HOW LOW CAN YOU GO?
HYPOTENSION AND THE ANESTHETIZED PATIENT.
Donna M. Sisak, CVT, LVT, VTS (Anesthesia/Analgesia)
Seattle Veterinary Specialists
Kirkland, WA
dsisak@ssvvet.com

THE ANESTHETIZED PATIENT

During the anesthetic event, the Veterinary Anesthetist must remain focused with a keen eye (ear, digit) on the patient at all times. As the Maintenance period begins, the Veterinary Anesthetist has two equally important tasks. The first one, maintaining the patient at the appropriate depth for the procedure (avoid too light and too deep). The second one, ensuring that the patient remains metabolically stable while anesthetized by very close monitoring of the patient’s vital signs.

The term “vital sign” refers to the variables that indicate the response of the patient’s homeostatic mechanisms to anesthesia (heart rate, respiratory rate, blood pressure, capillary refill time, and temperature). The patient’s vital signs indicate how well they are maintaining basic circulatory and respiratory function while anesthetized. Each of the vital signs is just as important as the other in maintaining a stable patient with a successful recovery.

With consideration and focus being on blood pressure, the Veterinary Anesthetist regards the monitoring of this vital sign as a very useful tool in the anesthetic event. Blood pressure is relatively easy to measure and can reflect the adequacy of blood circulation throughout the body.

BLOOD PRESSURE

Blood pressure (BP) is the pressure exerted by the circulating blood upon the walls of the vessels and is a key vital sign. The term “blood pressure” usually refers to the arterial pressure of systemic circulation. Mean arterial pressure is the driving force for perfusion (blood flow). Adequate perfusion is necessary to supply oxygen to tissue beds and organs of the body.

Mean arterial pressure (MAP) has been defined as:

\[ \text{MAP} = \text{diastolic pressure} + \frac{1}{3}(\text{systolic pressure} - \text{diastolic pressure}) \]

What determines mean arterial blood pressure?
\[ \text{MAP} = \text{SVR} \times \text{CO} \]

SVR = Systemic Vascular Resistance
CO = Cardiac Output
\[ \text{CO} = \text{HR} \times \text{SV} \]

HR = Heart Rate
SV = Stroke Volume (refers to the amount of blood pumped from one ventricle with each beat)
Blood pressure that is above normal limits is considered **Hypertension**. Blood pressure that is below normal limits is considered **Hypotension**.

### HYPOTENSION

To the Veterinary Anesthetist of small animals, hypotension is defined as a mean arterial pressure less than 60mmHg. A patient experiencing hypotension is at risk for compromised perfusion of peripheral tissues and visceral organs. Clinically significant hypotension can lead to many complications such as: renal failure, hypoxemia, reduced metabolism of drugs, delayed recovery and blindness. Untreated hypotension can eventually lead to respiratory and cardiac arrest. The clinical signs of hypotension are: weak palpable pulse, prolonged capillary refill time (CRT), tachycardia, decreased bleeding at surgical site, systolic pressure less than 70mmHg and mean pressure less than 60mmHg

### CAUSES, CONCERNS, AND CORRECTIONS OF HYPOTENSION

The major players contributing to a hypotension are: hypovolemia, reduced cardiac output, peripheral vasodilation.

The most common cause of hypotension in the anesthetized patient is **hypovolemia**. Extracellular fluid deficits may be due inadequate fluid intake, diarrhea, vomiting, and third space fluid accumulations. Vascular volume deficiencies may be due to whole blood or plasma losses or hypoproteinemia. Administering a bolus of crystalloids, colloids, or blood products (patient specific) is what is indicated in correcting hypotension as a result of hypovolemia.

Another cause of hypotension in the anesthetized patient is reduced **cardiac output (CO)**. Cardiac output is defined as the amount of blood pumped through the heart per minute (ml blood/min). Cardiac output is the product of **HR** and **SV**. Bradycardia (decrease in HR) can contribute to a reduction of CO. Physiologic states and common anesthetics could potentially cause bradycardia such as, drugs (opioids, alpha 2 agonists, inhalants), physiologic bradycardia, hypothermia, cervical or intracranial disease, and depth.

Bradycardia is common in the anesthetized patient. Treatment is not always necessary if blood pressure remains stable. A bradycardic patient that becomes hypotensive warrants treatment. An anticholinergic (Atropine or Glycopyrrolate) is used to treat bradycardia. In concert with administering an anticholinergic the Veterinary Anesthetist should also assess the patient’s body temperature, electrolytes, and acid-base status to confirm bradycardic episode is not secondary to any abnormalities where pharmacologic intervention will not remedy the situation.
Stroke volume is an important determinant of cardiac output. Stroke volume is intrinsically controlled by “preload”. Preload is the degree of stretch of the ventricles prior to contracting. It is the end-diastolic volume at the beginning of systole. Afterload is the ventricular pressure at the end systole. Preload is affected by venous blood pressure and venous return. Venous return is the volume of blood flowing back to the heart. A reduction in venous blood volume from blood loss, dehydration or inadequate fluid replacement will reduce stroke volume resulting in decrease CO. Also vasodilation (as a result of anesthetics) will decrease venous return (and preload). Another common cause of decreased preload is positive pressure ventilation (PPV). When providing PPV to the patient the intra-thoracic pressure may be high enough to compress the vena cava. During inspiration right atrial filling may be reduced which will lead to a lower stroke volume. Other factors that can compress the vena cava resulting in decrease venous return are: tumors, enlarged organs (patient placed in dorsal recumbency), increased intra-abdominal pressure from gastric dilation or volvulus, surgical manipulation.

