COMPLICATIONS AND APPROACH TO ANAESTHESIA FOR OBESE PETS

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Alex Dugdale outlines the rising incidence of obesity in companion animals and details its implications when considering using anaesthesia

COMPANION animal obesity is increasing in parallel with that of human obesity in western societies. Between 20 per cent and 40 per cent of dog and cat populations are now overweight or obese ([Figure 1]; Kopelman, 2000; German, 2006).

Obesity is defined as "excessive body fat content sufficient to cause impairment to health or bodily function" (NIH, 1985), and it is associated with increased morbidity and mortality.

The reduction of life expectancy that accompanies obesity is likely to be because of its medical sequelae, which include type two (insulin-resistant) diabetes mellitus, altered circulating lipid profiles, cardiovascular and respiratory disease, musculoskeletal disease, skin disease, infertility and neoplasia (German, 2006).

Obesity, however, may also be the consequence of other conditions, most notably endocrine disease (Clutton, 1988).

In addition, advancing age tends to reduce metabolic rate and physical activity, which can increase the risk of obesity developing, regardless of whether other medical conditions are present. This article will not discuss the medical consequences or causes of obesity, but it is important that each patient has sufficient investigation before anaesthesia to establish the presence and severity of any concomitant conditions that may additionally affect the course of anaesthesia and surgery.

General and pharmacokinetic complications

• Increased subcutaneous fat may result in poor drug delivery after intended intramuscular (IM) administration. Clinical examination may be hindered; the chest may be more difficult to auscultate and the abdomen more difficult to palpate (Figure 2).

• The low perfusion of adipose tissue may delay absorption of subcutaneously administered drugs.

• Veins and arteries may be more difficult to identify when large amounts of subcutaneous fat are present. Pulses may be more difficult to palpate.

• Anatomical landmarks may be obscured, thus making locoregional anaesthetic/analgesic techniques more challenging. If epidural anaesthesia is to be performed, then doses should be reduced by about a quarter, because of the smaller epidural “space” due to fatty tissue deposition and often engorged epidural blood vessels (because of increased intra-abdominal pressure).

• Intubation may be complicated by the presence of excessive fatty tissues in the oropharyngeal region.

• Surgical access is often more difficult, thus prolonging anaesthetic time, which increases risk and potentially lengthens recovery time.

• Increased body fat is accompanied by a reduction in total body water (on ml/kg basis), therefore, the volume of distribution of water-soluble drugs is reduced. For water-soluble drugs, for example the peripherally acting, non-depolarising, neuromuscular blocking agents, initial loading doses should be calculated according to the patient’s ideal bodyweight. Subsequent doses must be tailored to the patient’s requirements.

• For lipid-soluble drugs, not only is the volume of distribution increased, but their clearance is potentially delayed because they enter a larger mass of adipose tissue whose perfusion is relatively slow. General anaesthetic agents are lipid-soluble (they need to enter the brain to exert their effects). Problems may occur following large or multiple doses of the slowly metabolised thiopental when its cumulation in adipose tissues can delay recovery.

There is still controversy over the best dose for injectable agents. For rapidly acting anaesthetic induction agents, doses scaled according to lean/ ideal body mass may be preferable, because it is the rapid increase in brain concentration that produces the initial effect, the size of the fat sink only acting later to delay elimination.

However, for some of the anaesthetic adjuncts, such as opioids, and especially if administered intramuscularly, it may be preferable to dose according to the patient's actual weight, so that, after redistribution into fat, an effective plasma/ effect site concentration is achieved and maintained.

Nevertheless, if administered intravenously, a more profound effect – and side effects – may be observed initially. During prolonged administration of inhalation agents, which may easily occur in obese patients when surgery is often relatively complicated, the blood solubility of the agents becomes less important than their fat solubility for influencing recovery from anaesthesia.

Halothane is most fat-soluble, sevoflurane is slightly more fatsoluble than isoflurane, and desflurane (and nitrous oxide) are least fat-soluble. The potential for prolonged recoveries is, therefore, greatest with halothane, but also potentially a nuisance with sevoflurane and even isoflurane, although isoflurane tends to be the favourite choice (Adams and Murphy, 2000).

