# Review On Accident And Emergency Of Anesthesia And Recovery Measures

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**Abstract:** General anaesthesia entails keeping the patient unconscious, providing adequate analgesia, and relaxing the patient's muscles to facilitate surgery. Hence safeguarding the patient's airway from the mouth to the lungs and maintaining adequate ventilation is a major component of a general anaesthetic procedure. Safe and effective anesthesia of animals relies on preanesthetic patient assessment and preparation. Patients should be premedicated with drugs that provide sedation and analgesia prior to anesthetic induction with drugs. Emergency drugs and equipment, as well as an action plan for their use, should be available throughout the perianesthetic period. Additionally, intravenous access and crystalloid or colloids are administered to maintain circulating blood volume. Someone trained in the detection of recovery abnormalities should monitor patients throughout recovery. Most anesthetic problems can be avoided or minimized by proper pre-anesthetic patient evaluation, care in administering anesthetic agents, and frequent patient monitoring during the anesthetic period. Problems usually result from equipment failure or are respiratory or cardiovascular in nature. When there is accident and emergency of anesthesia, hypotension, hypothermia, abnormal heart rate, hypoventilation and cardiopulmonary arrest will be resulted. So, the presence of an appropriately trained and experienced anaesthetist is the main determinant of patient safety during anaesthesia.

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## 1. Introduction

General anaesthesia entails keeping the patient unconscious, providing adequate analgesia, and relaxing the patient's muscles to facilitate surgery. All these processes may, to a greater or lesser degree depending on the depth of anaesthesia, deprive patients of their respiratory reflexes. Hence safeguarding the patient's airway from the mouth to the lungs and maintaining adequate ventilation is a major component of a general anaesthetic procedure (Anaesth, 2004).

Most anesthetic problems can be avoided or minimized by proper pre-anesthetic patient evaluation, care in administering anesthetic agents, and frequent patient monitoring during the anesthetic period. Problems usually result from equipment failure or are respiratory or cardiovascular in nature. There are several ways of approaching anesthetic difficulties; the method chosen should be both scientifically sound and workable in your practice (Ba & Anesth, 2010). Prior to receiving an anesthetic agent each patient should be evaluated for at least respiratory and cardiovascular function. A more complete examination is certainly advisable. It is often helpful to categorize patients according to physical status and select anesthetic techniques, agents, and dosage emergency cases (Bednarski *et al.*, 2011).

Proper pre-anesthetic medication and preparation (withholding food and water, etc.) and the careful measuring and administration of injectable drugs slowly and to effect will aid in preventing anesthetic emergencies. Also beneficial is regular patient monitoring during the anesthetic and surgical period. Parameters to be monitored may include heart and respiratory rates, the depth of respiration and strength of the pulse (femoral pulse, etc.), capillary refill (recovery time following blanching of mucous membranes due to pressure) and the usual reflexes (Best, 2008).

The presence of an appropriately trained and experienced anaesthetist is the main determinant of patient safety during anaesthesia. However, human error is inevitable, and many studies of critical incidents and mortality associated with anaesthesia have shown that adverse incidents and accidents are frequently attributable, at least in part, to error by anaesthetists (Peter, 2011).

Monitoring will not prevent all adverse incidents or accidents in the perioperative period. However, there is substantial evidence that it reduces the risks of incidents and accidents both by detecting the

consequences of errors, and by giving early warning that the condition of a patient is deteriorating for some other reason (Booth *et al.*, 2013).

The introduction of routine monitoring in anaesthesia coincided with numerous improvements in clinical facilities, training and other factors likely to affect patient outcomes. The progressive reduction in anaesthesia-related morbidity and mortality is therefore linked to instrumental monitoring by association rather than proof from prospective randomised trials (AAG, 2007).

Anaesthetics are inherently unsafe as all drugs affect the cardiovascular and/or respiratory systems. As a result every anaesthetic requires careful monitoring to ensure risks are kept to a minimum, harmful trends are recognised and to allow time for appropriate action. The anaesthetist is the best monitoring tool, while good monitoring technology is available and useful, it should not be a substitute for observation skills and clinical judgement (AAG, 2007).

Monitoring is continual and should be recorded on a chart to show developing trends but also as a medical and legal document. Therefore knowing what the normal parameters are for your patient and recording them at the start is essential (Clancy & Nolan, 2002). Therefore, the objectives of this review are:

- To high light monitoring before and during anaesthesia with recovery measure
- To address accident and emergency of anesthesia with recovery measure

## 2. Literature Review

### 2.1. Monitoring before and during anaesthesia with recovery measure

2.1.1. Pre-anaesthetic Checks

Assessment prior to any sedation or general anaesthesia is a fundamental part of making the anaesthesia process as safe as possible. This should include, but not be limited to, auscultating the patient's chest. Listening to the heart and respiratory sounds with a stethoscope by both veterinary surgeon and nurse anaesthetist is important. Many patients will not have cardiac or respiratory disease but being comfortable with 'normal' chest sounds will enable the anaesthetist to more readily identify abnormal sounds and potential problems. Pulses should be felt to ensure they are of a good quality and there is no deficit between the pulse and heartbeat. It is important to take a temperature as well. Knowing what these parameters are normally for the patient will allow the anaesthetist to identify changes and assist them in making informed decisions during the anaesthetic and recovery (Cummings & Wetmore, 2016).

As well as a physical examination, a thorough history should be taken. If a patient has received trauma such as being in a road traffic accident, this can have effects on the heart and cause arrhythmias and/or complexes which become more apparent once the patient is anaesthetised. It is therefore crucial to identify physiological, pathological and drug- related factors before inducing general anaesthesia (Deyoung & Victoria, 1974).

