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Diabetes mellitus

Guidance for managing diabetes in practice

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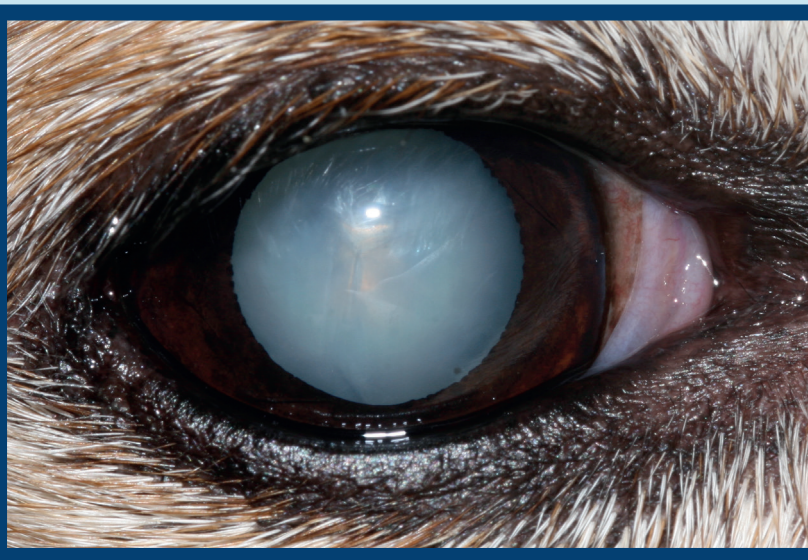

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Foreword

Diabetes mellitus (DM) is a common condition encountered in both dogs and cats. In many cases it can be managed well, but there are cases that are problematic initially, or which become unstable. This roundtable aims to provide brief, practical guidance to assist veterinary surgeons in first opinion practice to treat and monitor straightforward cases of DM, in order to provide good quality of life for both the diabetic pet and the owner; and to recognise when a case is more complicated and further testing, specialist input or referral may be needed. The initial summary of diabetes essentials outlines 'need to know' definitions and pathogenesis; owner considerations and engagement; successful treatment; and monitoring and instability. This is followed by the roundtable presentations and discussions, which provide a brief review of the pathophysiology of diabetes in dogs and cats; discuss the goals of treatment; consider both monitoring and diagnostic errors; and look at the role of nurse-led clinics. 10.12968/coan.2018.23.3.141

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NOTE: the essentials of diabetes management provided in the initial columns overleaf came out of the presentations and discussions. There was general consensus on these points; however, not all panelists agreed with all of the points.

These notes are not meant to replace the detailed (AAHA and ISFM) guidelines available, but to assist the first-opinion practitioner to make more effective use of those guidelines.

Definitions and pathogenesis

- Diabetes mellitus (DM) is by definition a deficiency of insulin (the hormone produced by the pancreatic beta cells in response to an increase in blood glucose); an inability to use insulin (insulin resistance); or a combination of these.
- Diagnosis of DM should trigger consideration of what is causing the diabetes, recognising that there are a number of different potential causes — it is not a single disease.
- Exactly what pathophysiology is driving the DM is often not known for a given animal.
- The pathogenesis and consequences of DM in dogs are different from those in cats; additionally, pathogenesis varies within each species.These differences are important as they lead to differences in responses to treatment and prognosis.
- There is a common role for obesity in cats with DM. It is worth working at weight loss in the overweight diabetic cat as this may lead to diabetic remission, whether permanent or temporary.
- If an entire female dog is diagnosed with DM then it is important that she is spayed at the earliest opportunity. While this will not always reverse the diabetes, it will certainly make stabilisation of the condition easier.
- Cats with DM due to acromegaly can be cured by hypophysectomy; however this surgery is not widely available.
- Diabetic dogs are likely to develop cataracts, even if well stabilised. If your canine DM patients develop cataracts this does not necessarily indicate poor diabetic management.
- Particularly on initial presentation, think about causes (e.g. pancreatitis in both dogs and cats, acromegaly in cats, dioestrus in female dogs), consequences (e.g. cataracts, urinary tract infections (UTIs), peripheral neuropathy), contributing factors (obesity in cats, not in dogs); complications and comorbidities (e.g. pruritis needing corticosteroid treatment; heart disease management; special dietary needs).

Monitoring and instability

- Building a relationship with the client is key for successful monitoring and management.
- The foundation of monitoring diabetic pets is the presence or absence of clinical signs: demeanour, water intake, appetite, weight and body condition score. Record everything in the clinical notes and preferably use a Diabetic Clinical Score. If the owner is happy, that generally indicates their pet has fairly well-controlled DM.
- Ask the owner to keep a paper or spreadsheet diabetic diary and/or record data via an App (RVC Pet Diabetes App/ MSD Animal Health Pet Diabetes Tracker App). The records should include the time, dose and type of insulin, clinical/ weight/behavioural information plus test (urine dipstick, blood glucose (BG)) results and their timing.
- If a diabetic pet has off-days, for example not eating, consider what else may be happening. Rule out simple explanations first, such as dental disease.
- Except for cats going into diabetic remission, negative urine glucose (UG) is not expected.
- Do not INCREASE the insulin dose based on urine tests alone. However, persistently negative UG in dogs or cats should prompt review to DECREASE the insulin dose, as it may indicate insulin overdosing — or remission in cats.
- Use of BG curves for routine monitoring of stable diabetics provides minimal advantage over clinical history and physical examination.
- Fructosamine reflects average BG over a period of time. Laboratory results vary greatly (in-house even more), so isolated fructosamine values are of little use: but **trends** can be useful. We expect diabetics to have periods of hyperglycaemia, so fructosamine is usually above the reference interval. Fructosamine within the reference interval indicates periods of hypoglycaemia, even if the owner has not noticed these.
- Consider simple and common causes of diabetic instability, e.g. administration error, insulin overdose, UTI etc., before testing for more complex causes.
- Finally, if a diabetic develops clinical signs atypical for DM, consider other more usual differentials for those signs: do not assume all problems are due to the DM.