• Increased circulating free fatty acids, triglycerides and cholesterol may compete with acidic anaesthetic agents for protein-binding with albumin, thus increasing free/active drug concentrations. In contrast, the increased alpha-1 acid glycoprotein concentration found in obese states can increase the binding of basic drugs (ketamine, opioids, local anaesthetics), thus reducing their free/active concentrations.

• Obesity is considered to confer an inflammatory state upon the patient (Trayhurn and Wood, 2004). Despite this, patients are generally considered to be at increased risk of wound infection/breakdown, although this may be partly a consequence of increased surgical time and the increased likelihood of hypoxaemia (see respiratory complications).

Fatty infiltration of the liver may accompany obesity, but is also associated with endocrine disorders. Hepatomegaly can increase the diaphragmatic splinting and hypoventilation that accompany obesity. Hepatic function is not normally reduced in uncomplicated obesity, but where other diseases are present and hepatic function is impaired, drug elimination may be delayed (Adams and Murphy, 2000).

• Renal function is not normally impaired in uncomplicated obesity and renal clearance may even be increased because of slightly increased renal blood flow (secondary to increased cardiac output) and glomerular filtration rate (Adams and Murphy, 2000).

• An increased risk of gastrooesophageal reflux may accompany an increased intra-abdominal pressure with increased intra-abdominal fat. Aspiration risk is, therefore, potentially increased.

• Cerebral blood flow is usually unaltered, because of cerebrovascular autoregulation, unless profound cardiovascular impairment is present.

• Increased fat mass may provide thermal insulation and predispose patients to heat stress ([Figure 2]).

Respiratory complications

• Increased fat mass within the oropharyngeal soft tissues may increase the risk of airway obstruction and may complicate endotracheal intubation.

• Increased fat mass both outside and within the chest cavity (and within the abdominal cavity) can reduce chest wall compliance, “splint” the diaphragm, reduce the total lung capacity, reduce functional residual capacity, reduce expiratory reserve volume and increase the work of breathing (Figure 3).

The residual volume usually remains within normal limits. Pulmonary compliance is also reduced and airway resistance may be increased.

Functional residual capacity (FRC) reduction means that patients are less tolerant of periods of apnoea and can desaturate (SpO_2 falls) quickly if airway obstruction occurs, but it also means that the depth of inhalation anaesthesia can change more quickly, so be aware of these patients deepening relatively quickly at the beginning of inhalation anaesthesia.

Pre-oxygenation should increase the oxygen reserve and protect against desaturation during short periods of apnoea, but often increases resorption atelectasis. FRC may also decrease below the closing capacity (the lung volume at which small airways begin to collapse), so that atelectasis may occur even during resting tidal breathing, and is more likely under sedation and anaesthesia (respiratory depression; Figure 3).

Ventilation-perfusion mismatching with subsequent hypoxaemia (and hypercapnia) are potential sequelae of atelectasis. In humans, the alveolar-to-arterial oxygen gradient increases, highlighting worsening hypoxaemia, with increasing obesity (Lotia and Bellamy, 2008).

• Oxygen consumption and carbon dioxide production are increased in obese patients because of the extra tissues that require metabolic support, although normocapnia is usually maintained in the awake animal by increased minute ventilation. The ventilatory reserve is therefore limited.

Most patients, while awake, are said to retain their normal response to hypercapnia and hypoxaemia. In humans, however, the Pickwickian syndrome has been described, whereby the chemoreceptors become progressively desensitised to hypercapnia so that hypoxaemia becomes the most important driver of ventilation.

Cardiovascular complications

• Blood volume is increased in absolute terms, but on a ml/kg basis is reduced.

• Venous and arterial access may be hindered.

• Increased risk of thromboembolic disease.

• The increased fat mass demands, albeit low, perfusion, which necessitates an increase in blood volume and cardiac output, which tend to reduce cardiovascular reserves. Myocardial oxygen requirements are increased; potentially in the face of hypoxaemia. Eventually, obesity-induced cardiomyopathy may result (Adams and Murphy, 2000).

