Management of Common Anesthetic Emergencies

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Management of Common Anesthetic Emergencies

by

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Introduction

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.

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 dosages accordingly. A system that may be used is:

**Physical Status**

<table>
<thead>
<tr>
<th>Number</th>
<th>Explanation</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>No disturbances of body function.</td>
</tr>
<tr>
<td>2</td>
<td>Slight to moderate disturbances of body function.</td>
</tr>
<tr>
<td>3</td>
<td>Extreme disturbances of body function.</td>
</tr>
<tr>
<td>4</td>
<td>Moribund disturbances of body function.</td>
</tr>
</tbody>
</table>

These numbers may be followed by an E for emergency cases.

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.).

Equipment Failure

Emergencies may arise from inadequate supplies of oxygen or anesthetic agent in the vaporizer. Thus, the anesthetic equipment should be thoroughly checked for function and fresh and adequate supplies of oxygen and anesthetic agent in the vaporizer. The contents of the carbon dioxide absorber should be replaced after six to eight hours of intermittent use. One cannot rely totally on the color change of the absorber material; minimal regeneration of this material occurs when it is not in use and so it may be nearly exhausted and yet, in color, resemble new material. A better indication of the state of the absorber material is its feel. Fresh material is moist and crumbles, while exhausted or near exhausted material is dry, hard and chalky.

The equipment should be assembled and the flowmeter and flush valve function checked. At this time the one-way valves and pop-off valve may be observed to be sure they are working; the system should be filled with gas and an examination...
made for leaks in the machine and rebreathing bag and patient hoses. The vaporizer may be turned on and its output checked.

The last accessory to be checked is the endotracheal catheter. The lumen should be patent and to insure a good seal the cuff should not leak. When in place if the endotracheal catheter cuff requires more than 5 ml of air to get an adequate seal a larger size catheter should be used. An over-inflated cuff may restrict air flow and may even totally collapse the catheter’s lumen.

A few seconds spent in anesthetic machine preparation will often save much time and grief later on.

Respiratory Emergencies

Cessation or arrest of respiration (apnea) usually follows depression of respiratory centers from overdosing with an anesthetic or breath holding from inhaling too high a concentration of inhalation anesthetic. Other possible causes include respiratory obstruction, use of muscle relaxants, hyperventilation, and reflex stimulation. Treatment for apnea consists of turning off or drastically reducing the amount of anesthetic agent, maintaining effective cardiovascular and pulmonary function, and having a patent airway. Supportive ventilation by means of “bagging” the animal at the rate to 10–12 beats per minute with a pressure of 18–20 cm of water may cease once the animal has regained spontaneous respirations. In severe cases analeptics may be used.7,11

A diminished ventilatory effort (hypopnea), with slow, shallow respirations will result in inadequate gaseous exchange. Such drugs as sedatives and narcotics, which are depressants, are common causes of slow breathing. Residual effects of muscle relaxants may produce muscle weakness and result in ineffective ventilation. Intubation for administration of oxygen and assisted respiration is indicated in cases of hypopnea.7

Hyperpnea is respiration which is abnormally fast resulting from stimulation during too light an anesthetic plane, hypoxia or hypercapnia.7 Elevated carbon dioxide is most often due to an exhausted CO₂ absorbent. An obstructed or partially obstructed airway may cause a lack of oxygen.

Although laryngeal spasms may occur in any species, it more commonly develops in the cat. Stimulation of the respiratory tract in a lightly anesthetized animal usually initiates the spasm. Coughing and inspiratory noises with some cyanosis follows. To relax the laryngeal spasms, muscle relaxants (succinylcholine) should be administered followed by intubation. The spasms can be prevented by local application of a topical anesthetic to the larynx, or waiting until the reflexes are gone.7,11

An obstructed airway is often encountered especially in certain breeds of dogs. If a partial obstruction exists, the respiration will be noisy. With the increased respiratory effort, the respirations become more diaphragmatic in character. With anesthesia and particularly deeper planes of anesthesia, the manifestations of the obstruction may be reduced and possibly will be unnoticed. Secretions, foreign bodies, tumors, vomitus and exudate can all be causes.4,7,10

The brachycephalic breeds present a special problem. Anatomical abnormalities of stenotic nares and elongated soft palates often complicate the problem. These breeds are more prone to heat strokes, and even simple restraint may be dangerous. Many will show respiratory distress and cyanosis with minimal stress or activity.

Problems of soft tissue airway obstruction can develop quite rapidly, making it important to know how to correctly manage respiratory complications in these breeds. The method of choice for induction of anesthesia is an injection of a short-acting barbiturate followed by immediate intubation. During anesthetic recovery, airway patency should be maintained by means of the endotracheal tube until the animal is nearly conscious. This may require that the larynx be sprayed with a local anesthetic so that the dog will tolerate the endotracheal tube.7,11

Emergence excitement should be avoided. With excitement high negative pres-
sure produced in the airway during inspiration may result in collapse of the larynx and eversion of the lateral ventricles. Sedatives have a proper use in this situation. They reduce the respiratory effort, which decreases resistance. Also the endotracheal tube is tolerated with the sedation. 11

Cardiovascular Emergencies

Shock is an inadequate profusion of tissues resulting in hypoxia of the cells. Blood pressure may be normal, but the tissues are poorly oxygenated. Shock can be classified into four groups: hemorrhagic, septic, cardiogenic, and neurogenic. Although all four types have different initiating causes, the process gradually develops to where little distinction can be made among them. 7

Regardless of the type of shock, general management should center around the same basic ideas.

