Anesthetic emergencies and cardiopulmonary resuscitation

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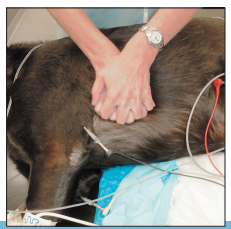

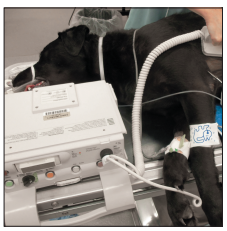

Introduction

Despite careful attention when monitoring the anesthetized patient, emergencies related to general anesthesia can and do occur. Most anesthetic emergencies can be divided into two groups:

- Unexpected crises that develop in normal, healthy patients presented for elective surgeries. These are often related to anesthetic machine and equipment errors and may be prevented with careful management.
- Events that develop as a result of the drugs given to produce general anesthesia. The side-effects of these drugs can be life-threatening.

This chapter reviews the most common life-threatening emergencies associated with anesthesia and their treatment.

Respiratory complications that result in anesthetic emergencies

APNEA

- Apnea (**192**) is most often induced by IV induction agents.
- Apnea can occur with any agent, including thiopentone, propofol, etomidate, alfaxalone, or ketamine.
- It is most likely to occur when large doses of the drug are administered in a rapid bolus.
- Propofol, etomidate, or alfaxolone should be administered slowly and 'to effect', minimizing the potential for apnea (see also Chapter 3). Thiopentone can cause some stage II excitement at subanesthetic dosing.
- Transient apnea is not usually a major concern if the patient can be ventilated.

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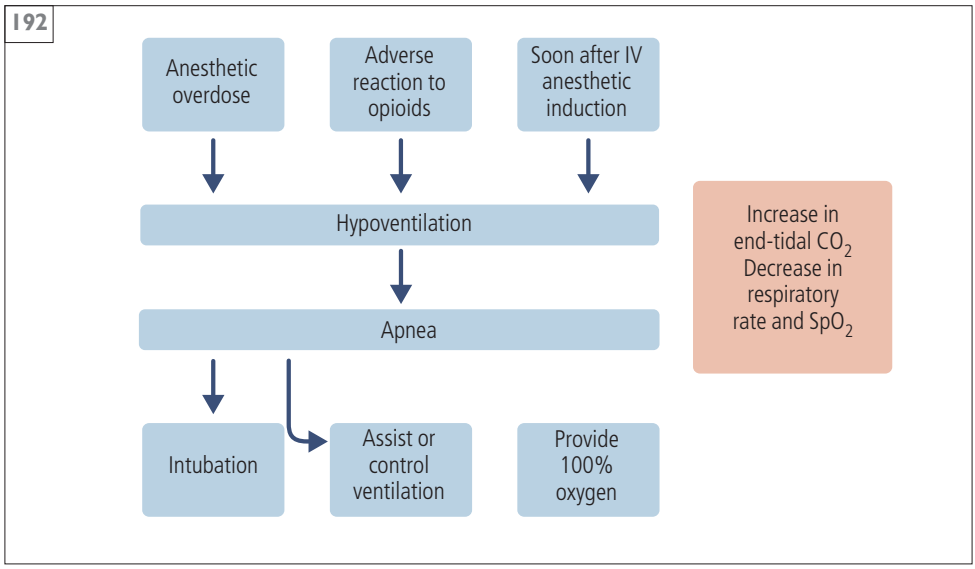


192 Diagramatic representation of the relationship between anesthetic-induced hypoventilation and apnea.

- Apnea is a significant clinical concern if the patient cannot be intubated and the airway is not controlled. (See also Chapter 8.)