Owner considerations

- Always consider owner quality of life: poor owner QoL may easily lead to euthanasia of a pet with DM.
- Most major concerns for owners of diabetic pets are related to the negative effects on the owner, such as not being able to send the pet for boarding, and a feeling of lack of control.
- Nurse-led clinics and a nurse contact can greatly assist in both informing clients and supporting them.
- People vary. Some owners want to know everything about their pet's DM, others just want to know what they need to do (injecting, diet etc.). Identifying which client-type you are facing can save time.
- Educating and reassuring the owner, and helping the owner to feel empowered, are important aspects of treatment.
- Engage the owner in decision-making regarding testing (for definitive diagnosis rather than 'diabetes') and treatment.
- Be realistic about costs and recognise financial restrictions. Money spent on investigation may mean lack of money available for treatment: which may mean euthanasia of the diabetic pet. Discuss the options early.
- Manage owner expectations, including costs, expected number of appointments and time to stabilisation. Also let them know that many diabetic pets do well.
- Provide information sheets for owners to take home, as many people will not take in all that they are told. Make sure they can ask questions later.
- Compliance may be improved by realistically discussing with the owner what is possible and what is not.
- Check what times for injections are practical for the owner. If stabilising in the practice, match the injection times to the likely injection times that the owner can manage at home.
- Encourage daily data gathering by the owner that provides pet QoL information. Quantitative QoL tools for diabetic dogs and cats lead to individualised and optimised diabetic care.

Successful treatment

- Each diabetic pet must be considered as an individual and treatment optimised to work with that patient.
- Insulin is needed in both insulin-deficient and insulin-resistant DM; animals with the latter may need higher doses of insulin.
- Teamwork is essential. Make use of veterinary nurses, if available, e.g. with nurse-led diabetic clinics.
- Have a practice policy on diabetic management to ensure continuity of care, including that given by locums, and that the message given to an owner is consistent. This improves management of DM cases.
- Successful treatment involves maintaining QoL for both the diabetic pet and its owner.
- The aim of treatment is to minimise hyperglycaemia and its effects while avoiding dangerous hypoglycaemia.
- Maintaining euglycaemia is ideal, but aiming for **perfect** euglycaemia increases the risk of dangerous hypoglycaemia. There is no evidence that perfect euglycaemia improves treatment outcome. In older diabetic pets it is less necessary to worry about possible long-term complications of hyperglycaemia.
- A stable daily routine for food, exercise etc. is important; matching this to what the owner can manage will improve compliance. Insulin injection should be BID but does not need to be rigidly 12-hourly: if 10 pm and 8 am is what the owner can manage, use it.
- Initial stabilisation can be carried out with the patient at home, with checks and dose adjustments e.g. every 7 days. In-patient stabilisation may be carried out faster, but is more expensive and may be less acceptable to the owner.
- Home BG monitoring can be useful (e.g. to confim/rule out hypoglycaemia): but BG monitors need to be maintained, and their calibration checked regularly.
- It is essential to use the correct insulin syringe: appropriate to the insulin concentration and of a size that is compatible with the insulin dose. An insulin dosing pen may be preferable — but training in correct use is necessary.
- No single insulin is suitable for all diabetic pets. If you successfully stabilise most patients on a given insulin type, keep using it.

This diabetes roundtable, held 15 November 2017, was sponsored by MSD Animal Health. It aimed to produce some simple, practical guidelines to assist first-opinion veterinary practices in managing their diabetic patients. The 'diabetes essentials' have been presented in the four leading coloums. The following pages outline the presentations and discussions from which the essentials have been gathered, and present additional information and explanation.

Background to canine and feline diabetes — Lucy Davison

Diabetes mellitus (DM) is a relative or absolute lack of insulin — the hormone normally produced by the beta cells of the pancreas in response to an increase in blood glucose. It occurs in both dogs and cats. Mattin et al (Vet Rec. 2014;174(14):349. doi: 10.1136/vr.101950) found a prevalence in dogs attending UK first-opinion practice of 0.34%. The median age of onset was 9.9 years, with dogs under 10 kg more likely to be diagnosed, and a trend towards diagnosis during winter. There are clear differences in prevalences between breeds (e.g. Davison et al. Vet Rec. 2005;156(15):467–71). Many cases are seen in Labrador Retrievers simply because these dogs are so common. It is not known whether different breeds get different types of DM or whether within a given breed several types of DM are found.

O'Neill et al (J Vet Intern Med. 2016;30(4):964–72. doi: 10.1111/jvim.14365) found a prevalence of 0.58% in cats, with cats over 6 years of age more likely to be diagnosed with DM and heavier cats (over 4 kg) more likely to be diagnosed. The risk was increased in Norwegian Forest, Burmese and Tonkinese cats. Nearly half the affected cats died or were euthanased within the period of the study, most within the first year after diagnosis.

Classification of DM is important (Gilor et al. J Vet Intern Med. 2016;30(4):927–40. doi: 10.1111/jvim.14357). At present there is no agreed veterinary classification system. In dogs and cats the human classification (type 1, type 2) is not entirely appropriate. Better classifications could lead to improved outcomes, as differences in pathogenesis may be driving differences in treatment responses and prognosis.

Insulin resistance is much more common in cats than in dogs. Insulin sensitivity is affected by growth hormone, glucocorticoids, progestogens, adipokines or cytokines, also by adrenalin or noradrenaline and by glucagon. Several conditions may lead to insulin resistant DM; acromegaly is among the most common in cats and hyperadrenocorticism in dogs. It can also occur iatrogenically (administration of synthetic glucocorticoids or progestogens); related to obesity in cats; in dioestrus and gestation in dogs; and in inflammatory states. It is important to recognise that insulin administration is usually needed in insulin-resistant diabetes, and higher insulin doses may be needed than in insulin-deficient individuals.

Insulin deficiency involves congenital or acquired lack of or abiotrophy of pancreatic beta cells, or the destruction of pancreatic beta cells, which may occur through a variety of mechanisms: autoimmune/immune mediated, due to exocrine pancreatic disease (pancreatitis), apoptosis, 'glucose toxicity' and others. It is more common in dogs than in cats. Once the beta cells have been destroyed the condition is irreversible and exogenous insulin therapy will be needed lifelong. The 'robustness' of the beta cells may vary between species and between individuals.

