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Cardiac Anesthesia

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Introduction

The purpose of general anesthesia is to provide analgesia, unconsciousness, muscle relaxation and suppression of autonomic and somatic reflexes. A patient with cardiac disease may require general anesthesia for angiography or angioplasty or for another reason unrelated to the heart. Safe anesthesia requires careful evaluation of the cardiopulmonary system. A preanesthetic work up should include a complete physical exam, blood work, an electrocardiogram and thoracic radiographs. The hemodynamic changes expected with heart disease need to be reviewed along with the effects of the cardiac drugs used to treat the condition. The potential interaction of these drugs with anesthetic agents also needs to be considered. An arrhythmia needs to be identified and treated before anesthesia. These animals may be a challenge to anesthetize.

Anesthesia for the Cardiac Patient

Before anesthetizing a cardiac patient, it is imperative that the anesthesiologist understand how the anesthetics work and the physiologic changes they cause. Many of these animals will have altered distribution of drugs due to decreased muscle mass and decreased albumin levels. Pulmonary compliance can be reduced due to chronically elevated pulmonary capillary pressure. This may lead to ventilation/perfusion mismatches. Decreased myocardial function can enhance the myocardial effects of anesthesia. The decrease in renal blood flow that accompanies general anesthesia, compounded with cardiac insufficiency, can cause a slowed elimination of many drugs, thus it increases the chance of overdosages.

Anticholinergics (atropine, glycopyrrolate) competitively antagonize postganglionic parasympathetic cholinergic receptors. They are primarily used in conjunction with general anesthesia to prevent salivary secretions and to treat bradycardia due to increased vagal stimulation. Glycopyrrolate does not cross the blood brain barrier and it has fewer cardiovascular effects than atropine. Atropine can cause drowsiness and potentiate CNS depressant drugs' effects. Sinus tachycardia and first and second degree AV block can be seen after administration of either of these drugs. Their routine use as a premedication should be examined because anticholinergics increase myocardial work, myocardial irritation and myocardial oxygen demand. The effects of the phenothiazines (acepromazine and promazine) and the butyrophenones (droperidol, leperone, azaperone) are mediated by antidopaminergic actions and by sympathetic nervous system suppression. The phenothiazines also decrease the seizure threshold. They cause a calming effect but lack analgesic properties. These drugs cause blockade of the sympathetic alpha-adrenergic receptors which causes hypotension with reflex tachycardia. Myocardial contractility is also depressed. Sensitivity to epinephrine-induced arrhythmias is decreased by their administration. In low doses, they are considered a good premedication choice for patients with mitral insufficiency, as they reduce afterload.

Benzodiazepines (diazepam and midazolam) enhance the activity of the central

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nervous system inhibitory neurotransmitters (GABA, glycine) and combine with benzodiazepine receptors in the CNS. These drugs are not used alone as premedications because they produce unreliable effects. Muscle relaxation occurs because of decreased polysynaptic reflex activity. They also have an anticonvulsant effect. Cardiac side effects of diazepam’s propylene glycol carrier are seen with rapid intravenous administration. These effects are hypotension, bradycardia, cardiac arrhythmias and apnea. When given slowly these agents have negligible depressant effects. 

Xylazine blocks the action of the alpha-2 adrenergic receptors, decreasing the release of norepinephrine. Its actions can be reversed by the administration of yohimbine. This drug should be avoided in cardiac patients due to its cardiopulmonary depressant effects. Initially it causes a transient blood pressure increase, but this is followed by a fall in blood pressure. It causes a bradycardia (parasympathetic activity) and it increases cardiac sensitivity to catecholamine induced arrhythmias. Overall, the cardiac output may decrease by as much as 33% due to an increase in peripheral resistance and bradycardia. Administration of glycopyrrolate along with xylazine to counteract the bradycardia does not alleviate the decreased cardiac performance, but in fact worsens it.

Synthetic opioids reversibly combine with specific opiate and nonopiate receptors in the CNS. This class of drugs includes morphine, oxymorphone, meperidine and fentanyl. Butorphanol and pentozocaine have synthetic opioid agonist-antagonist effects. These drugs cause a dose dependent respiratory depression and a bradycardia due to stimulation of medullary vagal nuclei, and are a good choice for a preanesthetic medication as they have little effect on the myocardium. Their respiratory depressive effect must be considered when premedicating an animal with pulmonary dysfunction.