• Although not yet well characterised in animals, in man, hypertension, atherosclerotic vascular disease and left or right-sided cardiac hypertrophy may be present. For example, chronic hypoxaemia leads to chronic hypoxic pulmonary vasoconstriction, which, in turn, leads to cor pulmonale.

• Polycythaemia may accompany chronic hypoxaemia, and the increased blood viscosity increases myocardial work, which may predispose to cardiac dysrhythmias and promote the development of thromboemboli. Fluid loading, in an attempt to reduce the blood viscosity, may be poorly tolerated and precipitate acute congestive cardiac failure.

Approach to the obese patient

• Although pre-operative weight reduction may help to reduce some of the risks and complications associated with anaesthesia and surgery, this should be done under expert direction. A crash diet over the few days before anaesthesia is not recommended. Many obese patients will have a degree of insulin resistance and may be prone to the development of hyperglycaemia.

Pre-operative food withhold of four to six hours is usually recommended, as for any elective procedure, with water being available right up until the time of pre-medication.

• It is of paramount importance that a thorough preoperative evaluation of the patient is performed. This could reveal the presence of other medical conditions that may warrant further work-up and/ or treatment – time permitting. For example, severe electrolyte disturbances may require treatment before anaesthesia is contemplated.

• IM and subcutaneous (SC) drug delivery for pre-medication may be followed by poor absorption. Longer needles can be used to try to ensure IM drug delivery. Alternatively, intravenous (IV) administration may be preferred. Pre-medication should aim to provide “light” sedation (to minimise further respiratory depression) because of the increased risk of respiratory embarrassment. If possible, after premedication, the patient should be closely observed for signs of respiratory compromise.

Pre-oxygenation is usually advised and requires a fairly close-fitting face mask and oxygen delivery (200ml/kg/min to 500ml/kg/min) for a good five minutes by the clock. If pre-medication was “light”, the patient may not tolerate pre-oxygenation by mask without becoming stressed; flow-by oxygen delivery is an alternative strategy, but is usually less effective.

• Induction of anaesthesia may be by IV or inhalation agents, although it may be preferable to ensure rapid control of the airway (orotracheal intubation), because of the possible increased risk of gastro-oesophageal reflux. IV access may be tricky, but catheterisation of a vein is desirable and ultrasound guidance is sometimes required.

It is usually suggested to dose according to lean bodyweight. For people, doses according to a body mass somewhere between ideal and actual have been suggested because, at least for humans, it appears that lean body mass increases with increased fat mass (although this has yet to be proven for animals).

One formula for use in morbidly obese people is given below, but, besides not being tested for animals, we also don't know exactly how to define and calculate ideal bodyweight for our small animal patients:

Bodyweight for human dosing = ideal bodyweight + 1/3 (actual bodyweight – ideal bodyweight).

• Having a variety of endotracheal tube sizes available, a bougie and a laryngoscope, is suggested. Cuffed endotracheal tubes are normally preferred for dogs. In cats, uncuffed tubes may not provide sufficient seal to protect the airway or allow provision of intermittent positive pressure ventilation (IPPV) and laryngeal masks may provide an alternative. Anaesthetic breathing systems should be chosen according to the animal's lean body mass.

• During maintenance of anaesthesia, it may be preferable to provide IPPV (+/- positive end-expiratory pressure [PEEP]) or, alternatively, continuous positive airway pressure (CPAP) if the patient is breathing spontaneously, although the cardiovascular impact of these strategies should not be forgotten (Dugdale, 2007a and 2007b).

• Tidal volumes for IPPV should be based on lean bodyweight to avoid over-inflation of the lungs and possible damage.

Inhalation agents, particularly isoflurane, tend to be favoured over injectable agents as the latter have uncertain pharmacokinetics in obese patients.