It can be helpful to do pre-anaesthetic blood tests. This may not be necessary in young, healthy routine cases but in diseased patients or those presenting as emergency cases it is crucial. Routine packed cell volume (PCV), biochemistry and haematology blood tests can provide invaluable information which may change the anaesthetic and/or fluid therapy protocol (Gornall *et al*., 2017).

2.1.2. The anaesthetist's presence during anaesthesia

An anaesthetist of appropriate experience must be present throughout general anaesthesia, including any period of cardiopulmonary bypass. Using clinical skills and monitoring equipment, the anaesthetist must care for the patient continuously. The same standards must apply when an anaesthetist is responsible for a local/regional anaesthetic or sedative technique for an operative procedure (Gray & Morris, 2013).

Accurate records of the measurements provided by monitors must be kept. It has become accepted that core data (heart rate, BP and peripheral oxygen saturation) should be recorded at intervals no longer than every five minutes, and more frequently if the patient is clinically unstable. It is recognised that contemporaneous records may be difficult to keep in emergency circumstances.

Local circumstances may dictate that handing over of responsibility for patient care under anaesthetic may be necessary. If so, hand-over time must be sufficient to apprise the incoming anaesthetist of all information concerning the patient's anaesthesia and the time and details must be noted in the anaesthetic record (Haller *et al.*, 2011).

Very occasionally, an anaesthetist working single-handedly may be called upon to perform a brief life-saving procedure nearby. Leaving an anaesthetised patient in these circumstances is a matter for individual judgement. If this should prove necessary, the surgeon must stop operating until the anaesthetist returns. Observation of the patient and monitoring devices must be continued by a trained anaesthetic assistant (Misra *et al*., 2016).

2.1.3. Monitoring the anaesthetic equipment

It is the responsibility of the anaesthetist to check all equipment before use as recommended in Checking Anaesthetic Equipment. Anaesthetists must ensure that they are familiar with all equipment that they intend to

use and that they have followed any specific checking procedure recommended by individual manufacturers. More complex equipment will require more formal induction and training in its use. The following monitoring devices are essential to the safe conduct of anaesthesia (Scale, 2015).

If it is necessary to continue anaesthesia without a particular device, the anaesthetist must clearly record the reasons for this in the anaesthetic record. Oxygen Supply: The use of an oxygen analyser with an audible alarm is essential during anaesthesia. It must be placed in such a position that the composition of the gas mixture delivered to the patient is monitored continuously. The positioning of the sampling port will depend on the breathing system in use (Smith & Mishra, 2017).

Breathing Systems: During spontaneous ventilation, observation of the reservoir bag may reveal a leak, disconnection, high pressure or abnormalities of ventilation. Carbon dioxide concentration monitoring will detect most of these problems. Capnography is therefore an essential part of routine monitoring during anaesthesia (Walker & Wilson, 1995).

Vapour Analyser: The use of a vapour analyser is essential during anaesthesia whenever a volatile anaesthetic agent is in use (Smith & Mishra, 2017).

Infusion Devices: When any component of anaesthesia (hypnotic, analgesic, muscle relaxant) is administered by infusion, the infusion device unit must be checked before use. Alarm settings and infusion limits must be verified and set to appropriate levels before commencing anaesthesia. It is essential to verify that these drugs are delivered to the patient. The infusion site should be secure and preferably visible(Warren & Jakobsson, 2014).

Alarms: Anaesthetists must ensure that all alarms are set at appropriate values. The default alarm settings incorporated by the manufacturer are often inappropriate and during the checking procedure the anaesthetist must review and reset the upper and lower limits as necessary. Audible alarms must be enabled when anaesthesia commences(Wig *et al*., 2008).

When intermittent positive pressure ventilation is used during anaesthesia, airway pressure alarms must also be used to detect excessive pressure within the airway and also to give warning of disconnection or leaks. The upper and lower alarm limits must be reviewed and set appropriately before anaesthesia commences (Zellem *et al.*, 2015).

2.1.3.1. Pulse oximeter monitoring device

Pulse oximeter was developed in early 1970s by Aoyagi in Japan. A pulse oximeter measures the amount of oxygen that is bound to hemoglobin and is reported as a percentage of saturation (SpO2). Since hemoglobin with oxygen and hemoglobin without oxygen absorb red and infrared light wavelengths differently, the percentage of one form versus the other can be measured. Hemoglobin absorbs more visible red light, which is why it appears blue. Complex computer programs help identify pulsatile movement (to differentiate from a vein) and calculate the percentage of saturation. In room air (21% oxygen), most mammals have a SpO2 between 95% and 100%. It is the greatest advance in the patient monitoring. Its' documented benefit in improved patient safety led to the widespread use of this technology in anaesthesia. It gives a value of oxygen saturation of the haemoglobin in the arterial blood. It is a simple, reliable and continuous noninvasive method of detecting hypoxaemia (Zeng *et al*, 2016).