Adequate ventilation should be provided. Optimum oxygenation of the blood is necessary and proper elimination of CO₂ is needed to prevent a respiratory acidosis. Restoration of blood volume and proper replacement of fluids can be done by monitoring CVP. Lactated Ringer's is an effective fluid for this use. In shock the animal is acidic and therefore, for immediate counteraction, sodium bicarbonate is added to the fluids. Sodium bicarbonate reacts readily with the acid-base system of the blood, whereas lactate must be metabolized by the liver. Attention must also be directed toward the heart. An elevated or rising CVP with lack of clinical improvement signifies a deteriorating heart. Isoproterenol exhibits a chronotropic effect on the heart and will increase the cardiac output. Supportive treatment should include corticosteroids and broad-spectrum antibiotics. 4,7

In treating shock, fluid replacement should be started before giving an intravenous corticosteroid since they initially cause a drop in blood pressure. In the pathogenesis of shock a pooling of blood occurs due to constriction of pre-and post-capillary sphincters in the capillary beds. With the accompanying stasis of blood, metabolic products particularly lactic acid, accumulate and cause a relaxation of the pre-capillary sphincter. The corticosteroids act primarily on the post-capillary sphincters. The sudden release of the post-capillary sphincter and the resulting decrease in vascular resistance produces the initial drop in blood pressure. To insure the proper replacement of blood volume, the CVP should be monitored. Fluids are to be given until the CVP reaches nearly 15 cm of water.

The approach of therapy to shock is done with some degree of urgency since the disorder can be progressive. Any undue movement of the animal should be avoided. All depressants and hypotensive drugs may overtax the animal's already weak compensatory mechanism and are not recommended. 7,9 Additional therapy will include use of vasoactive drugs, supportive ventilation and monitoring of essential respiratory and cardiovascular parameters.

Although arrhythmias may be a sign of impending cardiac hazards, these are frequently seen in well managed patients. Any disturbance of the electrophysiology of the heart may initiate the arrhythmia. A modification of the heart's automaticity, conductivity, rhythmicity, and contractility influences the mechanisms by which the irregularities are produced. Besides changes within the heart, arrhythmias may also develop from changes in the central nervous system or periphery.

An imbalance in the autonomic nervous system with changes in the parasympathetic or sympathetic tone is the most common cause of arrhythmia while under anesthesia. 4,7

Anesthetic agents, drugs, blood gases, arterial pressures, electrolytes and other factors are known to influence the development of arrhythmias. As an example, inadequate ventilation from hypercapnia and/or hypoxia will produce arrhythmias during anesthesia. Proper anesthetic technique when handling the patient will help avoid this.

Generally speaking, slow supraventricular rhythms such as wandering pacemaker,
sinus arrhythmia and A-V nodal rhythm are benign. Ventricular arrhythmias should be regarded as being potentially serious. An occasional unifocal ventricular extra systole are common under anesthesia and are usually not harmful. With multifocal ventricular extra systoles and ventricular tachycardia, the diastolic filling time of the heart is decreased. This change in the cycle of the heart, affects the cardiac output and blood flow in the coronaries. But even more serious is the chance that the heart will go into ventricular fibrillation. In a normal patient a supraventricular tachycardia may be well tolerated but in one suffering from cardiac disease or poor oxygenation, there may be serious effects.6,9,11

Bradycardia exists when the heart rate falls below 90 per minute. This finding should not be treated lightly since it may precede asystole. This arrhythmia can be circumvented with the proper preanesthetic medication. An anticholinergic; atropine being the one of choice, is used for this purpose. Atropine prevents the bradycardia by blocking the parasympathetic innervation to the heart.2,6,9

Anesthetic agents have a definite effect on the cardiovascular system of the animal. They have a direct effect on the heart and with high doses they depress respiration and cause arterial hypotension.

Some halogenated hydrocarbon anesthetic agents (halothane and methoxyflurane) depress the contractility of the myocardium. Halothane appears to be more of a depressant than does methoxyflurane. With the use of these agents, the myocardium is sensitized to the effects of the catecholamines. Catecholamine-induced arrhythmias are less apt to occur with methoxyflurane than with halothane.

Halothane exhibits more vasodilatation than does methoxyflurane, thus hypotension is more noticeable with halothane. Atropine may be used to reverse the effects of the parasympathetic nervous activity of halothane. This activity can be manifested as bradycardia or ventricular extra systoles and may eventually lead to hypotension.

