Tech Tip

DON'T GET HYPERTENSIVE OVER HYPOTENSION

20

Blood pressure is the driving force for blood flow (perfusion) through capillaries that supply oxygen to organs and tissue beds of the body. Blood pressure is needed to propel blood through high resistance vascular beds, including those of the brain, heart, lungs and kidneys. Blood pressure values are expressed in millimeters of mercury (mm Hg) and as three measurements: systolic, mean and diastolic. The systolic pressure is the pressure generated when the left ventricle is fully contracted. Diastolic pressure is the pressure measured when the left ventricle relaxes. Mean arterial pressure (MAP) is calculated as one third the systolic pressure plus two thirds the diastolic pressure. Mean blood pressure determines the average rate at which blood flows through the systemic vessels. It is closer to diastolic then systolic because, during each pressure cycle, the pressure usually remains at systolic levels for a shorter time than at diastolic levels. Most times, under anesthesia, a patient's mean pressure is what the anesthetist focuses on. A mean arterial pressure of at least 60 mm Hg is needed to properly perfuse the heart, brain and kidneys.

Mean arterial blood pressures consistently below 60 mm Hg can lead to renal failure, decreased hepatic metabolism of drugs, worsening of hypoxemia, delayed recovery from anesthesia, neuromuscular complications and central nervous system abnormalities, including blindness after anesthesia. Prolonged hypotension (> than 15-30 minutes) can lead to nephron damage. Although the effects may not be immediately apparent since 65-75% of nephrons need to be damaged before renal disease becomes clinically observable, the effects may play a role in the onset of renal disease later in a pet's life. Severe untreated hypotension can lead to cardiac and respiratory arrest. Hypertension, or excessively high blood pressure, can lead to problems as well. Ideally, any animal under anesthesia should have should have regular blood pressure monitoring because most anesthetic drugs affect bloodpressure in some way. Mean arterial blood pressure = cardiac output (CO) x systemic vascular resistance (SVR). Cardiac output is defined as the amount of blood pumped by the heart in a unit period of time. CO = Heart rate (HR) x stroke volume (SV = contractility). Systemic vascular resistance is the amount of resistance to flow through the vessels. Some vessels may be dilated, and therefore allow more flow at less resistance. Constriction of vessels may limit blood flow and require more pressure to get blood through. It's important to know that many of the drugs we use for anesthesia affect one or more of these systems in some way.

Pulse palpation: If no monitor is available, the manual palpation of an arterial pulse can give some indication of the state of the blood pressure. A palpable pulse pressure is the difference between the systolic and diastolic pressures. A difference of at least 30 mm Hg is necessary to palpate a strong pulse. Peripheral pulse palpation sites include the lingual, dorsal metatarsal, carpal, auricular and coccygeal. It is best to monitor the peripheral arteries because these pulses are lost at a much higher mean than the central (femoral) arteries. Potential cardiovascular abnormalities may be detected by regular palpation. Pulses should be assessed for strength, rate, and regularity and palpation should begin prior to induction so that differences in these can be tracked (monitor trends) from the very onset of anesthesia through recovery.

Blanching the mucous membranes with direct pressure should result in a refill time of less than 2 seconds. Delays in refill time can indicate intense vasoconstriction or hypotension.

Oscillometric devices work by picking up pulsation under an occlusion cuff placed over an artery. The cuff is connected to a monitor that can be programmed to measure blood pressure at specific intervals of time. These devices deliver systolic, mean and diastolic readings as well asthe heart rate. Most have alarms that can be set to alert when readings are out of the accepted range. The cuff size should be approximately 40% of the circumference of the limb (or tail) around which it will be placed. Cuffs that are too large will lead to artificially low readings andtoo small a cuff will give false high readings. Ideally, cuffs should be placed on a limb that is close to heart level (the level of the right atrium is the zero mark for blood pressure). Limbs well above the heart may give artificially low readings. Legs hanging well below the heart will give false highs. The cuffs are usually marked with the proper placement over the artery. They must not be applied too tightly as this may occlude flow and cause inaccurate readings as well as swelling distal to the cuff. Poor pulse signals from poor flow (the rear limbs during a severe GDV or large abdominal mass), or any movement of the limb during a reading will interfere with the device and may cause it to fail or deliver an inaccurate reading. These devices do not usually work consistently or at all on very small patients, although there are some newer, veterinary specific monitors out that claim to work accurately on small animals.