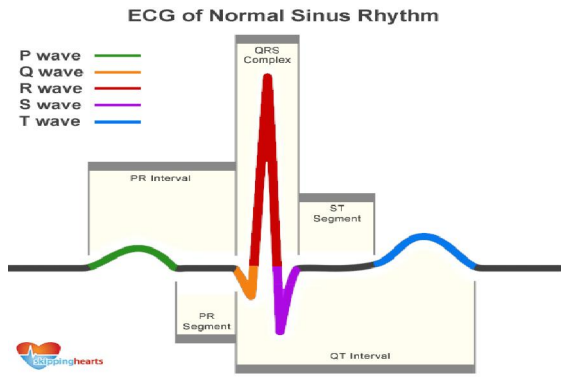
Pulse oximeters are valuable because they provide information on whether the patient has access to oxygen (eg, oxygen tank turned on), whether the patient is breathing, whether oxygen is exchanging in the alveoli, and whether CO is adequate for oxygen to reach the peripheral circulation (ie, the generation of a pulse at a peripheral site). The number generated by a pulse oximeter does not provide information about whether the amount of hemoglobin is adequate to support the patient but whether hemoglobin has oxygen attached to it(Anaesth, 2004).


Figure1: Pulse oximeter monitoring device (Anaesth, 2004)

2.1.3.2. Electrocardiogram (ECG)

The ECG is a recording of the electrical potentials generated by myocardial cells. The potentials produced are small (low voltage), so using conductive gel lowers the skin's electrical resistance, but artifacts and a poor signal can still be a problem. A lead records the electrical signals of the heart from electrodes that are placed at specific points on the patient's body. When a depolarization wavefront moves toward a positive electrode, it creates a positive deflection on the ECG; when a depolarization wavefront moves away from a positive electrode, it creates a negative deflection (Bednarski *et al.*, 2011).

ECGs are useful to assess the rate, rhythm, and conduction patterns of the heart. Each complex can also be inspected to look at individual waves (P, QRS, and T) and to see whether the association is normal and that the waves are a normal size. Abnormal waves can also be identified, which can aid in clinical diagnoses (eg, premature ventricular contractions, hyperkalemia, or ST-segment depression) (Best, 2008).



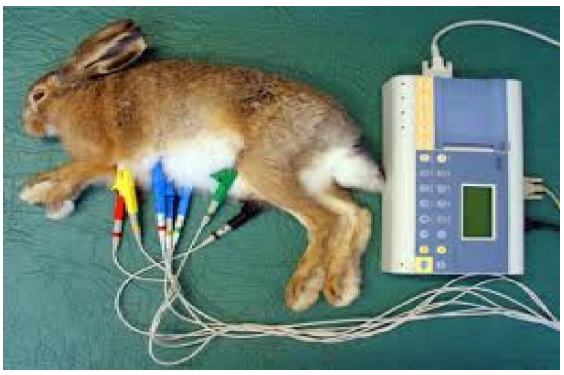



Figure2: ECG of normal sinus rhythm (Booth *et al.*, 2013)

Generation of ECG waves does not give any information about CO. Normal rhythms may be associated with poor CO, and abnormal rhythms (eg, atrial fibrillation) may be associated with adequate CO. Many ECG monitors autocalibrate the wave size; therefore, wave size may be distorted and misleading. Pulseless electrical activity (previously called electromechanical dissociation) can produce a fairly normal-looking ECG rhythm in the absence of a heartbeat or circulation. The decision to treat arrhythmia should be based on the physiologic effect of the arrhythmia and the potential to degenerate into a lethal rhythm (Booth *et al.*, 2013).

2.1.3.3. Blood pressure

Blood pressure is an indirect measure of blood flow and function of circulatory system. It can be measured non-invasively using a cuff and manometer or automated oscillometric method and invasively by placing a catheter in the peripheral artery. Blood pressure is often used as an indirect measure of CO and organ perfusion. Clinically, it can be measured in three ways: Doppler, oscillometric, and direct (invasive) (AAG, 2007).

Doppler. Use the Doppler probe to find an audible peripheral pulse, inflate a sphygmomanometer cuff to occlude flow to that vessel, and slowly deflate the cuff until the audible pulse is heard again. The pressure at which the pulse resumes is the systolic pressure. There is some evidence that in cats, blood pressure measured this way is somewhere between mean and systolic pressure (Clancy & Nolan, 2002).

Oscillometric. Automated blood pressure monitors use a cuff that measures changes in oscillations. The cuff is inflated until the pulse is occluded (no oscillations) and is slowly deflated until oscillations resume (ie, systolic pressure). The point of maximum oscillations is the mean blood pressure, and the diastolic pressure is usually calculated from the other two values. Anything that can change oscillations (eg, shivering) can negatively affect accuracy (Cummings & Wetmore, 2016).

For invasive (direct) blood pressure: A catheter is placed in a peripheral artery and connected to a pressure transducer. The transducer can then generate an arterial wave (graphically) and measure systolic, mean, and diastolic pressure (Deyoung & Victoria, 1974).

Depending on the method, at a minimum blood pressure monitoring will determine systolic blood pressure and often a mean and diastolic blood pressure. Although somewhat oversimplified, systolic pressure is most reflective of CO and diastolic pressure is most reflective of vessel tone. Mean blood pressure is the best indicator of organ (especially renal) perfusion. Higher blood pressure is usually associated with increased CO and increased perfusion; however, that does not occur with increases in systemic vascular resistance (Cummings & Wetmore, 2016).

Figure3: Blood pressure monitoring device (Cummings & Wetmore, 2016)

2.1.3.4. Capnograph

A capnograph (or capnogram) is an indirect measure of ventilation. It measures the amount of carbon dioxide in the breath at the end of expiration. Since carbon dioxide easily moves across the capillaries and alveoli, the assumption is made that the amount of carbon dioxide at the end of the breath (end-tidal volume) is equivalent to the carbon dioxide in the blood. In healthy, awake animals, the difference between the arterial and end-tidal measurements is indeed close (a difference of about 5 mm Hg). Therefore, capnography allows us to estimate ventilation on the basis of the amount of carbon dioxide in the breath. This amount of carbon dioxide is measured by comparing with a reference gas for absorption of infrared light. By plotting the amount of carbon dioxide exhaled over time, a capnograph can be generated (a graphic representation). The size, upper and lower limits, and shape of the capnogram is quite useful in clinical diagnoses of both respiratory and mechanical problems (Deyoung & Victoria, 1974).