HYPOVENTILATION

- Every anesthetized patient hypoventilates as a result of CNS respiratory center depression imposed by the anesthetic drug (**7.1**). The medullary respiratory center may not respond to higher levels of CO_2 with increased ventilation.
- Hypoventilation is an insidious problem, as most veterinarians and technicians assume that an adequate respiratory rate and respiratory efforts equate to adequate alveolar ventilation and gas exchange.
- The amount of gas exchanged with each breath (tidal volume) has two components: dead space gas and alveolar ventilation.
- Dead space gas is the air that is in the conducting airways and is not available for gas exchange. It remains relatively constant and is the first gas in and out of the mouth or nasal passages, trachea, and other conducting units of the respiratory tree.
- Under general anesthesia, tidal volume decreases and dead space gas remains the same, so alveolar ventilation must decrease.
- Hypoventilation can be confirmed by observing $ETCO_2$ values >45 mmHg and by observing less frequent respiratory efforts.
- Capnography is useful for the diagnosis of hypoventilation. (See also Chapter 5.)
- Mechanical ventilators may be used to prevent or treat hypoventilation during general anesthesia. (See also Chapter 8.)
- Assisting ventilation by occasional squeezing of the rebreathing bag (sighing) may help prevent hypoventilation and atelectasis.
- Hypercarbia may also result from anesthetic equipment malfunctions (see chapter 1, p.29 and Chapter 5, p.151).

LOSS OF AIRWAY

- Loss of airway is a condition that has resulted in the deaths of many otherwise healthy patients and is a primary reason for anesthetic emergencies in all species. Loss of control of the airway, especially when unrecognized by the anesthetist, leads to problems due to the CNS depression and respiratory depression induced by general anesthetics. Some examples of conditions that can lead to patient mortality include:
  - Inadvertent extubation and placement of the endotracheal tube into the esophagus.
  - Endotracheal tube occlusion by mucous plugs or blood.
  - Kinking of the endotracheal tube.
  - Overinflation of the endotracheal tube cuff.
  - Endotracheal tube is too short.
  - Endotracheal tube is too long, resulting in one-lung ventilation from improper tube placement.
- See Chapter 8 for further information.

HYPOXEMIA

- Hypoxemia is a common complication of general anesthesia. The five major causes of hypoxemia or low arterial oxygen tensions (PaO_2) include:
  - Low inspired oxygen concentration most commonly occurs with equipment failures and errors. For example, the oxygen flow meter is inadvertently not turned on or an endotracheal tube becomes kinked or obstructed. A common manifestation of this occurrence is the patient who appears to be 'waking up' or is at too light a depth of anesthesia. Hypoxia will cause a ventilatory drive when PaO_2 levels are <50–60 mmHg and the gasping behavior of the severely hypoxic patient can mimic arousal.
  - Hypoventilation, especially when FiO_2 (inspiratory fraction of oxygen) = 21% (room air). Oxygen supplementation should be considered in patients undergoing injectable anesthesia (e.g. IM tiletamine/zolazepam or ketamine in cats). Hypoxia secondary to hypoventilation while breathing room air can lead to prolonged recovery and adverse consequences such as blindness when cerebral blood flow and oxygen delivery are compromised during general anesthesia.

  - Barriers to diffusion of respiratory gases result from problems such as pneumothorax or pulmonary edema. Oxygen is usually affected first, since CO_2 is about 20 times more soluble than oxygen. Pulmonary edema, pleural effusion, and pneumothorax should be corrected as much as possible prior to general anesthesia. Occasionally, occult conditions will manifest during the course of general anesthesia and must be handled during the procedure.
  - Ventilation–perfusion mismatch occurs in small animal patients, but with less frequency than in large animals. Ventilation–perfusion mismatch can be seen with embolic events.
  - A right-to-left shunt (pulmonary) can occur.
- Patient oxygenation can be monitored via pulse oximetry or blood gas analysis. Pulse oximetry is more frequently used, as it is economical, noninvasive, continuous, and easy to apply to the patient (see chapter 5, p. 142).
- The administration of positive end expiratory pressure (PEEP) (see Chapter 8) can be helpful in the hypoxemic patient, as it increases alveolar participation in gas exchange and may recruit collapsed alveoli.
- Desaturation events or low SpO_2 occur with the following situations:
  - Severe hypoxia.
  - Inadvertent bronchial intubation.
  - Embolic events.
  - Pulmonary edema.
  - Pleural effusion.
  - Hypoventilation if not breathing 100% O_2.
  - Anaphylaxis.
  - Bronchospasm.
  - Low cardiac output.