Ultrashort acting barbiturates (thiamyal, thiopental, methohexital) can be used as induction agents. Their mechanism of action is not completely understood, but they cause CNS depression. Cardiac arrhythmias and myocardial depression are seen when the barbiturate is given as a bolus or in large doses. They also cause brief periods of apnea after administration. The cardiac arrhythmias and myocardial depression seen when thiopental is used alone can be decreased by concurrent administration of lidocaine. This is done by starting with the barbiturate and alternating its injection with lidocaine in increments of 4.4 mg/kg until a total dose of 8.8 mg/kg of each drug has been injected intravenously. Do not mix these drugs in the same syringe as an insoluble precipitate will form. This combination has been shown to lower the occurrence of the thiopental-induced arrhythmias by decreasing the amount of the thiopental required and by decreasing the sensitivity of the myocardium to catecholamine-induced arrhythmias.

The dissociative anesthetic agents include ketamine, phencyclidine and tiletamine. The administration of these drugs can cause irregular, apneustic and shallow breathing. An increased heart rate, increased blood pressure and increased cardiac contractility may occur in healthy patients. Ketamine should be used with caution in patients with a diseased myocardium or valvular insufficiency. It has been shown that its direct effect on the heart is to decrease the contractility of the myocardium. This combined with the fact that it increases the pulmonary vascular resistance, pulmonary arterial and capillary wedge pressure, mean arterial pressure and central venous pressure means that ketamine actually increases the workload on a diseased heart. This effect has been demonstrated in patients who have little cardiac reserve and a sympathetic system with limited ability to override the direct depressant effect. Ketamine also has an antiarrhythmic effect on the heart. It is not used alone because of its potential to cause seizures in dogs. Ketamine may be used in combination with other drugs (i.e. midazolam, diazepam, or oxymorphone) to minimize its undesirable effects. And when used this way is a good induction choice for cardiac patients. The combination of tiletamine-zolazepam has similar effects on the cardiovascular system as the ketamine-diazepam combination (heart rate and blood pressure increase).

Mask inhalation induction with isoflurane or halothane should also be considered for these compromised patients, but stress and excitement must be kept to a minimum. The patients should receive preoxygenation prior to induction and should be handled gently.
in order to reduce stress and struggling. An inhalant gas’ ability to produce anesthesia is dependent on alveolar ventilation, blood/gas partition coefficient, cardiac output, and alveolar to mixed venous anesthetic partial pressure difference, thus a left-right cardiac shunt or ventilation/perfusion mismatches will slow the rate of anesthetic induction.

Halothane causes direct depth-related vascular smooth muscle depression and vasodilation which leads to hypotension. It also directly depresses the myocardium decreases sympathetic tone, causing bradycardia at deep planes of anesthesia, and very importantly, it sensitizes the heart to catecholamine-induced arrhythmias.

Methoxyflurane’s cardiac depression is similar to halothane’s except that sensitization to catecholamine-induced arrhythmias is lessened. Methoxyflurane would not be a good choice for mask induction because induction time is long.

Isoflurane causes minimal cardiac depression when compared to the other two inhalants. It still slightly decreases the contractility, but the cardiac output is better maintained at anesthetic levels. It does not sensitize the heart to arrhythmias. Isoflurane is the best inhalant choice for a cardiac patient as it allows rapid and precise regulation of anesthetic length and depth.

Nitrous oxide has very few detrimental cardiovascular effects. When used in combination with another inhalant at 50% N₂O/50% O₂, it will decrease the minimum alveolar concentration by 20-50%. The use of nitrous oxide in cardiac patients must be considered carefully, especially in an animal with pleural effusion or pulmonary edema due to congestive heart failure. The use of nitrous oxide will not only decrease the dose of the anesthetic gas, but it will decrease the oxygen concentration of the inspired air.

A balanced anesthetic protocol is probably the best choice for these patients. An example protocol is oxymorphone and/or fentanyl, low-dose isoflurane, oxygen plus nitrous oxide, and possibly atracurium (neuromuscular blocker). By combining these drugs a smaller dosage of each is used, thus decreasing the adverse side effects seen when each is used separately.