Machines that just give an ETCO2 number provide information about ventilation and indirectly CO. If CO is insufficient to bring carbon dioxide to the lungs, then the amount exhaled (even with normal ventilation) will decrease. Machines with awave form readout (capnograph) provide a great deal of information about the patient. Inspection of the waveform can give information about the airways (eg, asthma), whether there is a leak in the system (machine or endotracheal tube cuff), and whether there is rebreathing of carbon dioxide (eg, carbon dioxide absorbant has expired, or one-way valves are not functioning). Capnographs are useful in cases of cardiac arrest, as they can indicate successful cardiopulmonary resuscitation as well as the return of spontaneous circulation (Gornall *et al*., 2017).

Capnography is based on the idea that the end-tidal carbon dioxide concentration reflects the arterial carbon dioxide concentration (PaCO2). Although this is true in healthy, awake patients, the difference between the ETCO2 and PaCO2 can increase with disease (eg, pulmonary thromboembolism), anesthesia, or positioning. The only true way to know arterial carbon dioxide is tomeasure blood gas. Hypoventilation should be treated if carbon dioxide is greater than 60 mm Hg or if the patient is acidemic. Anesthetic depth should be assessed. If needed, ventilation should be assisted or controlled until anesthesia is discontinued (Gray & Morris, 2013).

Figure4: Capnograph monitoring device (Gray & Morris, 2013)

2.1.4. Monitoring the patient

During anaesthesia, the patient's physiological state and depth of anaesthesia need continual assessment. Monitoring devices supplement clinical observation in order to achieve this. Appropriate clinical observations may include mucosal colour, pupil size, response to surgical stimuli and movements of the chest wall and/or the reservoir bag. The anaesthetist should undertake palpation of the pulse, auscultation of breath sounds and, where appropriate, measurement of urine output and blood loss. A stethoscope must always be available *(Haller et al.*, 2011).

The anaesthetic record is not just a legal document; it contains vital information about the anaesthetic and reflects the level of monitoring. It displays trends and patterns which may not have otherwise been easily identified. This could be the steady increase or decrease of heart rate or blood pressure, where between each measurement there is not a significant difference but over a period of time a greater than expected change is revealed indicating there may be need for intervention. Monitoring can be continuous or intermittent. Intermittent include physical, manual checks of the patient and should be performed every five minutes. Continuous monitoring comes from monitoring equipment which will allow the user to instantly notice any changes *(Misra et al.*, 2016).

Monitoring also allows for the assessment of anaesthetic depth. A sudden increase in depth is a significant warning sign that problems are on the horizon and action should be taken swiftly to address this. Reflexes provide invaluable information on depth of anaesthesia and can help the anaesthetist determine if the patient is painful or inadequately anaesthetised. A good analgesia protocol will allow for the anaesthetic maintenance agent to be kept to a minimum. This can be crucial in critical cases. Most anaesthetic related emergencies will be respiratory or cardiovascular thus making close monitoring and minimal depression of these systems important (Scale, 2015).

**2.2. Accident and emergency of anesthesia with recovery measure**

2.2.1. Common causes of anesthetic complications

General anesthesia poses minimal risk to most patients when performed by a capable anesthetist using appropriate protocols and proper monitoring. However, it is vitally important that the anesthetist remembers that every anesthetic procedure has the potential to cause the death of the animal. In spite of significant advancements in pharmacology & technology, the fundamentals of good patient monitoring and support of organ function are key to minimizing anesthetic risk and assuring a good outcome. Similarly, while knowledge of appropriate responses to an anesthetic emergency is essential, it is even more important to understand why emergencies arise and how they may be prevented (Zellem *et al.*, 2015). Common causes of anesthetic complications include:

Human error: Failure to obtain and interpret an adequate history or physical exam; lack of familiarity with the anesthetic machine or agents being used; incorrect drug administration (incorrect drug, dosage, route or concentration) and failure to recognize and respond to early signs of patient difficulty (Walker & Wilson, 1995).

Equipment failure or misuse: Carbon dioxide absorber exhaustion; empty oxygen tank; misassembly of the anesthetic machine or breathing circuit; endotracheal tube problems; vaporizer problems and pop-off valve problems (Anaesth, 2004).

Adverse effects of anesthetic agents: Every agent has benefits and contraindications associated with its use. Reducing the potential for adverse effects depends on several factors: Assessment of the patient and any potential risk factors; familiarity with side effects and contraindications of different agents and appropriate protocol choice, often including multi-drug use to achieve balanced anesthesia (Ba & Anesth, 2010).

Patient related factors include: Geriatric patients; pediatric patients; brachycephalic dogs/cats; trauma patients; systemic disease (Cardiovascular, respiratory, hepatic, or renal disease) and general poor condition (Booth *et al.,* 2013).

Both human error and equipment problems are generally preventable complications. Proper training and attention will prevent these situations from arising. If at any time you are uncertain about an animal's status, proper equipment use or protocol, do not hesitate to ask for assistance. Patient related complications can often be prevented by identifying potential risk factors and modifying the anesthetic plan to address the patient's special needs. Any risk factors noted during the physical exam should be noted in the medical record and brought to the attention of the veterinarian or technician in charge of anesthesia prior to the animal being medicated (AAG, 2007).

