Proceedings of the 33rd World Small Animal Veterinary Congress

Dublin, Ireland - 2008

Next WSAVA Congress:

Reprinted in IVIS with the permission of the Congress Organizers
Anaesthesia of the Patient with Cardiovascular Disease

Lynne Hughes MVB, DipECVA, DVA, FCARCSI, MRCVS
Veterinary Sciences Centre, UCD School of Agriculture, Food Science and Veterinary Medicine, UCD Belfield, Dublin 4, Ireland

Abbreviations
AS: Aortic stenosis
DCM: Dilated cardiomyopathy
HCM: Hypertrophic cardiomyopathy
MVI: Mitral valve insufficiency
VSD: Ventricular septal defect
SVR: Systemic vascular resistance

Introduction
Patients with cardiovascular system (CVS) disease have decreased reserves and ability to compensate for anaesthetic related alterations in heart rate, preload and afterload, and cardiac output. Cardiac disease itself may alter drug distribution, circulation time, uptake and renal and hepatic clearance. Anaesthesia may result in acidosis (respiratory or metabolic) which will depress myocardial contractility and hypothermia which decreases contractility, causes arrhythmias, increases blood viscosity and shifts oxygen-haemoglobin dissociation curve to left. Anaesthetic and related drugs have many effects on the CVS; whether they are detrimental or not depend on the type of cardiovascular disease which is present. Effects include:
• increasing heart rate and myocardial oxygen demand which can lead to cardiac failure (atropine, glycopyrrolate, ketamine)
• decreasing the heart rate and causing AV block (opioids, α2 agonists)
• decreasing SVR (afterload) ± causing hypotension (ACP, barbiturates, propofol, isoflurane)
• increasing SVR and cardiac work (α2 agonists, phenylephrine, noradrenaline) which can also lead to failure
• depressing myocardial contractility (halothane, thiopentone, propofol infusions)
• increasing myocardial contractility and oxygen demand (ketamine, dobutamine)
• increasing sensitivity to catecholamine induced arrhythmias (halothane, thiopentone, atropine)
• decreasing sensitivity to catecholamine induced arrhythmias (ACP)

Pre-anaesthetic assessment of patients with cardiac disease
• History: exercise tolerance, syncope, presence of cough, dyspnoea
• Physical examination: auscultation, apex beat, pulses, mucous membrane colour and CRT
• Initial treatment if emergency: oxygen, drugs, thoracocentesis etc.
• Tests: cardiac ultrasound, thoracic radiographs, ECG, pre-operative blood tests including arterial blood gas analysis.

Pre-operative planning
1. If not already stabilised, pre-treat the disease process (diuretics, lidocaine, dobutamine, drain effusions etc.) to minimise adverse effects of general anaesthesia.
2. Pre-oxygenate the patient for 5 minutes (using a tight fitting face mask) prior to induction of anaesthesia. This provides a reservoir of oxygen in the lungs, so that the patient does not become hypoxic following induction of anaesthesia
3. Calculate doses of emergency drugs in advance (adrenaline, atropine, lidocaine, dobutamine, phenylephrine, nitroprusside, esmolol)
4. Pre-place at least one IV cannula, unless to do so will cause the patient to struggle excessively. In many cases two IV cannulae are recommended. The goal of pre-operative planning is to anticipate potential problems.

Premedication and sedation
Opioids are seldom contra-indicated, especially if used with anti-cholinergics to prevent bradycardia (e.g. morphine 0.2-0.4mg/kg).
Benzdiazepines (midazolam or diazepam 0.2-0.3 mg/kg) may be combined with the opioid for pre-medication or used as co-induction agent.
Low dose acepromazine (0.01-0.02 mg/kg) may be used with an opioid in patients which will benefit from a slight reduction in afterload (MVI, VSD), but is contra-indicated in animals already receiving vasodilators and those with PDA and A5.
Low dose medetomidine (1-3 µg/kg) may be used where an increase in afterload is beneficial (HCM) but is contraindicated where increased myocardial work or bradycardia are detrimental (DCM, MVI, AV block, PS).
Xylazine is arrhythmogenic and is best avoided.
Atropine (20 µg/kg) or glycopyrrolate (5 µg/kg) should be used judiciously in conjunction with opioids and where it is crucial to prevent bradycardia (AV block, PS, PDA, VSD, MVI, DCM). It is essential that administration does not cause tachycardia which results in reduced diastolic filling and coronary perfusion and increased myocardial work.
All pre-medicated patients should be monitored carefully in case their condition decompensates.

Proceedings of the 33rd World Small Animal Veterinary Congress 2008 - Dublin, Ireland
**Intravenous fluids**
If right heart function is compromised, fluid therapy should be tailored by using central venous pressure measurement. Patients receiving diuretic therapy (e.g. MVI, DCM) may be dehydrated and hypovolaemic when presented for anaesthesia. These animals benefit from rehydration prior to anaesthesia. Adequate preload is essential in patients with PS, pericardial effusions and cardiac tamponade. Volume overload should be avoided in patients with mitral or tricuspid valve incompetence, DCM, HCM VSD and PDA. Opinion differs as to the most appropriate fluid; LRS provides adequate sodium to maintain fluid in the vascular space.