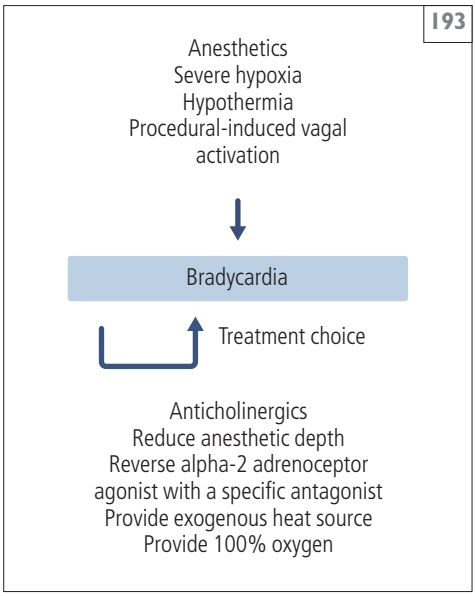
LARYNGOSPASM

- See Chapter 8, p. 189.

Cardiovascular complications that result in anesthetic emergencies

BRADYCARDIA (193)

- General heart rate guidelines under inhalant anesthesia:
  - Large dogs: approximately 60 bpm.
  - Small dogs and cats: 80–90 bpm.
  - Puppies and kittens will need to maintain a higher heart rate than an adult animal.
  - Blood pressure monitoring can be a useful guide to therapy. If the heart rate is borderline low with good blood pressure, additional intervention may not be necessary.
- Many of the drugs used as adjuncts to anesthesia increase vagal tone (e.g. opioids, alpha-2 adrenoceptor agonists).
- Many surgical treatments can stimulate vagal tone as well (e.g. airway surgery, ocular surgery, bladder surgery).
- Cardiac output is the product of stroke volume and heart rate. If the heart rate becomes critically low, output will suffer, especially under the conditions of general anesthesia, as cardiac output is already reduced by the anesthetic agents.
- If the heart rate is normal, increasing the heart rate dramatically will not increase the patient's cardiac output and may compromise myocardial oxygenation.
- Anticholinergic drugs such as atropine or glycopyrrolate are appropriate therapy for bradycardia of vagal origin:
  - They can be administered IV or IM.
  - They are often administered IV in critical situations.
  - They may be administered IM for prevention of bradycardia when opioids are used.
- Heart rate under general anesthesia should be higher than if the patient was not anesthetized, as anesthetic drugs depress contractility and decrease venous return in addition to their effects on heart rate.



193 Causes of bradycardia and its treatment.

- Anticholinergic therapy with alpha-2 agents (e.g. medetomidine or dexmedetomidine) is controversial. Reversal of the alpha-2 adrenoceptor agonist with an alpha-2 adrenoceptor antagonist, such as atipamezole, may be used if the bradycardia from these drugs is severe. A partial dose of atipamezole may be administered to increase heart rate while preserving some alpha-2 analgesia.
- Some bradycardia is not due to parasympathetic activity and will not respond to anticholinergic therapy (e.g. excessive inhalant anesthetic depth, hypothermia).