2.2.2. Responding to problems during anesthesia

Excessive anesthetic depth: Signs include: RR < 8 bpm and/or shallow respiration; mucous membranes pale or cyanotic; CRT > 2 seconds; HR < 60 bpm in a dog or 100 bpm in a cat; hypothermia; flaccid muscle tone. These signs must be interpreted in light of all available information. Excessive anesthetic depth is

usually a result of a vaporizer setting or drug dose that is too high for the patient. Occasionally the animal may have a pre-existing condition that increases their susceptibility to anesthetic overdose. If you are concerned that your patient is too deeply anesthetized, turn the vaporizer setting down or completely off and alert a supervisor (Booth et al., 2013).

Pale mucous membranes: May result from preexisting anemia, blood loss, anesthetic agents which result in vasodilation and hypotension, hypothermia or pain. Assess anesthetic depth and other vital signs and alert a supervising veterinarian (AAG, 2007).

Prolonged capillary refill time (> 2 seconds): Suggests that blood pressure is inadequate to perfuse peripheral tissues. Hypotension is one of the most common anesthetic complications and should be suspected in any animal with a prolonged CRT. Pulse and blood pressure should be evaluated. A systolic BP < 80 mmHg indicates hypotension and poor perfusion. If pulse or blood pressure is abnormal, the anesthetist should alert a supervisor and closely observe the animal for other signs of shock (Zeng *et al.*, 2016).

Dyspnea and/or cyanosis: Dyspnea indicates an inability to obtain sufficient oxygen using normal respiratory effort. Cyanosis indicates inadequate tissue oxygenation. Any patient showing signs of dyspnea or cyanosis should be brought to the attention of a supervisor immediately. The most common causes of respiratory distress during anesthesia include: Equipment problems (empty oxygen tank, flowmeter turned off, damaged circuit); airway obstruction (ET tube blockage, laryngospasm, aspiration) or respiratory; disease (pleural effusion, pulmonary edema, diaphragmatic hernia, etc); excessive anesthetic depth such that vital functions are compromised. Quickly evaluate other vital signs and anesthetic depth and equipment setup. Once oxygen delivery to the patient and patent airway has been confirmed, turn the vaporizer off and ventilate with 100% oxygen until mucous membrane color and SpO2 readings return to normal Monitor closely during resuscitative efforts to ensure cardiac arrest does not occur (Ba & Anesth, 2010).

Aspiration Pneumonia: Patients at risk for aspiration pneumonia are those that have esophageal (megaesophagus) or gastric fluid accumulation, and then either regurgitate or vomit during recovery. When this problem is anticipated, the esophagus and stomach should be suctioned to prevent aspiration. If the animal is in lateral recumbency and begins to vomit, lower the head and neck and hold the mouth open (don't get bitten). Once the vomiting has passed, assess the mucous membrane color, respiratory rate, and breath sounds, suction and or swab the oral cavity (again, don't get bitten) (AAG, 2007).

Hyperthermia: Hyperthermia may be a result of rough recovery, ketamine administration in dogs or myelography. Placing the patient on a cage floor, wetting with tepid water or directing fans at the patient are all options for correcting hyperthermia. Tranquilization may be helpful in patients that are agitated. Because severe hyperthermia can result in increased oxygen consumption, oxygen should be administered. Crystalloids help to improve circulating blood volume and cool the patient (Ba & Anesth, 2010).

Hemorrhage**:** The surgical incision should be monitored during recovery. Excessive bleeding at the surgical site, increase in abdominal girth along with clinical signs suggestive of hypovolemia (pale mucous membranes, prolonged refill time, tachycardia, and poor pulse quality) could be indicative of internal bleeding. The causes for bleeding could be due to a slipped ligature, bleeding from small arteries that were not bleeding during closure or a coagulation disorder. Direct pressure should be applied and the doctor informed. The clinician may elect to perform an ultrasound and or perform an abdominocentesis or thoracentesis. Therapy may consist of continued direct pressure, fluid resuscitation and or surgical reexploration (Zeng et al., 2016).

2.2.2.1. Hypotension

The minimum acceptable mean arterial pressure for anesthetized small animals is 60 mm Hg. Blood pressure monitoring can be obtained indirectly with the use of Doppler and oscillometric devices or directly from a cannulated artery. Some anesthetic drugs contribute significantly to hypotension. Inhalants cause a dose-dependent decrease in cardiac contractility and systemic vascular resistance. Acepromazine and propofol cause vasodilation. Using the lowest effective dose of an injectable agent and/or reducing the vaporizer setting will be the most beneficial in minimizing hypotension during the anesthetic event (Zellem et al., 2015).

Alternative causes of hypotension that should be considered include blood loss, dehydration with inadequate volume replacement, other drugs (eg, angiotensin converting enzyme inhibitor), anaphylaxis, and histamine release. Therapies to address and respond to hypotension should incorporate a reduction in anesthetic depth when possible and/or crystalloid or colloid bolus; when refractory hypotension exists, intervention with vasopressors or positive inotropes should be considered (Scale, 2015).

2.2.2.2. Hypothermia

A drop in temperature occurs during anesthesia as normal thermoregulation is disrupted and heat productions reduced. Direct anesthetic causes of hypothermia include α-2 adrenergic agonists via action at the α-2C receptor and opioids via action at opioid

receptors in the hypothalamic thermoregulatory center. Acepromazine, inhalants, and induction agents such as propofol and alfaxalone decrease temperature because of vasodilation and/or as a result of slowing metabolism. Additional causes of hypothermia include IV fluids, shaved fur, open body cavities, high fresh gas (O2) flow rates, and surgical preparation solutions (Clancy & Nolan, 2002).