Canine diabetes may arise as a congenital disease; due to hormonal antagonism; secondary to chronic pancreatitis; and possibly as an autoimmune condition. Knowledge of the underlying mechanisms may help to inform clinical management. The **congenital form** is seen particularly in Labrador Retrievers and Labradoodles. Affected pups usually present at 6–12 weeks old, when they may be smaller than their littermates; sometimes more than one in a litter is affected. If the owner is dedicated to their management, especially during the challenges associated with growth in the first year of life, these dogs generally have a good prognosis. **Hormonal antagonism** in dogs may involve hyperadrenocorticism, dioestrus (with growth hormone produced by the canine mammary glands), progesterone-secreting tumours, or exogenous hormones e.g. treatment with progestogens or corticosteroids. **An association with chronic pancreatitis** is recognised, but it is not straightforward to determine whether pancreatitis leads to diabetes or vice versa, particularly given the problems in accurately diagnosing pancreatitis and that circulating cytokines/inflammatory indicators do not always reflect what is happening in the pancreas. Dynamic testing is needed to detect the subtle effects of pancreatitis on beta cell function. Clinically, pancreatitis may lead to insulin resistance or variable insulin sensitivity, pain and variable appetite, damage to other islet cells, and the potential for development of exocrine pancreatic insufficiency. Shared putative risk factors for both pancreatitis and diabetes include endocrine disease, obesity, hyperlipidaemia, hyperglycaemia and possibly genetic factors. **Autoimmunity** may be involved in some cases of

DM. Rarely, dogs have autoantibodies to pancreatic proteins; in a few dogs the presence of a classic autoimmune 'insulitis' islet infiltrate has been documented. There is some evidence for an autoimmune component to DM from genetic studies.

Feline diabetes must be considered separately. Similarities in DM in cats and dogs include similar signs; that DM is complex and multifactorial; that both can suffer from pancreatitis associated with DM, and that insulin therapy is usually needed immediately after diagnosis. However, feline DM has a number of differences from canine DM: **obesity and inactivity are risk factors**; often, cats have functioning pancreatic islets remaining at the time DM is diagnosed — therefore a cat may go into diabetic remission once its blood glucose (BG) has been controlled; managing exercise and diet can be more challenging for outdoor cats than for dogs. In addition, in contrast to dogs, glycaemic control in cats may be improved by a high protein/low carbohydrate diet and managed weight loss; and finally, the proportion of diabetic cats having insulin-resistant DM is higher. **Congenital diabetes in cats is very rare**. **Recognised causes include chronic pancreatitis and hormonal antagonism** e.g. acromegaly. While obesity has been associated with insulin resistance and diabetes in cats, to date there is no evidence of pancreatic autoimmunity in feline diabetes. Feline acromegaly is usually due to a functional pituitary tumour producing growth hormone; about 95% of acromegalic diabetic cats have insulin-resistant DM. Older male cats are most likely to be affected. Typically (but not always) they develop an increased body size, broadened face and larger paws. Diagnosis is by documenting elevated serum IGF-1 and confirmed by advanced diagnostic imaging of the brain. In the UK, up to 25% of cases of DM in cats may be associated with acromegaly.

Discussion

There was general agreement that the collection of clinical signs and findings (e.g. polyuria/polydipsia (PU/PD), hyperglycaemia) that we call DM is a consequence of diseases rather than a disease itself, thus it is necessary to understand what the underlying disease is if it is to be treated properly: while diabetes needs to be treated with insulin, there is a need to think further. While remission is possible in some cats, this depends on the underlying disease process (weight loss is not going to be effective if the cat has DM due to acromegaly, for example). Therefore an initial thorough investigation is needed. Owners need to see a connection between the diagnosis, which they must pay for, and the resultant treatment. Consideration of the baseline tests needed is important and is discussed under the heading Diagnostic errors. At the most basic level we need to know whether the pet still has functional beta cells, or not, and whether it has insulin resistance. Some simple tests, e.g. urine culture to check for a urinary tract infection (UTI), are relatively inexpensive and may help rule out other causes of insulin resistance.

Goals of treatment — Stijn Niessen

The goals of treatment are to ensure good QoL of the pet and its owner. In clinical terms, the goal is to reduce the animal's blood glucose to a concentration where there is minimal or absent PU/PD, polyphagia, weight loss or risk for diabetic ketoacidosis, while preventing dangerous hypoglycaemia. When a specific cause is not known, controlling BG (avoiding excessive hyperglycaemia) is recommended to decrease pancreatic beta cell death and decrease insulin resistance, particularly in cats, as this might be associated with remission in this species.

The treatment goal includes remission where this is possible and where there is a clear way to proceed towards and and reach it. Whether this is possible will depend on the underlying disease. For example, with a female entire dog presenting with DM, neutering is advised as a matter of urgency, due to the high chance of dioestus-induced DM. Between 6–46% will go into DM remission once spayed (Pöppl et al. Res Vet Sci. 2013;94(3):471–3. doi: 10.1016/j.rvsc.2012.10.008; Fall et al. J Vet Intern Med. 2010;24(6):1322–8. doi: 10.1111/j.1939-1676.2010.0630.x.). Fall et al (2010) showed that the lower the BG at the time of diagnosis and the sooner the surgery was performed after diagnosis, the greater the chance of remission. Good surgical technique should prevent issues (e.g. with haemostasis), whatever the stage of the oestrous cycle. Even if remission is not achieved, good glycaemic control is much easier to achieve in the neutered than the entire female. Trying to stabilise the bitch first is not advised — and would be more difficult.

The staple ingredients of treatment have not changed: combining the most appropriate insulin regime and the most appropriate dietary regimen, to mimic as far as possible the lost function from the endocrine pancreas (beta cells). The excellent and complicated physiological working of the beta cells has to be replaced with a much simpler system in which food and insulin are both given twice daily. Diabetic remission is not a realistic goal in dogs, with the exception of entire females by spaying. Minimising complications of diabetes would be good, for example diabetic cataracts. However, cataracts appear to occur in dogs even with excellent diabetic control.

Studies have shown that quality of life (QoL) is the most important aspect both for the pet and its owner. DM puts great strain on the bond between pet and owner; this must be acknowledged. Of the top 10 QoL concerns expressed by owners of diabetic dogs, only one was directly pet-related — the pet's moods. The others were related to the negative effects on the owner: not being able to send the animal for boarding, lack of control when all the decisions are made by the vet and the owner is simply told what to do but feels they have no control or understanding; the effects on the owner's social life; restriction of activities; costs; and worry — both in general (and worry about injecting incorrectly and killing their pet if giving too much) and about hypoglycaemic episodes (Niessen et al. J Vet Intern Med. 2012;26(4):953–61. doi: 10.1111/j.1939-1676.2012.00947.x). The aims of treatment should therefore include educating and reassuring the owner and helping the owner to feel empowered.

There is no real evidence that diabetic dogs are more likely to develop cardiovascular complications. About 80% of diabetic dogs will develop cataracts within 16 months of diagnosis; most within 5–6 months (Davison et al. Vet Ophthalmol. 1999;2(3):169–72), this is not due to 80% of diabetic dogs being poorly controlled, rather they are very susceptible: cats with DM (even if poorly controlled) do not tend to develop cataracts.