**Induction of anaesthesia**
1. Pre-oxygenation is beneficial
2. Induction should be slow, to-effect and ‘stress-free’ to avoid catecholamine release and consequent arrhythmias. Rapid intubation of the trachea is only necessary if the patient has respiratory disease or a full stomach.
3. Etomidate causes few changes in heart rate or systemic vascular resistance and is suitable for most patients with cardiac disease. This drug is not widely available.
4. Propofol can cause a reduction in systemic vascular resistance which can be beneficial in similar conditions to ACP. Administer slowly ‘to effect’. Infusions cause myocardial depression.
5. Thiopentone often results in tachycardia which is best avoided in most patients with cardiac disease (see above)
6. Diazepam and ketamine is a suitable combination in patients requiring positive inotrope or chronotrope support (mitral or tricuspid valve disease, DCM, AV block, PDA) but is not appropriate for patients with HCM, VSD or AS. Mix equal volumes of the two drugs in the same syringe and administer 1 ml per 10 kg IV slowly
7. An alternative is mask induction with sevoflurane (or isoﬂurane), combined with tiny increments of propofol (<1 mg/kg), to avoid excitement. This technique results in atmospheric pollution.

**Maintenance of anaesthesia**
1. Provision of oxygen is essential to maximise myocardial oxygenation.
2. Halothane is arrhythmogenic, a negative inotrope and is best avoided. These disadvantages outweigh its minimal effect on SVR.
3. Isoﬂurane and sevoflurane are preferred over halothane as they are not arrhythmogenic; however both cause hypotension via peripheral vasodilation and some degree of dose-dependent myocardial depression. These effects can be minimised by co-administering an opioid infusion ± nitrous oxide (see below) ± neuromuscular blockade.
4. Opioid infusion (e.g. Fentanyl 0.1-0.7 µg/kg/min) reduces the requirement for inhalation agent without significant alterations in SVR. Anti-cholinergic premedication may be required to prevent bradycardia. Remifentanil is also suitable.
5. Nitrous oxide does not cause arrhythmias, myocardial depression or significant alterations in SVR. Moreover, it will increase the speed of induction of anaesthesia, decrease the MAC of volatile agents and provide some analgesia. Use of nitrous oxide should be avoided where hypoxaemia or right-to-left shunting is associated with cardiac disease (some VSDs and PDAs)
6. Fluid therapy: see above. Lactated Ringer’s Solution 5-10 ml/kg/h is required in most patients during anaesthesia and recovery
7. Intermittent positive pressure ventilation is beneficial as it maintains normal levels of carbon dioxide and prevents respiratory acidosis. However, large tidal volumes and high airway pressures reduce venous return and cardiac output. It is therefore preferable to increase respiratory rates, reduce pressures and to avoid the use of positive end-expiratory pressure.

**Monitoring of anaesthesia**
Careful ‘minute to minute’ monitoring is necessary to ensure a stable plane of anaesthesia, thus a person dedicated to that task is essential!
Deep anaesthesia will result in significant CVS depression with worsening of cardiac disease. However it is also important to avoid light anaesthesia which results in tachycardia, catecholamine release, increased SVR and arrhythmias. The following parameters should be monitored and recorded: heart rate and rhythm; pulse rate, rhythm and quality; respiratory rate, depth and rhythm; mucous membrane colour; capillary refill time; jaw tone; eye position; and reflexes.

**Useful monitoring equipment**
- Oesophageal stethoscope (heart rate and rhythm, respiratory rate)
- Pulse oximeter (haemoglobin saturation with oxygen and heart rate)
- E.C.G. (heart rate, rhythm and electrical activity)
- Blood pressure (adequacy of tissue perfusion)
- Capnograph (adequacy of respiratory function)

**Analgesia**
Use a ‘multi-modal’ approach i.e. use of analgesic drugs from differing pharmacological groups, which act on different receptors or parts of the pain pathway. Pain increases heart rate, myocardial oxygen demand and...
Anaesthesia/analgesia

Oxygen consumption and increases the risk of myocardial damage and arrhythmias (due to myocardial hypoxia). In surgical patients: use potent opioid analgesics (morphine or fentanyl instead of buprenorphine or butorphanol). NSAIDs may be used in conjunction with opioids unless there are specific contraindications to their use. Additionally, local anaesthetic techniques may be used if applicable to the surgical site (epidural / brachial plexus block), although care must be taken that epidural-induced vasodilation does not aggravate hypotension.

Post-operative care
1. Provide supplemental oxygen in recovery, especially if the patient is shivering, which increases oxygen requirements by up to 400%.
2. Continue analgesia for 48-72 hours
3. Continue fluid therapy until eating and drinking
4. Administer cardiac medication as soon as practical
5. T.L.C. (warm, dry bed, empty bladder etc.)

General principles of safe anaesthesia for the patient with CVS disease
• Pre-treat the disease process prior to anaesthesia if at all possible, e.g. diuretics, ACE inhibitors, pimobendan, digoxin, β blockers. Allow at least one week, and preferably two, to stabilise animals prior to anaesthesia.
• Do not withhold cardiac medication on the day of anaesthesia.
• Research the interactions of medications with anaesthetic drugs e.g. avoid ACP if the patient is receiving other vasodilator drugs, β blockers will mask tachycardia associated with light anaesthesia
• All general anaesthetics have effects on cardiovascular function. Identify in advance the main goals of anaesthetic management for the patient’s condition and use drugs that may help offset the underlying problem: always avoid drugs that will aggravate the disease process.
• Avoid hypoxaemia, hypercarbia and hypothermia during anaesthesia.
• Remember, older animals have decreased cardiovascular reserves, even if they do not appear to have overt disease.

References