Hypothermia is known to have negative consequences on coagulation and overall immune function and can lower inhalant anesthetic requirements. During anesthesia, hypothermia can result in decreased drug requirements as metabolic rate is reduced. Bradycardia can also be a consequence of moderate-to signifiant hypothermia caused by a reduction in automaticity of the sinoatrial and atrioven tricular nodes (Gray & Morris, 2013).

It is the author's impression that anticholinergics (eg, glycopyrrolate, atropine) are often in effective at treating such bradycardia when core body temperature is below 94°F (34.4°C). Efforts should be made to insulate the patient from cold surfaces and sustain body temperature above 98°F. Warming devices that can be used to sustain body temperature or treat hypothermia should ideally be designated for use in veterinary and/or anesthetized human patients, and manufacturer directions for safe use should be followed (Gornall *et al.,* 2017).

2.2.2.3. Abnormal Heart Rate

Abnormal heart rate (HR) and arrhythmias are commonly observed during general anesthesia. Monitoring for arrhythmias via electrocardiography is crucial during the perianesthetic period, and in combination with direct auscultation, SpO2, and audible Doppler, comprehensive cardiovascular monitoring is provided. Bradycardia generally refers to heart rates <50 bpm in large dogs, <70 bpm in small dogs, and <100 bpm in cats. Bradycardias are generally a result of administration of vagotonic drugs (eg, α-2 adrenergic agonists or opioids), stimulation of vagally mediated reflxes (eg, oculocardiac reflx, intubation), hyperkalemia, or consequences of anesthesia, including excessive depth, hypothermia, and hypoxia. When the cause of bradycardia is increased vagal tone, an anticholinergic should be administered (eg, atropine, glycopyrrolate). Bradycardia is an expected reflx following α-2 administration and need not be treated unless hypotension and/or reduced peripheral perfusion become present. In that case, an α-2 antagonist should be administered fist and given time to take effect before administering an anticholinergic. If the bradycardia is caused by excessive anesthetic depth, hypothermia, or hyperkalemia, the underlying cause should be corrected(Smith & Mishra, 2017).

Tachycardia and tachyarrhythmias generally refer to HRs >200 bpm in cats and >160 bpm in dogs. The concern with significant increases in HR during general anesthesia is a reduction in filing time and stroke volume, impairing cardiovascular function. Anesthetic-related causes of sinus tachycardia are drugs (eg, ketamine, anticholinergics), inadequate depth of anesthesia, pain, or response to surgery, hypoxia, hypercapnia, or hypotension. Additional patient factors to consider include hyperthyroidism, heart disease, anemia, shock, pheochromocytoma, and anaphylaxis. Treatment of sinus tachycardia should focus on the cause. For example, in an animal that is painful, additional analgesia should be provided (Warren & Jakobsson, 2014).

Intermittent and isolated ventricular premature complexes can be normal and harmless in healthy dogs and cats undergoing anesthesia. Intervention is generally not required unless irregular heartbeats become more frequent (>20 irregular bpm), cause a reduction in BP, or transition to ventricular tachycardia. Again, treatment should focus on the source, but in the face of ventricular tachycardia, lidocaine is the primary therapy, followed by procainamide for refractory cases. Care should be taken to always dose reducelidocaine in cats given higher susceptibility to toxicity (Zeng *et al*., 2016).

Table1: therapeutic intervention for anesthetic complications (Anaesth, 2004)

| Condition | Intervention | Dose |
|---|---|---|
| Bradyarrhythmia | Atropine<br>Glycopyrrolate | 0.02–0.04 mg/kg IM, IV<br>0.005*–0.01 mg/kg IM, IV |
| Tachyarrhythmia | Lidocaine<br>Procainamide | 2 mg/kg bolus over 1 to 2 mins, then 40–80 mcg/kg/min CRI (dog); 0.2–0.5 mg/kg bolus (cat)**<br>3–6 mg/kg bolus, then 10–40 mcg/kg/min (dog) |
| Hypotension | Dopamine CRI<br>Norepinephrine CRI | 5–10 mcg/kg/min<br>0.25–3 mcg/kg/min |
| Anaphylaxis | Epinephrine<br>Diphenhydramine | 0.01–0.05 mg/kg IV<br>1–2 mg/kg IM |
| Dysphoria | Acepromazine<br>Dexmedetomidine | 0.01–0.02 mg/kg IV<br>1–3 mcg/kg IV |
| Cardiac arrest | Antisedan<br>Atropine<br>Epinephrine<br>Naloxone<br>Flumazenil | 100 mcg/kg IV (arrest)/IM (non-life threatening)<br>0.04 mg/kg IV<br>0.01 mg/kg IV<br>0.02 mg/kg IV<br>0.01 mg/kg IV |

2.2.2.4. Hypoventilation

Hypoventilation is generally drug-induced. Opioids, propofol, alfaxalone, and inhalants can result in a dose dependent drop in respiratory rate; acepromazine and benzodiazepines can cause minimal respiratory depression. Other causes include impaired respiratory muscle effort (eg, rib fractures, pleural space disease, and obesity) and upper airway obstruction common with brachycephalic airway syndrome and tracheal collapse. Ventilatory monitoring is best accomplished with the use of capnography with a normal end-tidal carbon dioxide of 35–45 mm Hg. This in combination with pulse oximetry provides more complete information of gas exchange at the level of the lung (Anaesth, 2004).