If effective ventilation can be achieved along with a light level of surgical anesthesia, the occurrence or arrhythmias under either halothane or methoxyflurane is uncommon. Cardiac depression will be less if the addition of nitrous oxide is enough that the amount of hypotensive inhalant anesthetic needed to maintain the animal can be reduced.1,6,8

Some species difference does exist as to the ease of inducing arrhythmias. Cats are more susceptible to anesthetic arrhythmias than are other domestic animals. Why this is so, is not known for certain. Cats have been observed to develop arrhythmias during induction using inhalation anesthesia. A smooth and calm induction with a minimal amount of restraint is important with cats.6

Parenteral anesthetics do have a myocardial depressant action. Their use is most commonly for induction and consequently the depressant effect is transient.8

When confronted with managing an arrhythmia during anesthesia, attention should center on removing the cause. The anesthetic concentration should be reduced and high concentrations of oxygen administered to ventilate the animal. In cases of hypercapnia, hypoxia or deep levels of anesthesia, this should be adequate. Lidocaine may be given intravenously in doses of 0.25–2 cc to desensitize the myocardium. The lidocaine should be 1% strength and without epinephrine. Further measures to employ if the arrhythmia persists include fluid replacement, correction of acid-base imbalance, and maintaining proper body temperature. If a hypotensive anesthetic agent is being used, it may be necessary to switch to one that is less hypotensive. Drugs may be needed at this time to control the arrhythmia. Caution must be exercised as to the proper drug to use. The wrong drug may compound the problem.3,6,10

Ventricular fibrillation is characterized electrocardiographically by waves of varying voltage. When fibrillation occurs, chemical cardioversion may be tried if no electrical defibrillator is available. One half milliliter–2 ml. of procaine or lidocaine may be injected into the heart. Electrical defibrillation can be accomp-
lished through the intact chest wall or by direct placement on the heart. An intravenous injection of sodium bicarbonate and 0.5 mg epinephrine have been shown to increase the likelihood of cardioversion.1,2,7

In cardiac arrest there is a sudden cessation of the heart beat; often times preceded by ventricular asystole or ventricular fibrillation. The etiologies during anesthesia include:

1) excitement during induction.
2) ineffective pulmonary ventilation.
3) direct effect of anesthetic agents and drugs on the heart.
4) imbalance of the autonomic nervous system.
5) hypoxia and/or hypercapnia.

When cardiac arrest occurs, the clinical signs are apparent apnea, dilated pupils, lack of heart sounds, no hemorrhage and absence of a pulse. One can not delay in instituting resuscitative measures in the event of cardiac arrest. Without swift management, irreversible and possibly fatal changes can occur in vital organs, especially the brain. If left untreated, in 3–4 minutes cardiac arrest will result in brain damage. Because of the rapid effect on vital organs the prompt diagnosis and treatment is important.1,5,7

As quickly as cardiac arrest is observed, stop the administration of the anesthetic. A patent airway must be maintained. This is best done by intubation. Since apnea is likely to be present, insuring effective ventilation will be a part of the resuscitation. This will mean supportive ventilation, optimally with 100% oxygen by one of several methods. Attention must also be given to the animal’s circulation. The head should be tilted down at about 30° and fluids started to augment venous return. To restore circulation, indirect cardiac massage should be applied to the heart. In dogs and cats, the rate will be 60–80 per minute. If the chest is already open or if the indirect massage is ineffective then direct massage is the choice. If cardiac arrest is not reversed with massaging, 1–3 ml of 1:10,000 may be injected and followed by further massaging. Massaging is continued until adequate heart action has been noted for 5–10 minutes.1,5,10

The presence of a weak pulse upon palpation may signify a failing heart, cardiac arrhythmias, blood volume deficit or anesthesia that is too deep. Any treatment should be aimed at correcting the cause. Level of anesthesia will need to be lightened if the animal is too deep.

Calcium and isoproterenol are indicated where the animal has a failing heart. Bradycardia can be reversed by the use of atropine I.V. at pre-anesthetic doses. Any weakness of pulse due to loss of blood volume will require administration of fluids or blood.

Summary

We have briefly covered the subject of complications during the anesthetic period. Table 1 has been prepared as a summary and aid to assist the practitioner and his assistants in treating these problems as they occur. It is hoped that this information will assist the practitioner in formulating a plan for anesthetic emergencies.

REFERENCES

### Dosage and Route

To effect topical

0.02 mg/lb, I.V., I.M.

1 vial/80 cc saline I.V.

to effect drop by drop

2–5 ml I.V. slowly

1 cc/40 lbs.

2–5 ml, I.V.

5 mg/lb I.V.

2 mg/lb I.V.

2–5 mEq/lb I.V.

to effect

0.5–3 ml (1:10,000)

What’s Your Radiographic Diagnosis?

*Mr. Roth is a third year student in the College of Veterinary Medicine. Iowa State University.

**Winthrop Laboratories** brand of isoproterenol.

Winthrop Laboratories Division of Sterling Drug Inc., New York, N.Y. 10016.