HYPOTENSION

- Key concepts:
  - Low blood pressure can become an anesthetic emergency when tissue perfusion is compromised.
  - Tissue perfusion is a balance between cardiac output, organ blood flow, and peripheral vessel tone.
  - MAP is the best estimate of tissue perfusion pressure in a clinical situation.
  - A MAP of 60 mmHg should provide sufficient blood flow to the vital organs, such as the brain, heart, and kidneys.
- Inhalant anesthetic agents cause hypotension by two basic mechanisms:
  - Vasodilation (especially isoflurane and sevoflurane).
  - Cardiac output reduction by decreasing contractility.
- If the patient has an MAP <60 mmHg, therapy should be initiated. This therapy can consist of decreasing anesthetic depth, improving peripheral fluid volume, increasing cardiac output, or use of vasopressors (see Chapter 5).
- Decreasing anesthetic depth:
  - Decrease the vaporizer setting to the lowest necessary to provide general anesthesia. The addition of sedative/analgesics such as opioids or alpha-2 adrenoceptor agonists may provide analgesia and allow lower vaporizer settings through a MAC sparing effect.
- Adequate peripheral fluid volume:
  - Patients anesthetized with inhalant agents need fluid support regardless of any surgical blood loss or insensible fluid losses.
  - Venous return is the primary determinant of cardiac output in a healthy patient (Frank–Starling Law of the heart); an anesthetized, vasodilated patient will have greatly diminished venous return without fluid support. The patient will also be experiencing decreased contractility as a result of inhalant agent administration.
  - Fluids administered during anesthesia are usually given at a rate of between 5 and 10 ml/kg/hour with a balanced, isotonic crystalloid solution in a healthy patient with normal cardiac function. This rate can be maintained in an anesthetized patient for 3–4 hours and then reduced.
  - Occult hemoconcentration is a very common finding in patients presented for anesthesia, especially if they have been hospitalized. It is not necessary to withhold water prior to anesthesia, but many hospitalized patients may be too stressed to drink well.
  - Induction with propofol can cause profound vasodilation and hypotension. When encountering patients with a higher than expected heart rate, one of the first things to check is the volume status of the patient to determine if the high heart rate is a compensation for hypovolemia. A fluid challenge of 5–10 ml/kg can be very helpful in reducing the heart rate to a normal value.
  - Hypoproteinemic patients (albumin <20 g/l [2.0 g/dl] or TP<40 g/l [4.0 g/dl]) benefit from colloid administration (e.g. hetastarch or plasma), while anesthetized. Crystalloid administration may need to be reduced to avoid further dilution of plasma protein, but the patient's volume needs must still be met.
- Central venous pressure monitoring via jugular catheterization is the gold standard for monitoring fluid overload, but careful attention to breath sounds and auscultation for crackles will be helpful as well.
- Sometimes, careful peripheral volume support and control of anesthetic level is not sufficient to correct a hypotension problem. A logical next step is to increase cardiac output if possible. Usually this is achieved by inotrope administration. Some of the more commonly used sympathomimetic drugs for this purpose include dobutamine, dopamine, and ephedrine (see Chapter 5).

HEMORRHAGE

- Hemorrhage affects delivery of oxygen to tissues when blood volume losses are high. In general, a PCV of >0.2 l/l (20%) and a TP >35 g/l (3.5 g/dl) in the anesthetized patient is desirable. If a patient's blood loss is >20 ml/kg, or if the blood loss is not

measurable but the PCV and TP are lower than the above values, some sort of oxygen carrying solution is required.
- If blood products are not available, Oxyglobin® can be used as an oxygen carrier and will provide colloidal support as well. Oxyglobin® dose rates vary from 5–30 ml/kg depending on the species, the severity of the situation, and the preoperative volume status of the patient.

CARDIAC ARRHYTHMIAS

Definition

- Arrhythmias are defined as an abnormality in heart rhythm and/or rate in the site of origin of the cardiac impulse, or as a disturbance in the normal conductance of the cardiac impulse.
- Arrhythmias become anesthetic emergencies when they interfere with cardiac output to an extent that they decrease tissue perfusion.