The treatment to improve preload should first be addressed by eliminating the factors that might mechanically be causing a reduction in preload: high airway pressures (cautious with PPV), intra-abdominal pressure (distension, organ displacement, etc). Initially a bolus of crystalloids can be administered. If this does not improve blood pressure than a bolus of colloids or blood products may be warranted.

Myocardial contractility is the intrinsic ability of the heart to contract independently of preload and afterload. Factors that affect the hearts ability “to pump” and maintain adequate blood pressure are: drugs (Inhalants, Propofol, Alpha-2 agonists), cardiac disease, sepsis, electrolyte imbalances, and profound hypothermia.

If poor contractility is suspected minimizing the amount of inhalant being delivered to the patient should be done immediately. Other anesthetics such as opioids should be administered (either by bolus or constant rate infusion) to the patient to support depth. If this adjustment does not improve the patient’s hypotensive state then vasopressors such as Dopamine or Dobutamine should be considered. These drugs should be used cautiously with intense supervision (appropriate dosing) as they are not benign. Most common side effects noticed with the use of these drugs are tachycardia and arrhythmias.

Another cause of hypotension of the anesthetized patient is peripheral vasodilation. Peripheral vasodilation is the increase of the internal diameter of blood vessels. As vasodilation occurs the lumen of the vessel increases in diameter allowing for less resistance of blood flow. The result being decreased Systemic vascular resistance. Systemic vascular resistance (SVR) is the resistance of blood flow through the vasculature. During anesthesia, the patient will have some degree of reduction of SVR as a result of the anesthetics.
(Inhalants, Acepromazine, Propofol). These drugs are vasodilators and can produce severe hypotension.

A decrease in blood volume and/or vascular tone will lead to decreased SVR. Blood loss, histamine release, dehydration, sepsis, severe hypercapnia, anaphylaxis are all factors that may lead to a decrease in SVR.

The treatment for decreased SVR is a direct approach and conservative. Reducing the amount of inhalant delivered (supporting depth with other anesthetics) and minimizing the amount of peri-operative Propofol delivered along with aggressive administration of intravenous crystalloids (confirmed cardiac function for fluid therapy) is anesthetist's approach. After an allotted amount of time (within 15 minutes) if the conservative approach is unsuccessful, the anesthetist will consider the use of drugs (vasoconstrictors) that will increase vascular tone by directly stimulating the receptors (alpha) in vascular smooth muscle. The administration of these drugs should be carefully monitored as their effect may cause an immediate increase in SVR. Phenylephrine and Ephedrine are vasoconstrictors. Phenylephrine is administered as a CRI, Ephedrine is usually given as a bolus.

CONCLUSION

The Veterinary Anesthetist faces many challenges when anesthetizing a patient. Close monitoring of the patient’s awareness (depth) and vital signs (BP, RR, HR, temperature) and a quick response to any complications that may occur is imperative for a safe recovery.

In particular, the monitoring and maintaining of a patient’s blood pressure can assure the anesthetist of the potential for less complications or compromise of the patient immediately and after the anesthetic event.

The Veterinary Anesthetists response to a hypotensive patient should be without hesitation. A combination of events will need to take place in order to restore the patient’s blood pressure to normal. Initially, the anesthetist should assess the patient for any mechanical problems (proper placement of blood pressure cuff, pop-off value position and oxygen flows, surgical manipulation). In addition, the patient’s depth should be assessed (and supported if need be with other anesthetics) and the inhalant should be decreased to minimize the potential vasodilating effects to the patient. Perfusion (CRT) should be evaluated and an IV crystalloid bolus (dog = 10-100mls/kg, cat = 5-60mls/kg) determined by the magnitude of the hypotension should be started. If hypotension persists, the anesthetist can then administer a colloid bolus (5mls/kg) to maintain colloid oncotic pressure. Blood products may be necessary if hypotension is as a result of hemorrhage. Sympathomimetics (drugs that mimic the effect of transmitter substances of the sympathetic nervous system such as catecholamines, Epinephrine, Norepinephrine, or Dopamine may be needed if fluid therapy does
not improve the patient’s blood pressure. These drugs are administered at precise doses via CRI and the patient should be closely monitored.

**DRUGS AND DOSES**

Dopamine – 5.0 - 10.0 mcg/kg/min

Dobutamine – 5.0 - 15.0 mcg/kg/min

Phenylephrine – 1.0 - 3.0 mcg/kg/min

Ephedrine – 0.1 - 0.5 mg/kg per dose – IV or IM

Awareness, anticipation, preparation and a quick response are critical for a successful anesthetic outcome.

**REFERENCES**

3. Lumb William V., Jones E.Wynn, Lumb and Jones' Veterinary Anesthesia, ed3, Baltimore, 1996, Williams and Wilkins

Additional references available upon request.