In dogs there is no reason to aim for perfect euglycaemia; there is no evidence that this will improve treatment outcome but it would increase the risk of dangerous hypoglycaemia.

Keep the treatment protocols as simple as possible for the client, which improves compliance; at the same time be flexible with protocols, to enable the owner to have a good QoL. The protocol should be adapted for the individual pet and owner. Twice daily is the minimum frequency needed in the vast majority of cases, but 12-hourly is not necessary: whether that is treating at 8 am and 10 pm rather than 8 am and 8 pm, or injecting TID not BID — whatever works for that owner and their pet. Once daily is not ideal but can be considered if the owner absolutely refuses BID. Take care with protocols not to get too strictly mathematical and ignore the individual pet and individual owner's needs.

Monitoring of QoL continuously is important — if we get this wrong it leads to euthanasia: if the protocol significantly impacts negatively on the owner's QoL it reduces long term compliance.

Quantitative QoL tools for diabetic dogs and cats lead to individualised and optimised diabetic care (Niessen et al. J Vet Intern Med. 2012;26(4):953–61. doi: 10.1111/j.1939-1676.2012.00947.x; Niessen et al J Vet Intern Med. 2010t;24(5):1098-105. doi: 10.1111/j.1939-1676.2010.0579.x).

In cats, genetic factors and acquired factors (such as abdominal fat and lack of exercise) act together to produce insulin resistance, hyperinsulinaemia, impaired glucose tolerance, acquired raised glucose, glucose toxicity, pancreatic beta cell failure, reduced insulin secretion, high glucose and DM. Understanding the pathophysiology enables treatment goals, because it IS possible to affect the condition by affecting the acquired factors. Goals should therefore include reducing abdominal fat (weight loss) and increasing activity as well as treating the DM with insulin to remove the high glucose that is negatively affecting the beta cells. DM in cats might be reversible if treatment is started fairly quickly and effectively — this does not mean aggressively. It has been proven that high blood glucose leads to glycogen deposition in the cat pancreas and to beta cell apoptosis within 2 weeks, so in cats DM complicates itself.

It is important to be aware of the possibility of acromegaly in cats (Niessen et al. PLoS One. 2015;10(5):e0127794). Cats with acromegaly have a specific cause for their DM. However, not all cats with acromegaly show obvious phenotypic signs. Acromegaly can be treated by hypophysectomy. At the RVC, with treatment of 55 cats, mortality was less than 10%, 85% of the survivors were non-diabetic within 1 week–2 months and the remaining 15% had their insulin requirements normalised. However, this surgery is not widely available.

Fleeman and Rand (J Am Vet Med Assoc. 2003;222(3):317-21) showed that there is large day-to-day variability in serial blood glucose concentration curves in a given dog. At best, blood glucose curve measurements provide us with trends. If we discover a hypoglycaemic event we have to take that seriously as the consequence can be severe. They can perhaps indicate average duration of action of the insulin in the patient. Briggs et al (J Am Vet Med Assoc. 2000;217(1):48–53) showed that the history and physical examination findings are the most important aspects when it comes to believing whether or not a diabetic patient is doing well. The clinical assessment is less likely to 'lie' to us than the blood glucose curve. The RVC's Diabetic Clinical Score has been designed with this in mind: it is based on scores of 0–3 for each of unintended weight loss, PU/PD, appetite and attitude/activity. This enables comparison of a pet from one day to another. Scoring can easily be done by the owner and used to see how the animal is doing from day to day, and also communicated to the veterinary team. It has been validated using 24-hour continuous glucose monitoring, water diaries etc. Use of a Diabetic Clinical Score should be standard whenever seeing a diabetic pet. It encourages the vet to think about the clinical signs and take the history and the collective data is used for diabetes research. There are free Apps for diabetic pet management that enable the pet owner to share information with the veterinary practice, which fosters communication and empowers the owner and involves them in the management. For example the RVC's Pet Diabetes App is a diabetic pet management tool that has been developed with and for owners of diabetic pets, and that also gathers anonymous data that are used for studying diabetes mellitus and improving the welfare of diabetic pets.

In conclusion, the goals are: treat the underlying disease when possible, as that can cure the DM (in cats, acromegaly, obesity); when a specific cause is not known, decrease beta cell death and decrease insulin resistance, particularly in cats; also in cats perhaps reduce glucose toxicity (although there is not yet good evidence of correlation between improved glycaemic control and remission — it may be that the cats that respond to tighter glycaemic control are those with more working beta cells remaining); personalise treatment to the pet-owner combination and make it doable for the owner; ensure that there is team work and that the owner feels they have control: this increases QoL for pet and owner; empower the owner.

Discussion

The discussion covered several practical aspects associated with management, particularly minimising the number of pets that will be euthanased: which requires avoiding excessive costs and maximising QoL for both the pet and the owner.

Home monitoring methods that are free, such as a diabetes diary, 24-hour water intake monitoring, or using an App (RVC Pet Diabetes App, MSD Animal Health Pet Diabetes Tracker App), should be encouraged. If a pet is maintaining weight, not showing excessive thirst, eating with a normal appetite and is generally considered to be well, then frequent consultations are unnecessary. When possible, clients should be given a nurse contact for if they have any queries: see the presentation on nurse-led clinics (below). The role of home BG monitoring was discussed; it was noted that it is possible for a pet to have a good QoL within a range of BG measurements. Trying to maintain perfect normoglycaemia or near normoglycaemia to avoid potential long-term complications, as in humans, is not absolutely necessary (most of the complications in humans take decades to develop). Note that human diabetics with BG in the low-normal range tend to feel less well than those with slightly high BG. When a diabetic pet is checked, the most important aspects are the history (thirst, appetite, exercise, demeanour at home etc.) and a complete clinical examination including body weight, body condition score (BCS) and muscle score, and coat condition (especially in cats). Urinalysis provides rapid information, and urine culture is relatively inexpensive.

Consider how financial aspects can be raised with the client. It would be useful to be able to provide numbers: 'the typical dog/cat with diabetes being treated at this practice will need X to Y appointments and take about A to B months to reach a point where less intensive dose adjustments and monitoring are required. Many dogs/cats (XX%) are 'stabilised' within this time, after which the typical costs will be £X per month and X appointments will be needed per year, although there can be extra costs associated with…'

Ideally stabilisation should involve close monitoring (e.g. repeated BG measurement), but where finances prohibit this, there is a place for cheap diabetic management involving educated compromises, with the owner accepting that there is an increased risk of hypoglycaemia during the stabilisation period.