Types of arrhythmia (*Table 31*)

- **Bradyarrhythmias (slower than normal rates)**. Should initially be treated with an anticholinergic drug (e.g. atropine or glycopyrrolate). Atropine (0.04 mg/kg IV) should be used if the heart rate is falling rapidly and severely. Glycopyrrolate is frequently used as a preanesthetic medication to stabilize the heart rate due to its longer duration of action than atropine (see Chapter 2).
- **Ventricular arrhythmias**. If ventricular arrhythmias (VPCs) are discovered during anesthesia, several criteria can be used to determine how disruptive the arrhythmia is likely to be. In general, arrhythmias may be more of a problem during anesthesia if they are multifocal, if the VPCs are >12/minute, or if they are associated with pulse deficits.
- It is important to differentiate VPCs from ventricular escape beats. The two conditions have different therapeutic managements.
- When VPCs are detected during anesthesia, consideration must be given to what may be causing them and if they can be corrected during the anesthetic period.

Causes of arrhythmias (*Table 31*)

- Iatrogenic:
  - Caused by a drug administered as part of the anesthetic protocol.
  - Commonly used anesthetics and adjunctive drugs that can cause arrhythmias include thiopentone, propofol, xylazine, medetomidine/dexmedetomidine, and anticholinergics. However, any drug may cause an arrhythmia in a sensitive individual.

Table 31 Arrhythmias: causes, types, and treatments

Causes	Types	Treatments
• Iatrogenic • Metabolic • Hypercarbia • Hypoxia • High sympathetic tone (pain) • High vagal tone • Primary cardiac diseases • Hypothermia • Hyperthermia	• Rate (tachycardia, bradycardia) • Rhythm (regular or irregular) • Site of origin (atrial, supraventricular and ventricular) • Disturbance in the normal conductance of the cardiac impulse (single or multifocal)	• Treat primary causes first • For bradyarrhythmias, use atropine (0.02–0.04 mg/kg IV) or reverse with atipamezole if it is induced by medetomidine or dexmedetomidine • For ventricular premature contractions (multifocal with pulse deficit), use lidocaine (2 mg/kg IV) • For ventricular escape beats, use atropine

- Metabolic and electrolyte disturbances:
  - Metabolic acidosis.
  - Hypokalemia.
  - Hyperkalemia. (**Note:** Hyperkalemia will cause bradycardia.)
  - Calcium disturbances.
- Hypercarbia:
  - High levels of CO_2 stimulate the sympathetic nervous system and can cause rhythm disturbances, as well as have a direct depressant effect on the myocardium.
- Hypoxia.
- Sympathetic stimulation or pain:
  - Paradoxically, the plane of anesthesia may need to be deepened and/or the addition of analgesic drug therapy may be warranted.
- High parasympathetic tone (vagal tone):
  - Manipulation of airway, bladder, or globe of the eyes may cause a sudden decrease in heart rates.
  - Brachycephalic breeds tend to have a higher vagal tone.
- Primary cardiac disease, such as:
  - Cardiomyopathy.
  - Primary rhythm disturbances.
- Hypothermia:
  - Usually causes bradycardia.

Investigating arrhythmias

- Blood gas and electrolyte analysis can be very useful when looking for the source of the problem.
- Once an arrhythmia has been detected, blood pressure monitoring is very helpful. If the patient is able to maintain an acceptable MAP under anesthesia even though it has an arrhythmia, that is a good indication that the anesthesia can continue.
- Failure to maintain an adequate MAP is an indication that anesthesia should be terminated as quickly as possible.
- If a procedure requires general anesthesia despite significant ventricular arrhythmia, a test dose of lidocaine (2 mg/kg IV) can be given. If the patient responds to lidocaine therapy, a lidocaine CRI (50 mcg/kg/minute) can be started.

