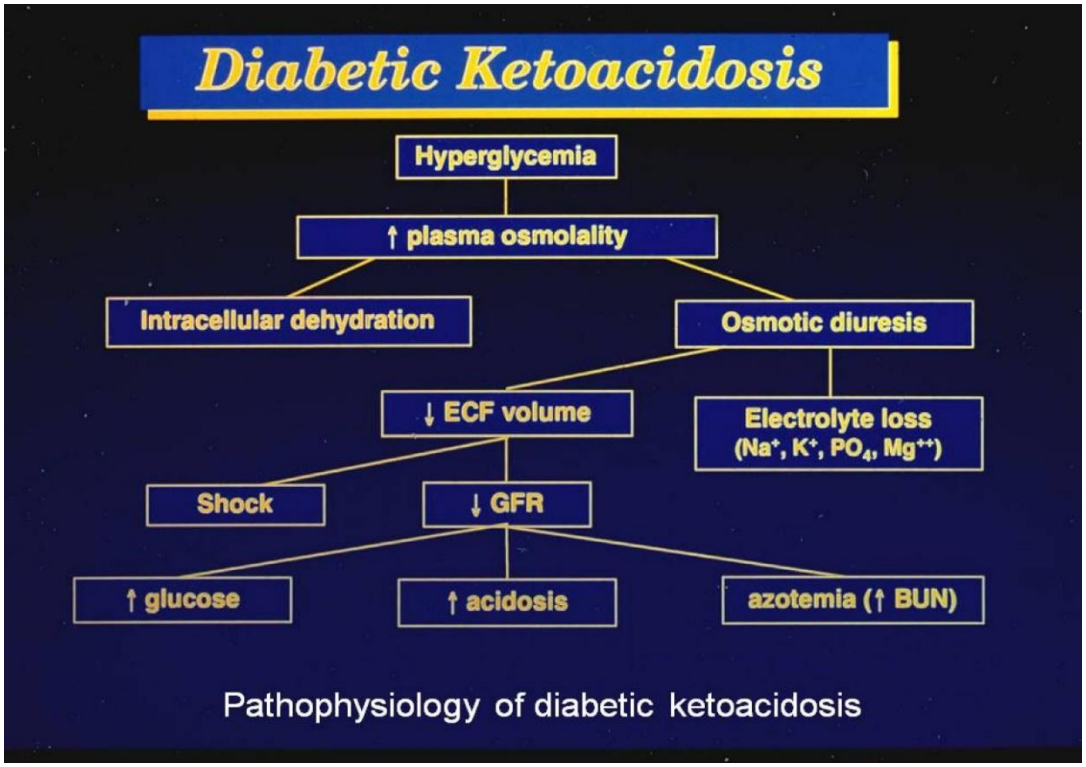
A fluid pump, syringe driver or burette should be utilised in order to achieve correct volumes being administered.

When calculating total fluid requirements we need to include:

- Maintenance fluid needs (in a non-diabetic) approx. 50mls/kg/day
- Replacement of ongoing losses associated with polyuria, typically 25ml/kg/day
- Correction of volume deficits over a period of 24hours (typically 50-100ml/kg/day)

The rate of fluid administration depends on the clinical assessment of hydration status, degree of shock, and the presence of concurrent disease, which could limit the rate of infusion.

In many cases Hartmanns solution (lactated Ringers, LRS, No 11) should be used, in order to aid with acidosis. In severe cases sodium bicarbonate can be administered, but this should only be done with the use of blood gas analysis analysers and syringe pumps/ syringe drivers. Dial in giving sets are not accurate enough, and are dependent on positioning of the animal, if administering sodium bicarbonate. In many cases the acidosis will be corrected as the ketone levels are decreased. This occurs as soon as fluid administration occurs as the total circulating volume is increased and therefore has a dilution effect on the amount of ketones and hyperglycaemia.



Electrolytes.

Many DKA patients can initially present with a ‘normal’ potassium level at initial assessment. Diabetes and especially DKA cause total potassium and phosphate depletion because of a shift of these electrolytes out of the cells into the serum to replenish losses and to help off-set acid-base imbalances. IVFT (given during the treatment of DKA) will further dilute / decrease electrolyte concentrations as will insulin-mediated uptake of phosphate and potassium by the cells, and renal losses. Potassium and phosphate levels needs to be monitored prior to administration of treatment and throughout.

	Typical Guidelines	Guidelines for DKA
Serum Potassium (mEq/L or mmol/l)	KCl (mmol/l) to add to 1L of fluids	KCl (mmol/l) to add to 1L of fluids
>5.0	Wait	Wait
4.0-5.5	10	20-30
3.5-4.0	20	30-40
3.0-3.5	30	40-50
2.5-3.0	40	50-60
2.0-2.5	60	60-80
<2.0	80	80

Total hourly potassium, administration should not exceed 0.5mEq/kg bodyweight. Figures from Syme (2016).

Phosphate levels should be monitored alongside the other electrolytes (Na, Cl and K). Phosphate ions follow the same path as the potassium ions. The high extracellular glucose levels encourage movement of water, potassium and phosphate out of the cells. When fluid replacement and insulin therapy, the electrolytes are taken up into the cells. Hypophosphataemia often becomes most severe on the second day of therapy.