Provide clear, accurate and consistent messages for clients and assure them early on that support will be given, e.g. using nurse clinics. Regular nurse appointments aim to avoid the development of crises for the pet — and of increased costs for the owner for emergency out-of-hours veterinary consultations.

Monitoring and insulin titration — Grant Petrie

Managing owner expectations is very important. Essential points to articulate are: it takes time to stabilise a diabetic patient; the dose of insulin typically increases in order to achieve stability — but may reduce at times, for example with remission; the frequency of treatment may change; and there may be a need to change the insulin product and start titrating again.

There are currently two sets of detailed advice available, the AAHA Diabetes Management Guidelines for Dogs and Cats (2018 edition at https://www.aaha.org/public_documents/guidelines/diabetes%20guidelines_final.pdf) and the ISFM Consensus Guidelines on the Practical Management of Diabetes Mellitus in Cats (J Feline Med Surg. 2015;17(3):235–50. doi: 10.1177/1098612X15571880).

Remember these are guidelines only – each practitioner will need to adapt any recommendations to the specific and unique circumstances of each diabetic patient.

In deciding the initial dose of insulin, safety should prevail. Primary practice may not necessarily be the place for aggressive management; rather the safe, slower approach, starting with a twice daily low-dose of insulin (0.25–0.5 IU/kg) then increasing, using an intermediate-acting preparation in dogs, and a long-acting preparation in cats.

Current expert opinion (0.25–0.5 IU/kg BID) differs from the data sheet of the only licensed insulin product for dogs (Caninsulin®, MSD Animal Health), which advises a starting dose of 0.5 IU/kg SID (0.5–1.0 IU/kg SID in the UK). In the UK (and the EU in general) it is necessary to follow the Cascade: specifically licensed products first before considering products licensed for other species such as ProZinc® (Boehringer Ingelheim) licensed for cats or lente insulin licensed for humans.

In cats, two products are licensed: Caninsulin® 1–2 IU/cat BID (1 IU if pre-insulin BG <20 mmol/l; 2 IU if >20 mmol/l) and ProZinc®, where the UK datasheet recommendation is 0.2–0.4 IU/kg every 12 hours. Products licensed for humans that might be used (following the Cascade) include insulin glargine and insulin determir (both long-acting). Based on ideal bodyweight and pre-insulin BG, expert opinion is 0.25 IU/cat BID if BG <20 mmol/L and 0.5 IU/cat BID if BG >20 mmol/l. Most cats start on 1 IU BID and the initial dose should not exceed 2 IU BID.

Oral hypoglycaemic drugs are ineffective in dogs and should only be used in cats facing euthanasia due to owner absolute refusal to give insulin.

On day 1 of stabilisation, blood glucose (BG) is measured at the clinic every 2–4 hours to assess if the animal is responding to the insulin and to check that it is not becoming hypoglycaemic. Monitoring continues until the nadir has been established — this may take 10–12 hours, or longer. Based on the initial response, the dose of insulin is assessed; if the BG nadir is too low (<4.5 mmol/l), reduce the dose in dogs by 25% and by half a unit in cats.

The goal on day one is not to achieve perfect glycaemic control but rather to avoid hypoglycaemia while starting to control hyperglycaemia. If the BG falls in response to insulin injection, without the animal developing hypoglycaemia, then the pet can be sent home for 5–14 days before review. This allows time for the owner to adjust to delivering diabetic management. At re-assessment, a detailed history and thorough physical examination are undertaken. The clinical benefit of treatment is determined plus a BG curve to assess the glycaemic response. From the glucose curve it can be determined whether the insulin is effective, the time of onset of action of the insulin, the time of peak effect, the BG nadir and the duration of action of the insulin. This is used to guide decision-making. On the ideal curve, the BG stays below 14 mmol/L at all times, the nadir is between 4.5–8 mmol/L and the duration of action is about 12 hours. However, even in clinically stable diabetics, this is rarely achieved. Decisions about changing the dose or frequency of insulin can be considered. For example, if the BG nadir is <4.5 mmol/L, reduce the dose by 25–50%, if the nadir is >8 mmol/L and there are ongoing clinical signs, increase the dose by 10–20% in dogs and by 0.5 units in cats. If the nadir is early (<6 hours) and the duration of action is considerably shorter than 12 hours, consider switching to a longer-acting preparation or injecting more frequently. If the nadir is late (>10 hours) and the duration of action is considerably longer than 12 hours, consider switching to a shorter-acting preparation or injecting less frequently. For more detailed consideration about dose and frequency adjustments, please refer to the AAHA or ISFM Guidelines. The animal is then further re-assessed every 1–3 weeks with a similar protocol to the first re-assessment — a full history, a thorough physical examination and glucose measurements — until stability is achieved. Stabilisation may take several months. The definition of a well-controlled diabetic would be resolution of clinical signs and absence of hypoglycemia.

Fructosamine, which is formed by the binding of serum proteins, notably albumin, to glucose, reflects average BG levels over the previous 1–3 weeks. It does not inform about the degree of hypo- or hyperglycaemia. Fructosamine is measured at diagnosis but not necessarily during stabilisation. It can be a useful trend monitor for improving/worsening glycaemic control, but it is important to be aware that normal fructosamine may NOT equal good glycaemic control. Remember that the reference interval for fructosamine is for non-diabetic patients. Diabetic animals typically have periods of hyperglycaemia, so fructosamine is usually above the reference interval. Diabetic cats heading to remission may have normal fructosamine. When assessing fructosamine concentrations, always look at the clinical picture too.

When monitoring stable diabetics, the most powerful tool is the presence or absence of clinical signs (PU/PD/polyphagia, weight loss). Ensure your diabetic patients are weighed weekly. Stable body weight/ideal BCS plus the absence of clinical signs (and no complications) suggests that reasonable glycaemic control has been achieved. In the stable diabetic, no further investigations are necessarily required. **A happy pet usually means a happy owner!**

Urine testing can be useful when monitoring diabetics, particularly as a trend monitor. Get owners to check the urine twice a week; changes in the pH, presence of blood or ketones may herald UTI or ketosis. It is expected that stable diabetics will have glucose in the urine. Persistent absence of glucosuria in cats may indicate remission. However, UG correlates poorly with BG and the insulin dose should not be changed based on UG alone.