Normal systolic blood pressures in the conscious patient are 100-160 mm Hg, normal diastolic pressures are 60-100 mm Hg and normal mean arterial blood pressure ranges are 80-120 mm Hg. Hypotension is classified as MAP of less than 60 mm Hg. It is important to be able to identify the cause of a blood pressure abnormality to know how to begin treatment for it. There are generally three things to consider when looking for causes of hypotension. Look for drugs or physiological/pathological factors that may reduce systemic vascular resistance (SVR), look at heart rate, and look for things that affect stroke volume (preload/contractility). As mentioned earlier, many of the drugs used in anesthesia cause some degree of hypotension, and less often, hypertension. Knowing the side effects of these drugs and how they work will help in determining treatment. Drugs that decrease SVR (and cause vasodilation) in a dose dependent manner include acepromazine, thiobarbiturates, propofol, isoflurane and sevoflurane. Other physiologic factors that may cause a decrease in blood volume or vascular tone include hemorrhage, inadequate volume administration or replacement, dehydration, shock, sepsis, anaphylaxis or severe hypercapnia (high CO2). Patients with acid/base abnormalities should be stabilized prior to anesthesia if possible to help reduce the possibility of hypotension. Drugs that can decrease heart rate include opioids, alpha 2 agonists, and the inhalant drugs isoflurane and sevoflurane. Patients with intracranial disease, hypothermic patients, and extremely fit pets may have low heart rates (bradycardia). Anesthetic drugs affecting the contractility of the heart include the inhalants, thiobarbiturates, propofol, and alpha 2 agonists. The inhalant drugs are potent vasodilators, with up to a 50% reduction in cardiac contractility at surgical planes of anesthesia as well. The other drugs' affect on contractility is more transient and less profound. Alpha 2 agonists and phenylephrine cause vasoconstriction of blood vessels which results in hypertension. The effects of hypertension from the alpha 2 agonists is transient, lasting only a few minutes before the vessels relax and hypotension can result. The dissociative drugs, Ketamine and Telazol have indirect positive effects on the cardiovascular system and thus increase heart rate, but this can cause a reduction in stroke volume. Patient positioning can affect blood pressure. Obese, bloated, or patients with large abdominal masses placed in dorsal recumbency may be hypotensive due to excessive pressure on the caudal vena cava. This pressure may compromise venous return and result in hypotension. The same can happen when positive pressure ventilation is used.

Certain disease states can cause hypertension including pheochromocytomas, pulmonic stenosis, heartworm disease, and hyperthyroidism. Ideally these patients will have their hypertension well controlled before surgery. The exception may be the pheochromocytoma patient

whose hypertension may spike up during surgery when the tumor is manipulated. A nitroprusside CRI may be indicated for these patients. If a patient develops hypertension under anesthesia that is not related to a disease state, the cause is most likely related to inadequate anesthetic depth and/or inadequate analgesic administration. Adjusting anesthetic depth and providing additional pain medications should result in normotension.

Step one in developing a plan for treatment of hypotension is determining the cause. If the patient is otherwise normal and healthy, the anesthetic drugs are most likely the cause of hypotension. The effects of these drugs are dose related and therefore the best first treatment always involves reducing the dose of the drug, or reducing anesthetic depth. Anesthetic protocols that include appropriate analgesics, pre-operatively and peri-operatively will allow lower doses of all anesthetic drugs to be used, lowering the side effects of each drug as well. Any patient anesthetized with inhalant drugs and/or premedicated with acepromazine will have some degree of vasodilation. Intravenous fluid administration of crystalloids at a rate of 10 ml/kg/hr is recommended in any patient under anesthesia to help "fill the space" caused by vasodilation and to replace normal ongoing losses that occur for patients (with normal cardiovascular and renal function, patients with certain cardiac diseases may not be able to "handle" excessive fluid overload) under anesthesia. Fluid therapy is best begun before hypotension exists. For suspected hypovolemia a fluid bolus of "one hour's worth" the patient's maintenance rate may be given (i.e. 35 kg pet = 350 mls bolus, along with maintenance fluids). Reassess following the bolus. If the patient is instrumented with a Doppler monitor you may be able to hear the improvement and "stronger" flow. Blood loss should be replaced with 2-3 times the suspected amount of loss. One ml of blood loss should be replaced with 2-3 mls of crystalloid. Excessive hemorrhage may require replacement with colloids including Hetastarch and blood products.

If blood pressure fails to respond to these therapies, and surgical stimulation does not fix the problem, then pharmacologic intervention may be necessary. Pharmacologic agents stimulate the cardiovascular system through two primary mechanisms. Vasopressor effects increase MAP through changes in heart rate, myocardial contractility or affecting the tone of the vasculature. Inotropic effects increase contractility and cardiac output. The two most common drugs used for this purpose in dogs and cats are dopamine and dobutamine. Less commonly, ephedrine and phenylephrine can be used. In extreme circumstances, epinephrine and norepinephrine may be indicated. Before beginning dopamine or dobutamine therapy it is important to ensure propervascular volume. Side effects of these drugs include tachycardia and possible arrythmias. Tachycardia is more prevalent in hypovolemic patients or with overdose. ECGs should be monitored when beginning therapy. Therapy should be reduced or discontinued at any sign of side effects. The half life of both drugs is relatively short and side effects should diminish with the discontinuation of therapy. These drugs are given as a constant rate infusion with the dose varying from 0.5-20 mcg/kg/min. Infusions should be started slowly and increased to the desired effect while the heart rate and rhythm are monitored closely.

Blood pressure should be routinely measured on any patient undergoing general anesthesia. The best way to prevent hypotension is to detect changes in blood pressure as soon as they begin.

References:

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