Other complications that result in anesthetic emergencies

HYPOTHERMIA

- Extremes in body temperature can cause considerable problems in anesthetized patients. Most patients will lose body heat as a result of general anesthesia. Heat loss is caused by:
  - Inhalants. These cause vasodilation, which promotes heat loss in the patient.
  - High oxygen flow rates, which will increase body cooling because compressed gas is very cold and dry.
  - Hair removal and surgical preparation solutions.
  - An open body cavity.
- Body temperature should be kept as near normal as possible for most anesthetized patients.
- The effects of hypothermia are:
  - It causes a significant stress response in the postoperative period (sevenfold increases in catecholamines).
  - It results in impaired coagulation.
  - It results in impaired tissue perfusion and delayed wound healing.
  - Temperature affects the MAC of the inhaled anesthetics, which is a measure of potency, so the colder the patient, the less anesthetic will be required.
  - It causes a bradycardia that is unresponsive to anticholinergics.
- Steps should be taken to reduce the amount of body heat lost by anesthetized patients. Forced air warmers, circulating water heating pads, and fluid warmers are among some of the devices that are very useful.

HYPERTHERMIA

- High body temperature can be seen in anesthetized patients and in the postoperative period.
- Hyperthermia can be a concern with heavy coated animals and animals that are panting.
- Inadvertent overheating with warming devices in the heavy coated animal during general anesthesia is a frequent cause of intraoperative hyperthermia.
- It is also a concern with metabolic and genetic hyperthermic syndromes (malignant hyperthermia). Triggers include all inhalant anesthetics and succinyl choline. Greyhounds may have more hyperthermic problems.
- The ability to monitor body temperature can be very helpful in identifying these problems in the early stages. Capnography will also register the increased CO_2 levels seen when muscle metabolism is high, which can be one of the first signs of malignant hyperthermia.
- Treatment of hyperthermia involves:
  - Early identification of the problem.
  - Rapid body cooling.
  - Heating pads turned off or removed.
  - Cold water baths.
  - Cold water enemas.
  - Ice packing (protect skin from direct contact).
  - Dantrolene therapy if malignant hyperthermia suspected.
  - Stopping the cooling process at 40°C (104°F).

Postanesthetic hyperthermia in cats

- More and more cats are seen with high postoperative temperatures.
- The temperature increases come several hours after the end of the anesthetic and occur with a variety of anesthetic protocols.
- Hydromorphone is strongly associated with hyperthermia in cats following anesthesia.
- Treatment has consisted of:
  - Opioid reversal.
  - NSAIDs.
  - Acepromazine.
  - Direct body cooling.

Cardiopulmonary resuscitation

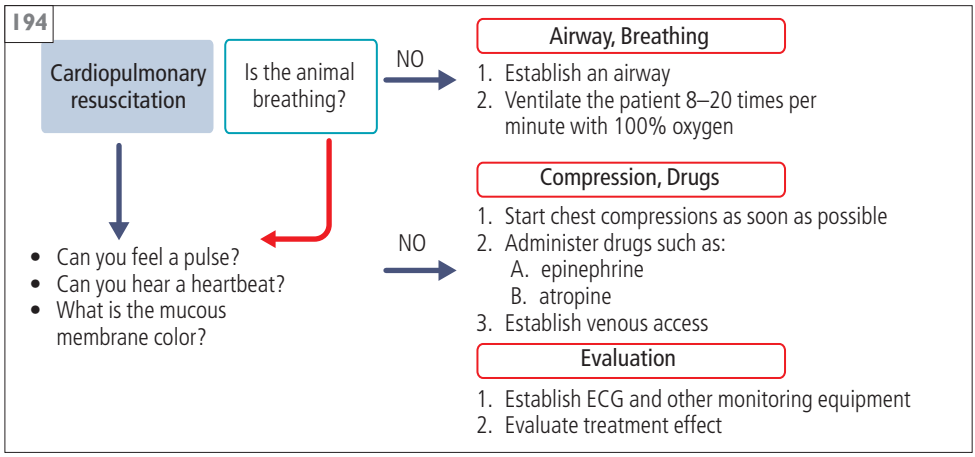
IDENTIFICATION OF ARREST VIA CHECKING OF VITAL SIGNS

- Can you hear a heartbeat?
- Can you feel a pulse?
- Is the animal breathing?
- What is the mucous membrane color?