Single BG measurements are of limited use. They are most useful for suspected hypoglycaemia. A pre-insulin BG of 10–15 mmol/l in a stable diabetic might indicate fair glycaemic control. The BG nadir can be used to guide dose changes but the time of the nadir may vary, making spot BG tests potentially unreliable. Blood glucose curves are not useful for routine monitoring of stable diabetics. There is day-to-day variability (Fleeman and Rand, 2003) and curves obtained at the practice often vary from those obtained at home, due to differences in stress, appetite and exercise.

Remission in feline diabetes is a realistic goal but it can be difficult to predict when it might occur. Clues include persistently negative urine glucose, clinical signs of hypoglycaemia or documented hypoglycaemia, unexpectedly normal fructosamine levels or a blood glucose curve that looks too close to normal.

Monitoring unstable diabetic patients

Unstable diabetics are those that show no clinical or glycaemic response to insulin doses >1 IU/kg, or suboptimal response at 1.5 IU/kg, and those that were stable but have subsequently relapsed. These patients show ongoing or recur-

rence of clinical signs of DM or they may develop complications. It is important to remember that an animal showing diabetic clinical signs may have another disease with similar signs. Furthermore, many ageing diabetic pets may have concurrent diseases.

Complications of DM occur due to the actual disease, such as ongoing clinical signs of PU/PD, polyphagia, weight loss or due to chronic hyperglycaemia, such as cataracts, peripheral neuropathy and ketoacidosis. Alternatively, complications can arise from inappropriate insulin therapy, such as hypoglycaemia or insulin-induced hyperglycaemia (the Somogyi effect). If a patient is unstable then further evaluation is needed. Rule out insulin administration issues and stop diabetogenic medications (if possible). Review the diet and weight management. Check serial BG concentrations. Then progress to rule out concurrent diseases with appropriate tests (for instance haematology, chemistry, SpecPL, IGF-1 etc.) and other investigations pertinent to the presenting signs.

Discussion

Asked about maximum dose adjustments, Grant said that he looks at the pre-insulin BG, the nadir, how much insulin it has taken to get to this point and how much leeway there is to take the BG down to a safe nadir. Conservatively, he uses a 10% increase in dogs and 0.5 IU in cats, but he will go up to 25–30% increase in dogs if the nadir is particularly high.

The Panel noted that there are many different ways to stabilise a diabetic — use the one that works for you! The broad principles are the starting dose (based on the optimum body weight), the type of insulin and the fact that it takes at least 3 days to equilibrate after a dose change. Some clinicians also consider pre-insulin BG in choosing the initial dose rate. Stijn added that key factors are that management should be individualised and that time is allowed for the insulin to equilibrate.

For an animal referred to a hospital for stabilisation, frequent dose changes may be undertaken, but for most UK vets, checks every 1–3 weeks at the practice and assessment of the insulin dose may work well. For these diabetic pets, stabilisation is primarily at home. Owners should be encouraged to participate in the process but we have a duty to make this achievable and not onerous. Monitoring and recording clinical signs is again vital. Owners need thorough education and support to ensure they dose correctly and know how to spot complications (such as hypoglycaemia) and how to deal with them. Home BG testing may be appropriate for a proportion of owners. While owners should be involved as much as possible with monitoring, they should be discouraged from changing the dose, without veterinary advice, based on that monitoring.

Monitoring is 'everything' — diet, appetite, activity, water intake, body weight, rather than being reliant only on BG curves, which can be very variable and misleading. Simple clinical scoring using a Diabetic Clinical Score is very useful.

When to change the diet was discussed; this may be easiest during initial stabilisation while the animal is polyphagic. Diet change after stabilisation may alter insulin requirements and require re-stabilisation.

It was agreed that feeding before or after injecting needs to be flexible: many recommend feeding 30–60 minutes after injection but with a picky animal that might not eat, injecting during or immediately after feeding might be safer.

Achieving optimal body weight should be a goal and there should be consideration for the dietary requirements of concurrent diseases. Diets with a fixed formulation are preferable, and avoid changing diets once the animal is stable.

If a patient is unstable, then think about possible explanations; and if stabilisation is proving difficult, discuss the case with a specialist and consider referral.

Diagnostic errors — Michael Herrtage

Mike started by considering how we diagnose DM: the clinical signs of PU/PD, polyphagia, weight loss, enlarged liver, raised BG; glucose and ketones in urine. Other possible causes of clinical signs and test results should be considered.

Hyperglycaemia: stress may cause BG to reach 10–20 mmol/l; this is more common in cats. Iatrogenic high BG can be seen with glucocorticoid or progestogen treatment, alpha-2 agonists, and glucose-containing IV fluids. Hormonal causes: dioestrus, hyperadrenocorticism, phaeochromocytoma, acromegaly. If it is dioestrus-related, there is about a 20% remission rate with spaying.

Glucosuria: the renal threshold is about 12–14 mmol/l but it is important to recognise that the renal threshold varies between individuals: in humans it can vary between 8–20 mmol/l. In general it might be slightly lower in dogs than in cats. Glucosuria can be due to stress, renal tubular dysfunction (Fanconi syndrome, primary renal glucosuria, renal failure and nephrotoxins). There may be interference with the test due to glucose contamination in the collecting vessel; vitamin C, or pigment in the urine making the colour change more difficult to detect, or iatrogenically with glucose-containing IV fluids.

Haematology (CBC) and serum biochemistry: a diabetic is likely to have high cholesterol and high triglycerides as lipid-protein metabolism is also affected — this fits with the diagnosis; there is no immediate need to look for other causes. Similarly, the liver enlarges due to hepatocellular swelling, causing cholestasis, so alkaline phosphatase and alanine aminotransferase rise. Stabilising animals with high liver enzymes is not more difficult. It is useful to check pancreatic lipase (cPLi, FPLi) and DGGR lipase on a routine screen and be aware if these are raised (but is it cause or effect); if the animal becomes difficult to stabilise, this information could be useful and it may be time to carry out an abdominal ultrasound. If the animal is at all sick then urea, creatinine, phosphate and electrolytes are useful; if the animal is progressing to ketoacidosis that is important. A stress leucogram would be expected: being an unstabilised diabetic is a stressor. Anaemia of chronic illness is NOT likely with diabetes, therefore if the animal is anaemic, look for what else is going on.