Nitrous oxide may perhaps be used as an adjunct, but if patient oxygenation is of concern, then the requirement for high inspired oxygen concentrations limits the usefulness of nitrous oxide. Positive pressure ventilation, however, may impair cardiovascular performance through its mechanical effects (raised intrathoracic pressure) and chemical effects (if hypocapnia is achieved; Dugdale, 2007a and 2007b).

• If the animal is to be positioned in dorsal recumbency, the increased mass of fat within the abdomen may increase pressure on the great vessels (resulting in aorto-caval compression) and potentially reduce venous return and cardiac output. Changes in position should, therefore, be made slowly and the patient may benefit from a slightly head-up tilt.

• Balanced (multimodal) analgesia should be provided where possible; however, there may be contraindications to the use of NSAIDs if concomitant hepatic, renal, gastrointestinal, or some types of endocrine disease are present. Patients may already be receiving NSAIDs as orthopaedic disease is a frequent complication of obesity.

These drugs also compete for protein-binding.

Loco-regional anaesthetic techniques may be more challenging because of obscured landmarks. However, the use of nerve locators and/or ultrasound may help to overcome some of these problems. Epidural doses should be reduced by around a quarter (see previously).

• In view of the cardiovascular complications, intra-operative dysrhythmias may be more likely in obese patients and perhaps precipitated by anaesthetic agents (which promote cardiorespiratory depression).

Intra-operative monitoring should include heart (pulse) rate, ECG, breathing rate, SpO_2, temperature, blood glucose, end tidal CO_2 tension and arterial blood pressure where possible. Blood gas and electrolyte monitoring may also be warranted.

Direct arterial blood pressure may be difficult to monitor as access to superficial arteries may be complicated by overlying fat. However, indirect arterial blood pressure monitoring may also be problematic because of excessive fat surrounding the various appendages that would be used, especially for oscillometric devices reliant on pressure-sensing cuffs. Doppler flow probes may be easier to use alongside a manually-operated sphygmomanometer.

• Intra-operative fluid therapy will depend upon the presentation of each case and should be tailored to individual patient requirements. Electrolyte disturbances may also require addressing. In general, however, for cases in which preoperative hypovolaemia or preoperative congestive cardiac failure are not present, then intra-operative infusion of Hartmann’s solution at approximately 5ml/kg/hr should be safe.

• Neuromuscular blocking agents may be used and are usually dosed according to ideal bodyweight rather than actual bodyweight. If the depolarising agent suxamethonium is used, its effect may be shortened because plasma cholinesterase concentration tends to increase with body mass.

Mechanical ventilation must be available if neuromuscular blocking agents are to be administered.

Monitoring the degree of neuromuscular blockade is highly advised, and adequate reversal of the block must be ensured before anaesthetic administration is discontinued.

• Patient monitoring should be continued into the postoperative recovery period where possible, and even for some time after tracheal extubation until you are sure the patient is breathing adequately. Using drugs to promote a rapid recovery should ensure adequate maintenance of an airway once the trachea has been extubated. Alimentation should be resumed as soon as possible.

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References and further reading

- Clutton R E (1988). The medical implications of canine obesity and their relevance to anaesthesia, *British Veterinary Journal* **144**: 21-28.
- Dugdale A (2007a). The ins and outs of ventilation, 1. Basic principles, *In Practice* **29**: 186-193.
- Dugdale A (2007b). The ins and outs of ventilation, 2. Mechanical ventilators, *In Practice* **29**: 272-282.
- German A J (2006). The growing problem of obesity in dogs and cats, *The Journal of Nutrition* **136**: 1,940S-1,946S.
- Kopelman P G (2000). Obesity as a medical problem, *Nature* **404**: 635-643.
- Lotia S, Bellamy M C (2008). Anaesthesia and morbid obesity, *Continuing Education in Anaesthesia, Critical Care and Pain* **8** (5): 151-156.
- National Institutes of Health (1985). Health implications of obesity: National Institutes of Health Consensus Development Conference Statement, *Annals of Internal Medicine* **103**: 1,073-1,077.
- Trayhurn P and Wood S (2004). Adipokines: inflammation and the pleiotropic role of white adipose tissue, *British Journal of Nutrition* **92**: 347-355.