ACTION TO BE TAKEN

If the signs of cardiopulmonary arrest are present, immediate action must be taken (see **194** for a procedural flow chart).

- The longer the time between identification of the arrest and cardiopulmonary resuscitation (CPR), the less likely there will be successful resolution of the problem. Animals that have experienced an anesthetic mishap or accident and who are basically healthy are much more likely to be successfully resuscitated than animals that arrest with many underlying disease problems.
- CPR can be divided into four steps: airway, breathing, circulation/compressions, and drugs (*Table 32*).



194 Procedural flow chart for performing cardiopulmonary resuscitation.

Table 32 Cardiopulmonary resuscitation sequence, drugs, and dosages

Airway	**Validation of airway patency**	
Endotracheal intubation with: • Endotracheal tube • Laryngoscope • Suction of blood or fluid	Correct endotracheal tube placement by: • Visualization of tube in the airway • Proper chest or anesthetic reservoir bag excursion • Palpation of endotracheal tube in the trachea • Use of capnography	
Breathing	**Frequency**	**Sequence**
Use an anesthetic machine with a breathing circuit or Ambu bag	Initially 2 breaths, 1–2 seconds in duration, with 100% oxygen	After establishment of a patent airway

(*continued*)

Table 32 Cardiopulmonary resuscitation sequence, drugs, and dosages (continued)

Breathing	**Frequency**	**Sequence**
	Evaluate spontaneous ventilation	If yes, observe the animal; if no, check for pulse and rhythm and consider reversal agents for drugs that may cause apnea
	No spontaneous ventilation	Give 10–12 breaths/minute with ≤20 cmH_2O
Circulation/compressions	**Frequency**	**Sequence**
Chest compression	80–100/minute with a 1:1 compression:relaxation ratio	Chest compressions should be initiated first and continued with no pauses during administration of endotracheal intubation, ventilatory breaths, placement of IV catheters, ECG assessment, palpation of pulses, or administration of drugs
Drug	**Dose and route**	**Indication and details**
Atropine	0.04 mg/kg IV 0.08–0.1 mg/kg IT*	Bradycardia. Can repeat every 3–5 minutes for a maximum of 3 doses
Atipamezole	0.1–0.2 mg/kg IV	Bradycardia induced by alpha-2 adrenoceptor agonists; alpha-2 adrenoceptor antagonist
Calcium gluconate (10% =100 mg/ml)	0.5–1.5 ml/kg IV slowly	Hyperkalemia and documented ionized hypocalcemia. Do not give via IT route
Epinephrine	0.01 mg/kg IV 0.03–0.1 mg/kg IT*	Increase rate and force of cardiac contractions, increase peripheral vasoconstriction. Repeat every 3–5 minutes
Lidocaine	2–4 mg/kg IV 4–6 mg/kg IT*	Ventricular tachycardia, ventricular premature contractions. Repeat every 3–5 minutes
Magnesium sulfate	0.15–0.3 mEq/kg IV slowly over 10 minutes	Treatment of refractory ventricular arrhythmias, including ventricular fibrillation and life-threatening mutilfocal ventricular tachycardia
Naloxone	0.02–0.04 mg/kg IV 0.04–0.06 mg/kg IT*	Opioid antagonist
Sodium bicarbonate	0.5–1 mEq/kg IV	Metabolic acidosis
Vasopressin	0.2–0.8 U/kg IV 0.4–1.0 U/kg IT*	Increases peripheral, coronary, and renal vasoconstriction, but less than epinephrine
External defibrillation; shock energy	2–5 joules/kg 50 joules for small dogs and cats 100 joules for medium dogs 200 joules for large dogs	Asystole, ventricular fibrillations
Internal defibrillation; shock energy	0.2–0.5 joules/kg	Open chest is necessary for this procedure

*Medications administered intracheally (IT) should be diluted in 3–5 ml of sterile water.