Full urinalysis should be performed: urine specific gravity (SG), pH, protein, glucose and ketones. Most diabetics have minimally concentrated urine e.g. 1.020; note that SG will not be raised appreciably by glucosuria. If there is evidence of haematuria or proteinuria then it is important to check if there is a reactive sediment: infections are a common cause of insulin resistance and a common reason why diabetic animals go into ketoacidosis. Any proteinuria should be quantified, and proteinuria should lead to taking a baseline blood pressure measurement and carrying out a fundic examination. If there is a UTI then culture and sensitivity from a cystocentesis sample is important. Owners should be provided with urinalysis sticks and told to contact the practice in the event of ketosis. Note the nitroprusside reaction used in the dipsticks detects acetoacetic acid and acetone, but not beta-hydroxybutyric acid. Shock-like states promote production of beta-hydroxybutyric acid, which is not detected by the dipsticks. After treatment with insulin, this is metabolised to acetoacetate, hence some animals may be ketone-negative initially but become ketone-positive after 2–3 days of treatment, despite clinical improvement. This is more likely to occur in cats, and with diabetic cats that are 'off-colour', the possibility of ketoacidosis developing should be considered.

It is not necessary to perform tests looking for other complicating conditions unless there is a real reason to suspect such a condition. Even if an animal has become unstable, unless it shows clear clinical signs indicating e.g. hyperadrenocorticism or hyperthyroidism there is no reason to carry out specific tests for these. It is important also to be aware how diabetes may affect such tests: an unstable diabetic will be stressed therefore will show exaggerated responses on the ACTH stimulation test e.g. cortisol of 650–700 nmol/l, and no suppression at 8 hours on a low-dose dexamethasone suppression test — but once the diabetes is stabilised, these values normalise.

Stabilisation of a diabetic pet requires some understanding by the owner: you should explain what you are trying to achieve. Owners vary. Some know little about DM and will do exactly what they are told to. It can be harder with owners who think they know but have incorrect ideas about DM management, as they may not follow the plan given, which makes stabilisation difficult.

Diabetes management does require some daily routine of feeding, exercise amount etc. Pets that are really stable, by a month after diagnosis only come in for syringes and insulin, and are otherwise little seen by the practice. But about 30% are unstable; perhaps 5% are brittle.

Insulin requirements are increased by infection, oestrus, pregnancy and ketoacidosis. Mike recommends giving insulin then feeding the pet about 30 minutes later. If an owner normally feeds first, a common mistake is that if the pet does not eat the owner fails to give the insulin: however, inappetence is probably a sign that something is wrong and needs investigating, but insulin is still required at this time.

Hypoglycaemic crises should be explained to the owner and they should be told what to do: it is very upsetting for the owner not to know what to do. Note that insulin tends to be metabolised more quickly in cats than in dogs, and profound hypoglycaemia may go completely unnoticed in cats — they may not show clinical signs. It is important to be aware of this.

It is important to use a syringe size that is compatible with the dose of insulin being given and matches the insulin concentration, and to be consistent with the syringe type used. One reason for a diabetic pet that has been stabilised in hospital becoming unstable after returning home is because the wrong syringe size has been supplied to the owner. The pens are very good but do require training in how to use them, how to prime them, or they do not work well.

Monitoring should include fluid intake, body weight and BCS, urine glucose/ketones. Owners might record these once or twice a week rather than daily. Water intake can be monitored in a single-pet household; if water intake increases there is likely to be some glucosuria — this can be monitored without touching the animal. For urine glucose and ketones, make sure the owner records the time of day the sample was taken and how this relates to feeding times: if the urine was sampled first thing before feeding (and insulin) then a bit of glucose would be expected — but not ketones; at 4–6 hours after insulin dosing a negative urine glucose would be expected.

A single BG measurement is not very helpful, but can be reassuring for people (to confim an animal is not hypoglycaemic).

Serum fructosamine interpretation is problematic, because values from different laboratories can vary markedly. However a fructosamine in the lower part of the reference range for that laboratory must indicate periods of hypoglycaemia (since we know that the animal will have periods of hyperglycaemia for parts of the day), which would explain if the pet has been a bit 'off': human diabetics feel better when their BG is slightly high rather than slightly low.

Investigation of the unstable diabetic should first cover the basics — these can be checked by the vet or nurse asking the owner questions, checking: is the animal being fed the correct diet; is the amount given being measured; is there a risk a cat has 'two homes' and is being fed elsewhere? Is the owner administering the insulin correctly? Resuspending the insulin before taking each dose out of the vial? Drawing it up correctly so that they are giving the correct dose? Storing the insulin correctly (not freezing it)? Owners can easily not take in all the information they were given initially, even with handouts and online guides available. Are the urine dipsticks in date? Check the animal for evidence of infection, disease, evidence of oestrus or pregnancy. If it is ketoacidotic then you know it is unstable and doing a blood glucose curve will not help. Check the diabetic record/App data — you can see trends, perhaps spot where things started to go wrong (e.g. a Labrador in autumn eating rotten apples from an orchard!). Only after these things consider checking a BG curve: always plot the results on a graph; a list of numbers does not mean anything. Remember that not all owners are happy using the blood sampling kits. Also, that blood glucose monitors do need maintenance and need checking against laboratory results: the AlphaTrak® (Zoetis) may read higher than the real value while BG monitors for humans tend to read low. The MiniMed® continuous BG monitoring system avoids the discomfort and inconvenience of 2-hourly blood sampling, and avoids missing a peak or trough that falls between two measurements; it still needs calibration. Always remember there is wide variation in day-to-day curves even in a diabetic pet with good glycaemic control. The point about the Somogyi effect is that after an initial fall in BG (in response to excess insulin), the BG then goes up and stays high, so it may look like an insulin resistance curve. Some animals are insulin resistant, so if the animal is not stabilising on a 'usual' sufficient dose, then try a higher dose, above 1 IU/kg/injection, even up to 2 IU/kg/injection: but if that doesn't work, then start thinking about other conditions that might be affecting the animal — obesity, infection, concurrent illness, hyperadrenocorticism, exogenous glucocorticoid administration, dioestrus, progestogen administration, acromegaly, hypo- or hyperthyroidism, impaired insulin absorption, excessive insulin antibody formation, phaeocromocytoma, glucagonoma.

UTIs are common and are not always clinically obvious but will affect the diabetes. It is important to do culture and sensitivity, not simply treat for 7 days with an antibiotic.

Before increasing the insulin dose, first check that there are no problems with insulin administration etc. that could explain the failure to stabilise. If the animal stabilises on a high dose, then accept that it needs the higher dose, but if it is failing to stabilise, then think further, look for an underlying aetiology.