Normal respiratory rates for dogs and cats often vary based on size and positioning with most dogs breathing at 6–10 breaths/minute and cats at 16–20 breaths/minute. In a patient that is not spontaneously ventilating while under anesthesia, depth indicators should be assessed. If deemed adequate, the inhalant should be decreased. Intermittent manual and/ or mechanical ventilation may need to be considered until spontaneous ventilation resumes (Cummings & Wetmore, 2016).

2.2.2.5. Cardiopulmonary Arrest (CPA)

CPA is defined as the sudden cessation of functional ventilation and effective circulation. CPA may be a result of any disease process which disrupts cardiac and/or pulmonary homeostasis. Potential causes of cardiopulmonary arrest include hypoxia, shock, metabolic disorders, trauma, vagal stimulation, anesthetic or other drugs and environmental influences (hypo or hyperthermia) (Bednarski *et al.*, 2011).

The existence of cardiac arrest must be recognized early if we are to effectively resuscitate the patient. In the wake patient consciousness is lost within 10-15 seconds. In the anesthetized patient a declining blood pressure will be one of the first signs you will see. Other signs include the absence of a palpable pulse or audible heart sound, the absence of breathing effort (agonal breaths should not be considered effective breaths) and fixed and dilated pupils. If there is any question that CPA has taken place the patient should be treated as such until proven otherwise Walker & Wilson, 1995).

The goal of cardiopulmonary-cerebral-vascular resuscitation is to provide adequate ventilator and circulatory support until spontaneous functions return. Once it is determined that CPA has taken place an airway is established and the patient is ventilated once every three to five seconds. Chest compression is begun at a rate of 80 – 120 compressions per minute. IV access is obtained either peripherally or centrally and in some cases intraosseous. Epinephrine and atropine are used in the treatment of asystole. Defibrillation is indicated when the patient has ventricular fibrillation. CPA is a rapidly vasodilating disease therefore, fluids should be given rapidly intravenously, in aliquots sufficient to maintain effective circulating volume (Deyoung & Victoria, 1974).

Table2: Emergency Drugs

| Drug | Dose | Comments |
|---|---|---|
| Adrenaline | 0.01-0.02mg/kg<br>0.005-1µg/kg/min | Vasoconstriction, cardiac contractility, bronchodilation. |
| Dopamine (low dose) | 1-4µg/kg/min | Splanchnic vasodilation, natriuresis and diuresis. |
| Dopamine (medium dose) | 5-10µg/kg/min | Arrhythmias with tachycardia. |
| Dobutamine | 2-20µg/kg/min (dogs)<br>1-5µg/kg/min (cats) | Cardiac contractility<br>arrhythmias, tachycardia and vasodilation with seizures |
| Phenylephrine | 0.15mg/kg<br>1-3µg/kg/min | Vasoconstriction with reflex bradycardia. Increased coronary blood flow but decreased splanhnic blood flow. |
| Vasopressin | 0.4-0.8IU/kg (dogs)<br>1-4mU/kg/min (dogs) | Causes vasoconstriction<br>Low doses will cause vasodilation in cerebral, renal, pulmonary & mesenteric vessels. |
| Noradrenaline | 0.05-2µg/kg/min | Vasoconstriction & Increases blood flow |

(Walker & Wilson, 1995)

2.2.3. Cardiopulmonary resuscitation-Response Protocol (rules of ABCDE)

The goal of CPR is to deliver oxygen to the lungs by artificial ventilation, and then transport the oxygen to body tissues by external cardiac compression. The essential steps in responding to a cardiac arrest can be summarized with the mnemonic ABCDE (Airway, Breathing, Circulation, Drugs, and ECG) (Gornall *et al.,* 2017).

Airway (A): Establish a patent (secure) airway as quickly as possible; Clear the airway of any obstructions (excessive mucus, tongue, foreign objects); Perform endotracheal intubation with a cuffed endotracheal tube and If this is not an available option, consider transtracheal catheter ventilation (using a 14g needle or over-the-needle intravenous catheter with a 3mm endotracheal tube connector connected to O2 line or a Bain Circuit) or complete

tracheostomy using a tracheostomy tube set (Gray & Morris, 2013).

Breathing (B): Attach endotracheal tube to a source of 100% oxygen (preferable to room air, if possible); administer 6 ~12 breaths/minute; ratios of one breath per 5 chest compression are used when simultaneously performing chest compression; the amount of gas volume is 10 ~ 20 ml/kg at a peak inspiratory airway pressure of 20 ~ 25 cm H2O; inspiratory time is approximately set at 1.5 seconds and I: E ratio approximately 1:2~3 and a continuous flow of 100% oxygen at 50~150ml/kg/min administered though endotracheal tube or a cannula inserted transtracheally. Although not as efficient, mouth-to nose ventilation is performed when endotracheal tube and respiratory assist device is not available, and could be life-saving (Haller *et al.,* 2011).

Circulation (C) - Cardiac compression: Place the patient in lateral recumbency on a firm surface and compressing the chest at a rate of 80 ~ 120 compressions per minute, devoting equal time to compression and relaxation. Compress the heart in small animals (<10 kg) from both sides, taking advantage of the cardiac pump mechanism of establishing cardiac output. In larger animals (>10 kg), compress over the junction of the dorsal and middle third of the 5~7th intercostal space, relying on the thoracic pump mechanism for generating cardiac output. Compress the chest to depress the chest wall by 30%. The duration of compression (cardiac systole) should be at least 50% of the total compression-relaxation cycle to produce maximal flow. Release the pressure completely during relaxation to allow cardiac filling (AAG, 2007).