Phosphate supplementation is recommended if the phosphate concentration drops below 0.35mmol/l. The phosphate supplementation rate suggested in many formularies (0.01-0.03mmol/kg/h over 4-6 hours) is often insufficient in these patients and doses up to 0.12mmol/kg/h for 12-48hours may be required. Regular monitoring of phosphate (every 4-12hours) is necessary, with dose dependent adjustments. Take care as many phosphate supplements are supplied as potassium phosphate and you will need to take care of not over supplementing the potassium.

Severe hypophosphataemia can lead to haemolysis. In cats haemolytic anaemia will occur when phosphate concentrations decrease to less than 0.3 to 0.45mmol/l. Always check for haemolysis in

the sample, especially on the second day where phosphate levels can drop severely. In some severe cases blood transfusions can be indicated. Figures of PCVs dropping below 20% being a cut-off point.

Insulin Therapy.

The main therapy for DKA patients is through insulin, and this can be achieved in two different formats dependant on the equipment that is available in practice.

Intramuscular Insulin:

- Begin treatment with 0.2IU/kg intramuscular bolus of neutral (soluble/regular) insulin.
- Repeat intramuscular injections of 0.1IU/kg hourly according to blood glucose measurements, measurements need to be kept within 8-15mmol/l.
- If the blood glucose drops below 8mmol/l, add 5% dextrose to the intravenous fluids, monitor blood glucose and continue insulin therapy if possible.
- Use neutral/soluble/regular insulin for the initial therapy or until the animal begins to eat reliably. Once eating the animal can be changed onto maintenance stabilisation using a longer-acting insulin.

Intravenous Constant Rate Infusion (CRI):

- Mix the neutral/soluble/regular insulin with fluids such that it will be delivered at a dose rate of 2.2IU/kg/day.
- Use of a syringe driver or fluid pump needs to be used in order to have accurate administration of the insulin. A flow rate of 1-2mls/kg/hr can be administered for the insulin, and addition fluid therapy can be run alongside.
- The insulin mixture needs to be protected from light, covered with aluminium foil, or bandage, and freshly made up every 24hours.
- As insulin binds to plastic tubing in drip lines, prior to administration the fluid mixture needs to be run through the line until a stable solution has been achieved (30-50mls expelled).
- Blood glucose needs to be checked after 1hour then every 1-2 hours thereafter.

- Dextrose can be used as required to maintain the blood glucose between 8 and 15mmol/l.
- Long acting insulin can be introduced when the animal starts to eat.

There is a danger that 'Piggy Backing' can occur with Neutral (Soluble) insulin. This is when the on-board insulin simply refers to the amount of insulin still circulating in the bloodstream that may still be working. Stacking or piggybacking insulin refers to when a patient gives more insulin before the previous bolus of insulin has finished working. This creates even more on-board insulin. When insulin stacking is done without care or proper understanding and direction from your doctor it can lead to serious episodes of hypoglycaemia (low blood sugar). Soluble insulin can last anywhere from one to four hours, so we do need to instigate careful monitoring of patients started on this type of insulin.

Feeding on In-Patients.

Once the patient is eating sufficient regular volumes of food it can then be changed on to a regular more long-acting insulin. Daily energy requirements for these patients needs to be calculated, alongside the daily volume / weight of diet to be consumed.

1. Calculate the RER of the animal

RER = 70 x $(\text{bwt kg})^{0.75}$ for animals <2kg or >45kg, or 30 x (bwt kg) + 70

2. Add in the illnesses factor.

RER x Illness factor = kcal/day

3. Choose the specific diet, which is most beneficial for the patient, and the method of feeding.

4. Divide the energy content of the diet (kcal/ml or gram) by the energy requirement of the animal (kcal/day) to achieve the daily amount of food required.

5. Divide the total amount to be given in a day by the total amount of feeding wished to be given, or by maximum volume of each feed.

Illness factors are generally no longer used in practice, but do help to give you an ideal of what effect disease processes or recovery have on calorific requirements. Use your nutritional assessments to check whether sufficient calories are being consumed.

Anaesthetic Protocols for Diabetic Patients.

Management of blood glucose concentrations is the most important consideration when anaesthetising patients with diabetes mellitus. Owners of stable diabetic patients that are to undergo a procedure under anaesthesia should be recommended to give a half dose of insulin on the morning of the procedure. The procedures should be scheduled so that the diabetic patient is the first procedure of the day, so that it can be fed post procedure as soon as possible.

Following pre-medication a blood glucose sample should be taken for a baseline level. If the patient is already hypoglycaemic (blood glucose <4mmol/l) then intravenous glucose should be administered. Throughout the procedure if the blood glucose drops below 4mmol/l, administration of glucose should be instigated. A 5% dextrose drip can be used.

Intravenous glucose is highly irritant, and should be given diluted with saline or water. Supplementation is more effective when a loading dose is given followed by a continuous rate infusion. This will help reduce any peaks or troughs in blood glucose levels. On recovery from anaesthesia, monitoring of blood glucose should occur until the animal has recovered fully to in order to eat. A highly digestible (low fat) diet should be offered.

Pre-medication and use of alpha-2 agonists should be avoided as they cause an elevation in blood glucose during the procedure, and once reversed a refractory decrease in blood glucose level. Very careful monitoring needs to be utilised if alpha-2 agonists are therefore used.