Do not miss the simple things that could affect diabetic stability or cause a clinical sign: clinical examination and history are the most important parts of diagnosis. Proper and full physical examination and history should be carried out whenever the animal comes in (if a cat with diabetes is unstable and not eating, consider whether it has a mouth problem before worrying about pancreatitis). The physical examination tells you a lot more than the fructosamine. Body weight and BCS are very important: in diabetic dogs the target is no weight loss, or recovery of lost weight. In overweight cats, 2% weight loss a month is the aim. Unintentional weight loss (or unintentional weight gain) is a warning sign. Try to achieve and maintain ideal BCS.

Discussion

Most topics had already been discussed. It was noted that UTI should be distinguished from subclinical bacteriuria as the latter will not affect DM.

Nursing clinics for diabetic patients — Nicola Ackerman

Not every practice is set up to provide nurse-led clinics, not all practices have veterinary nurses and not all nurses are happy to run such clinics. However, these clinics can be useful. In the practice Nicola works in, any client with a pet who has been diagnosed with anything — renal disease, diabetes, arthritis, obesity, will be offered a nurse appointment. The nurse's job is to help them — to make sure that the client is able to do what they need to do — give pills for example — and to discuss the holistic aspects: can the dog get up stairs? Is a discussion of dog ramps needed?

Owners generally find their pet's diagnosis of DM overwhelming, and they don't always tell the vet that. They don't say that they don't think they will be able to give the injections; they usually just agree to everything. So the nurse's role is to go through everything the owner needs to know, and to confirm everything that the vet has already discussed with them. This includes: do you understand what diabetes is? Do you know what the insulin is for? Most clients nowadays will go home, look up on the internet and come back saying 'I've researched this and found…' — which may be a website saying that you can cure obesity and diabetes using ketogenic diets!

So it is important to go though what diabetes is, what insulin is, what the goals of treatment are (to reduce the visible signs of excessive thirst, frequent/excessive urination, increased hunger, weight loss and lethargy, weakness or fatigue, and to avoid or minimise the potential long-term consequences: cataracts or blindness, UTIs, hepatic lipidosis (fatty liver), ketoacidosis (a potentially dangerous change in the pet's blood pH), neuropathy (weakness of the limbs), nephropathy (kidney disease)); what the signs are — people don't understand that their dog is peeing more because it's drinking more — and why we're looking at water consumption and how it's changing. She explains to the owner what the consquences of DM are and why/how these happen. It is important to cover the development of cataracts, particularly in dogs, and plantigrade stance in cats. Other topics include insulin storage, how to administer insulin, looking after the paraphernalia, how the animal responds to insulin and insulin dose changes.

The nurse is looking to set owner expectations, such as 'the vet will only change the dose once a week or less often, and by X amount, and it might take several months to stabilise you pet and it might not happen.' Feeding is discussed: timing, what to feed; exercise; how to identify hypos, and how to treat them (if they are going to use jam, check they don't use sugar-free…), diabetic diaries and home monitoring are explained. And the nurse shows how to lift the scuff and give the injection. The solvent for Nobivac® vaccines (MSD Animal Health) has the same stopper as the Caninsulin®, so the client can be given solvent vials to take home and practice drawing up.

There is a lot to cover, therefore it is advisable to encourage the owner to bring in everyone who is going to be involved with the care of the pet, and to explain that this is going to take 30–60 minutes and they need to have that time set aside for this. Make sure a consult room has been booked for that hour. While talking to the client, the nurse can write notes, bullet points, into an email for the client and email it to them so they have it immediately, a written record. The nurse becomes the primary point of contact: it is generally easier for a client to get to talk with a particular nurse than with a particular vet.

When the client comes for a vet appointment and prescription, the practice always makes an appointment for the client to return in 3–6 months. The client always sees the nurse first, who takes bloods if the vet wants those, weighs the pet and checks the BCS, also e.g. faecal monitoring, Schirmer tear testing, muscle condition score, history taking. Then the nurse gives all the info to the vet, who can check the information before seeing the client and giving the pet a clinical examination and giving the prescription.

The nurse's job is to aid in compliance, so all clients are given the nurse's working email (set to ping to other clinic nurse if away). The nurse also encourages the use of apps such as the RVC's Pet Diabetes App/MSD Animal Health's Pet Diabetes Tracker App and any helpful websites that can assist the owner. If the diabetic pet is not at an ideal BCS then there are more frequent nurse appointments to help with that.

The nurse is always working to the RCVS Code of Conduct and the Veterinary Surgeon's Act, and working under the direction of a vet. The nurse does not make the decision to change the insulin dose, for example: which can be useful in conversation with the client to encourage them not to tinker: 'I'm not allowed to adjust the insulin dose — it's important that those decisions are made with veterinary involvement.' Good protocols in place in the practice are very helpful.

Discussion

Discussion concentrated on several practical aspects. Make sure the client knows that the nurse cannot make decisions: the vet decides for how long they are happy to issue a prescription and what they want the nurse to do. Nurse appointments are not free to the client but they free up vet time, reducing stress. Vets should make better use of such resources: time spent training nurses is saved in the longer term. Also, nurses are a valuable resource and important part of the vet-owner-nurse team, and should be recognised as such. Guidelines and a checklist support the nurse and help make sure everything is covered in that initial consult; a handout for the client is also important. Check in advance whether the client wants to use an insulin syringe or VetPen® (MSD Animal Health), so the right equipment is booked out, and what times suit the owner best for timing of giving the insulin. The biggest problem in using the pen is people not priming it — not removing the air bubble. Offer 'hand-holding' clinics, in which the client comes in with their pet twice daily until they are happy to continue injecting at home: often only once is needed.

The current datasheet for Caninsulin® states that the vial should be shaken thoroughly before first use. This will not denature the insulin during the stated period after opening (4 weeks in the UK) but temperature extremes, e.g. freezing, will.

Locums need to know how they can make use of the nurse; practice guidelines should be used both to support locums and to avoid a locum trying to 'instantly fix' a diabetic and possibly destabilising what was a stable patient.

Encourage clients to monitor (even e.g. water intake unchanged/increased/decreased). Many clients may not be able to afford to buy an AlphaTrak® but will already have a (human) glucometer at home. So long as you explain and accept the limits, they can still be of some use for at-home BG monitoring if you look at trends and the big picture.

Having everyone in the practice saying the same thing makes a huge difference: consistency of message is important. Having the vet say the pet needs to lose weight can be useful so it is not only the nurse saying this.

Avoid specific weight loss targets — they can be demotivational. Rather use the carrot approach and encourage and congratulate for any pet weight loss as that is very motivational.

Client evenings (no pets) are useful — the support group social aspect makes a huge difference to owners. Clients who have found the diet or the VetPen® useful can really help in encouraging others to try it. CA