Monitoring

It is imperative that cardiac patients be monitored closely. Their parameters change rapidly and close observation is the only way to catch a significant change before it becomes critical. In addition to monitoring respiration and heart rate, other parameters such as blood pressure, central venous pressure, urinary output, ventilation, core body temperature, and arterial and venous blood gases should be monitored.

Direct blood pressure monitoring provides information about organ perfusion. Capillary refill time and mucous membrane color correspond roughly to capillary perfusion. If a blood pressure monitor is not available, measuring urine output will give some idea of renal perfusion and therefore systemic blood pressure. If mean systemic arterial pressure is <60 mm Hg, urine output will be < 1.0ml/kg/hr. Anuria can occur with normal blood pressure so urine monitoring alone may not always be a truly accurate indication of blood pressure.

The compromised heart can not adjust to hypoxemia. PaO₂ < 60 mmHg causes an increased cardiac work load and thus oxygen demand is increased. Postoperative shivering causes an increase in whole body oxygen usage, thus hypothermia should be avoided. Pulmonary ventilation monitoring via blood gases or a respirometer is a good idea.

Cardiac depression occurs at pH < 7.2, as intracellular acidosis inhibits binding of calcium, decreasing myocardial contractile strength. Venoconstriction can also result from acidemia, which in turn can cause cardiac overload with the diseased heart. Alkalosis causes increased myocardial work and oxygen demand. Blood gas analysis will aid in monitoring the acid-base balance of the patient. Monitoring of an ECG prior to and during anesthesia to detect arrhythmias is important. If an arrhythmia is present, it should be treated as soon as possible to improve cardiac output.

Fluid administration should be monitored carefully to avoid excessive fluids that may increase the cardiac workload. Too few fluids can reduce preload and also cause inadequate volume, resulting in decreased systemic perfusion. Significant blood loss
should be replaced to avoid loss of oxygen carrying capacity and reduced systemic organ perfusion.\(^1\) The type of fluid administered needs careful consideration. If the patient has a history of congestive heart failure or the possibility is good that it may develop (i.e. enlarged heart on radiographs) the use of a fluid without sodium is advisable. Five percent dextrose in water is a good choice.

**Summary**

Anesthesia can be performed safely on the cardiac compromised patient with proper consideration of the disease pathophysiology and the cardiac and anesthetic drugs being used. Monitoring need not be extensive but it should be complete. An example that all small animal practitioners will encounter is the old dog with mild to moderate mitral insufficiency that requires a dental.

First, the physical exam and history must be complete. The cardiac drugs the dog is receiving need to be reviewed along with the pathophysiology of the disease.

Next, a safe anesthetic protocol must be decided on depending on the results of the physical and diagnostic work-up (CBC, serum chemistries, ECG and chest radiographs). If the dog is easily excited, a light premedication with a synthetic opioid should be administered. If the dog is calm enough to mask induce without premedication this can also be considered.

Induction choices include thiopental/ lidocaine, synthetic opioids, midazolam/ ketamine or mask induction with preferably isoflurane. If possible, it is ideal to preoxygenate this animal especially if there is respiratory dysfunction.

The use of isoflurane or isoflurane/ \(\text{NO}_2/\text{O}_2\) is the preferred method for maintaining these animals. Halothane combined with \(\text{NO}_2/\text{O}_2\) could be used if isoflurane is unavailable.

These patients must have closer monitoring than the normal healthy animal. The monitoring need not be invasive but it should be as complete as possible. The heart rate and respiration should be noted. A blood pressure monitor would be ideal but a combination of pulse pressure, mucous membrane color and capillary refill time would suffice. ECG monitoring beginning at induction should also be included. The animal’s body temperature needs to be maintained as close to normal as possible.

Fluids for an animal with congestive heart failure or the potential of should be 5% dextrose/water (avoid fluids containing sodium). If there is no radiographic heart abnormality and no clinical signs of heart failure then the usage of lactated Ringer’s would be adequate. The fluids need to be carefully monitored to avoid administering too much or too little.

All currently prescribed cardiac medications should be continued at their indicated dosages and dosing intervals during the anesthetic episode.

There is nothing extremely complicated to anesthetizing the cardiac patient. If the work-up and physical exam are complete and the disease and drugs have been reviewed, an anesthetic protocol can easily be decided upon and followed.

**References**


