

## The Top 5 Anesthetic Complications

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### **COMPLICATION** (medical definition):

An unanticipated problem that arises following, and is a result of, a procedure, treatment, or illness. A complication is so named because it complicates the situation.

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As we prepare for an anesthetic event we respect the knowledge and skill required by the anesthetist to monitor and manage a patient to a successful outcome. The anesthetist should have a general understanding of: the anesthetic agents, methods for delivering (and assessing) the anesthetic agent, and the appropriate action required in the event of an anesthetic-related complication/emergency. Despite thorough patient monitoring/supportive care by an astute anesthetist complications can still occur.

The duration of the event can influence outcome; the longer the patient is anesthetized the greater the chance for a complication. Anesthesia time should be carefully planned out by the clinician (surgeon) and anesthetist to prevent a prolonged anesthetic experience for the patient.

Five complications that commonly occur:

Hypothermia

Abnormal heart rate (arrhythmias)

Hypoventilation

Hypotension

Difficult recovery

### **Hypothermia**

During an anesthetic event the patient's thermoregulatory center is affected by anesthetic (opioids) agents potentiating hypothermia.

Acepromazine, propofol, alfaxalone, and inhalants decrease temperature due to vasodilation – eventually slowing the metabolism.

Additional causes of hypothermia include: endotracheal intubation, clipped fur, open body cavities (chest, abdomen), IV fluids, increased high fresh oxygen flow, and surgical prep solution.

The consequences of hypothermia include: decreased metabolism (decreased anesthetic requirements), decrease immune response, delayed wound healing, and coagulopathies. As hypothermia progresses patients become bradycardic and unresponsive to anticholinergic (atropine, glycopyrrylate) therapy. This bradycardic episode will decrease the patient's cardiac output (CO) resulting in hypotension (decreased tissue perfusion) and ischemia (inadequate blood supply to tissues and vital organs).

Cardiac arrhythmias (atrioventricular block (AV block), ventricular premature contractions (VPC's)) can also occur as the patient's temperature continues to drop.

Hypothermia also results in prolonged recoveries and shivering. Shivering increases the patient's metabolic rate and oxygen consumption – this can compromise organ (kidneys) function.

Treatment begins with prevention. In the premedication (premed) period, pre-warming can help minimize temperature loss. Warm laundry and circulating warm air blanket can be used to “tuck in” the patient providing he/she will tolerate this.

Peri-operatively, warming devices ( circulating warm water and warm air blankets, Hotdog warming device, decreasing oxygen flow rates to minimum oxygen requirement ( oxygen maintain consumption), warm environment, warm IV fluids and prepping solutions, should be instituted as best as possible.

Minimizing anesthesia will also help prevent hypothermia.

### **Abnormal heart rate**

Throughout the anesthetic event the anesthetist closely monitors the patient's cardiovascular status. Mucous membrane (mm) color, capillary refill time (CRT), heart rate/rhythm are usually assessed in 5 minute intervals.

An electrocardiogram (ECG) is used to monitor the heart's electrical activity – helpful in identifying arrhythmias. An esophageal stethoscope is an inexpensive piece of equipment used to obtain heart rate and sounds; this is a very valuable monitoring device for not only hearing heart sounds – also good for airway sounds. The use of a Doppler (blood pressure monitor) can also aide the anesthetist in acquiring the patient's heart rate.

Bradycardia (decreased heart rate) is a very common anesthetic complication. Generally speaking, bradycardia refers to heart rates <50 bpm in large dogs, < 70 bpm in small dogs, and, 100 bpm in cats.

Causes of bradycardia: use of vagotonic drugs (alpha-2 adrenergic agonists or opioids), increased vagal tone (intubation, oculocardic reflex), hyperkalemia (increased potassium), hypothermia, hypoxia (decreased oxygen at the tissue level), and excessive depth.

Treatment begins once the cause for bradycardia is determined.

Vagal induced bradycardia is treated with the use of an anticholinergic (atropine, glycopyrrolate) given IV. Anticholinergics can be used preemptively for patients suspected of having high vagal tone (brachycephalic).

Reflex bradycardia caused by the use of alpha-2 agonists (dexmedetomidine) need not be treated unless hypotension/reduced perfusion occur.

Bradycardia caused by hyperkalemia, hypothermia, or excessive depth it is advised to treat the underlying cause.

## **Hypoventilation**

Hypoventilation (also known as respiratory depression) ( $\text{CO}_2 > 45\text{mmHg}$ ) is inadequate ventilation to perform essential gas exchange. It is insufficient elimination of  $\text{CO}_2$  from the body and a reduction of oxygen delivery to the tissues. The concentration of  $\text{CO}_2$  in the blood stream rises in the circulating blood and produces a state known as hypercapnia (or hypercarbia). This is a common concern in the anesthetized patient as a result of insufficient (spontaneous) ventilation.

Hypoventilation is usually drug induced. Opioids, propofol, alfaxalone and inhalants can result in dose-dependent respiratory depression. Acepromazine and benzodiazepines cause minimal respiratory depression.