Blood flow generated by cardiac compression will temporarily sustain cerebral and myocardial viability only if oxygenation is adequate. However, cerebral blood flow is less than one-fifth normal and coronary flow even less during external compression. Complications, which may arise from external compression, include sternal and rib fractures, blunt traumatic damage to intrathoracic and intraabdominal viscera and pneumothorax (Haller *et al.*, 2011).

Aim of compressions is to manually force blood through the heart and, ultimately, to the tissues (cardiac pump theory). External chest compression squeezes the heart between the thoracic walls, forcing blood into the aorta. Blood flow depends on the rate and pattern of compression. It is also believed that compressions may assist circulation by increasing pressure in the chest, indirectly inducing blood flow (thoracic pump theory). A prolonged compression time (50 -60% of the cycle) favors flow produced by changes in intrathoracic pressure. For large dogs, interposed abdominal compression may aid circulation to cranial half of body. Each compression should result in a palpable femoral pulse. If a pulse is not detected, the method of compression should be adjusted. If external compressions are ineffective-consider open-chest cardiac massage (Walker & Wilson, 1995).

Drug (D) administration: Intravenous fluids - given at 10 to 20 ml/kg to offset peripheral vasodilation. More rapid rates of fluid administration are not indicated unless severe hypovolemia exists. Commonly used emergency drugs: Atropine - Used as treatment for bradycardia; dexamethasone - Corticosteroid used in the treatment for shock; dopamine - Increases force of myocardial contractions, increases heart rate; doxapram - Respiratory and CNS stimulant; epinephrine - Increases rate and force of cardiac contractions. Increases systemic vascular resistance and diastolic blood pressure resulting in improved coronary and cerebral blood flow; Lidocaine-most commonly used to treat ventricular arrhythmias (PVCs or ventricular tachycardia) used to raise threshold for fibrillation; naloxone-Narcotic antagonist and Sodium bicarbonate - Treatment for metabolic acidosis (Cummings & Wetmore, 2016).

Routes of drug administration: First choice is a central venous catheter (jugular) which will provide a more rapid onset of effect compared with administration through peripheral catheters If peripheral catheter is used, external cardiac massage must effectively establish circulation if the drug is to reach the heart. Administration of certain drugs (epinephrine, atropine, lidocaine) into the endotracheal tube lumen allows prompt absorption across the tracheal mucosa and serves as a useful alternate route for drug administration. This is usually done using 2-5 ml of saline as diluent for the drug (at 2 times the IV dosage) administered into the lumen of endotracheal tube, followed by 2-3 large breaths with artificial ventilation. Intracardiac administration of epinephrine is rarely indicated and in fact discouraged. It may be used when the chest is already open. Other routes which permit rapid uptake of drugs include the intralingual and intraosseous routes (Walker & Wilson, 1995).

Electrocardiography (E): If ECG is available we can recognize and treat cardiac arrhythmias. Electrocardiographically, there are three forms of cardiac arrest: Asystole is the absence of any electrical activity (flat line). Initial drug of choice is epinephrine which increases arterial wall tone and peripheral resistance allowing better intrathoracic arterial flow by reducing the tendency of vessels to collapse from the pressure induced by chest compressions. Epinephrine also increases diastolic pressure and renders the fibrillating heart more susceptible to defibrillation. It also diverts blood flow away from nonvital, towards

vital tissues. Ventricular fibrillation appears as completely chaotic, irregular, bizarre deflections. There are no recognizable P or QRS waves. The treatment is to defibrillate with DC current. Lidocaine and sodium bicarb may then be tried if fibrillations are still present. Electromechanical dissociation is present when there is a normal wave form but no effective cardiac output. This form of cardiac arrest carries a fairly poor prognosis (AAG, 2007).

**3. Conclusion And Recommendations**

There are many potential complications with any anaesthetic and it is the important role of the veterinary nurse anaesthetist to monitor patients and highlight changes to the veterinary surgeon but also to have an understanding of the drugs being used. Careful monitoring of the patient during anaesthesia will allow the nurse anaesthetist to promptly identify approaching problems and proceed accordingly. Proper pre-anesthetic medication and preparation (withholding food and water, etc.) and the careful measuring and administration of injectable drugs slowly and to effect will aid in preventing anesthetic emergencies. Also beneficial is regular patient monitoring during the anesthetic and surgical period. Parameters to be monitored may include heart and respiratory rates, the depth of respiration and strength of the pulse (femoral pulse, etc.), capillary refill (recovery time following blanching of mucous membranes due to pressure) and the usual reflexes (pain, jaw, palpebral, etc.). Anaesthetics are inherently unsafe as all drugs affect the cardiovascular and/or respiratory systems. As a result every anaesthetic requires careful monitoring to ensure risks are kept to a minimum, harmful trends are recognized and to allow time for appropriate action. The anaesthetist is the best monitoring tool, while good monitoring technology is available and useful. The presence of an appropriately trained and experienced anaesthetist is the main determinant of patient safety during anaesthesia. Therefore based on the above conclusions the following points are recommended:

- Considering proper pre-anesthetic patient evaluation and careful in administering anesthetic agents.
- Frequent patient monitoring during the anesthetic period and avoid problems usually result from equipment failure.
- Check all equipment before use as recommended in Checking Anaesthetic Equipment.
- Identifying potential risk factors and modifying the anesthetic plan to address the patient's special needs.

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