Upper airway obstruction (brachycephalic syndrome (BAS), neurologic disease (chemoreceptor's sensitivity to carbon dioxide ( $\text{CO}_2$ )), and impaired respiratory muscle effort (rib fractures, obesity, bandage) are other causes for hypoventilation.

Throughout the anesthetic event the anesthetist maintains and closely monitors the patient's respiratory function. Respiratory rate/rhythm/effort are evaluated at 5 minute intervals. The anesthetist should be comfortable with "hands-on" monitoring – using one's own senses (sight, sound, touch) in determining patient's respiratory status.

Mechanical monitoring by the anesthetist involves the use of equipment that will aide in assessing the patient's ventilation. A combination of devices, pulse oximetry ( $\text{SpO}_2$ ) and capnometry ( $\text{CO}_2$ ) can enhance anesthetic management (respiratory) in the patient. The use of both of these devices in conjunction can provide the anesthetist with valuable information regarding gas exchange at the pulmonary level.

Treatment to correct hypoventilation begins once the cause is detected. Initially anesthetic depth should be assessed to rule out the possibility of the patient being too deep. Intermittent manual or mechanical ventilation may be needed until the patient regains spontaneous ventilation.

Partial or full reversal agents (for opioids) may need to be considered to improve respiratory depression once in recovery.

## Hypotension

Hypotension is defined as mean arterial pressure (MAP) < 60mmHg, a systolic arterial pressure (SAP) < 80mmHg. Hypotension results in decrease perfusion to vital organs (heart, brain, kidneys, etc.). The main “players” contributing to hypotension are: hypovolemia, decreased cardiac output (CO), vasodilation.

A few commonly used anesthetic agents contribute significantly to hypotension. Acepromazine and propofol cause vasodilation. Inhalants cause a dose-dependent decrease in cardiac contractility and systemic vascular resistance. By using the low doses of acepromazine (diluted) in addition to reducing the vaporizer setting, hypotension can be minimized (or improved on).

Other causes of hypotension include: dehydration, blood loss, histamine release, and anaphylaxis.

The clinical signs of hypotension are: weak palpable pulse, prolonged CRT, tachycardia, systolic pressure less than 70mmHg and mean pressure less than 60mmHg.

Correcting fluid deficits either through the administration of crystalloids or colloids (or blood products if hemorrhage) is indicated. Perfusion (CRT) should be evaluated and an IV crystalloid bolus (5 – 10mls/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.

In situations where IV fluid therapy is not sufficient then sympathomimetics (dopamine, dobutamine) or vasopressors (phenylephrine) should be considered.

## Difficult recovery

The recovery period for some patients can be challenging and unpleasant. It is during the recovery period where most deaths occur. Patients in the recovery period should have supervision for at least the first few hours post-extubation.

Two common recovery complications are: **delayed recovery** (>30 mins since termination of anesthesia) and **rapid recovery** with or without pain.

Delayed recovery may be an indication excessive depth or slow elimination (hepatic, renal disease, poor perfusion, etc.) of anesthetic agents. Hypothermia can also cause a delayed recovery.

A prolonged recovery may be an indication of a serious condition that may eventually result in death of the patient.

A slow recovery causes depressed ventilation and slow elimination of inhalant anesthetics; this will further exacerbate hypothermia and slow metabolism (of injectable anesthetics) resulting a slower return to consciousness.

If a slow recovery is a result of hypothermia appropriate warming therapy (warm environment, warm IV fluids, warm water or warm air circulating blankets, and warm laundry) should begin immediately. Pre-warming and minimizing anesthesia time can help prevent this from happening.

If reversible anesthetic agents were used (opioids, alpha-2 agonists, benzodiazepines) reversal agents (antisedan, flumazenil) should definitely be considered to expedite the recovery process (keeping in mind patient comfort).

On the other hand, a **rapid recovery** is also not desirable. Patients waking up too quickly can be very distressing to all involved. These patients are in danger of injuring themselves (and others).

Ideally, all patients should be placed in a warm, quiet environment prior to extubation to minimize as much post-anesthetic excitement as possible.

During a “stormy”, rapid recovery it will be important to determine what may have caused this inappropriate “wake-up”. Is it a result of pain or dysphoria, anxiety/agitation for anesthetic agents? This can be a very challenging time not only for the patient but also for the anesthetist/recovery nurse. It may require a multi-step process to get these patients “settled”.

Various anesthetics (dissociatives, tranquilizers, opioids) can cause unwanted behavior changes. If dysphoria is suspected as a result of opioid use, a partial reversal may be required.

Butorphanol 0.05mg/kg IV can be administered to take away some of the unwanted behavior.

A low tranquilizer may be beneficial for mild sedation and calming. Acepromazine 0.01mg/kg IV or dexmedetomidine 0.002 mg/kg IV can be used.

If pain is the cause of a rough recovery, additional analgesia should be administered.

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Additional references available upon request