First step (airway)

- Is the animal intubated?
- Is the airway controlled?
- If the patient is under general anesthesia, is the patient correctly intubated and the tube unobstructed.
- If the patient is under general anesthesia, the inhalant anesthetic should be immediately terminated and the breathing circuit flushed with 100% oxygen to rapidly clear the residual anesthetic.

Second step (breathing)

- Previous recommendations for veterinary patients were to provide a ventilation rate of 20–24 breaths per minute.
- Newer guidelines suggest a lower rate of ventilation (10 breaths per minute, a tidal volume of 10 ml/kg, and an inspiratory time of 1 second delivered simultaneously with cardiac compressions) to avoid decreases in myocardial (due to positive pressure ventilation related reduction of venous return to the heart) and cerebral (due to lower arterial CO_2 resulting in cerebral vasoconstriction) perfusion.
- Ventilate the patient with 100% oxygen.
- Make sure the vaporizer has been turned off.
- Does the chest expand easily? If not, check that the endotracheal tube is not obstructed or the patient does not have a chest full of blood (hemothorax) or air (pneumothorax).

Third step (circulation/compressions)

- Chest compressions should be started very quickly. The patient should be positioned in lateral recumbency with the back towards the person doing the compressions in order to prevent the animal from sliding around on the table (**195**).
- The chest wall should be allowed to recoil completely after being compressed to approximately one-third to one-half of the width of the chest, with no pauses during administration of ventilator breaths or other necessary tasks (IV catheter placement, ECG assessment, or administration of medications).
- Forward flow of blood is the goal of chest compressions.
- There are two theories:
  - The cardiac theory utilizes direct compressions of the heart to provide forward flow.
  - The thoracic pump theory hypothesizes that changes in intrathoracic pressure created by cardiac compressions are responsible for forward flow.
- The larger the animal, the more difficult it is to perform effective compressions.

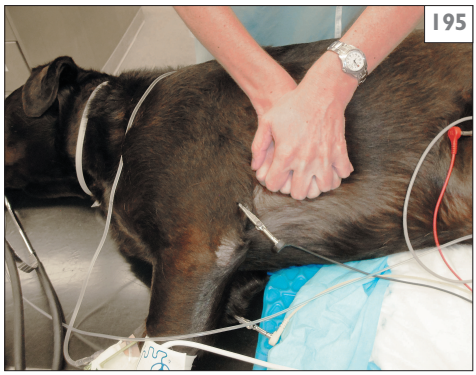
The current recommendation for chest compression is that there should be 100–120 compressions per minute. The compressions should be delivered in uninterrupted cycles of 2 minutes.

Fourth step (drugs)

- Epinephrine (1:10,000) should be administered as soon as the arrest is identified.
- Several dosing regimes are available (0.01–0.1 mg/kg), but in a crisis it can be difficult to take the time to look up the weight of the animal and calculate a dose.
- Alternatively, administer 0.5–1.0 ml to a small dog or cat, 2 ml to a medium size dog, and 3 ml to a large dog.
- Epinephrine should be given IV. If venous access is not available, a double dose can be administered down the endotracheal tube and washed in with several milliliters of water or saline.
- If a slow heart beat is identified prior to the arrest, atropine should be given (0.04 mg/kg IV).
- Cardiac compression is necessary during bradycardia in order to deliver the IV emergency drugs to the heart through the compression-propelled forward blood flow.
- The following drugs are used for CPR:
  - Epinephrine: 0.01 mg/kg IV or 0.03–0.1 mg/kg IT. This dose may be repeated every 3–5 minutes.
  - Vasopressin: 0.2–0.8 U/kg IV or 0.4–1.0 U/kg IT.
  - Atropine: 0.04 mg/kg IV or 0.08 mg/kg IT with saline.
  - Lidocaine: 2–4 mg/kg IV.
  - Sodium bicarbonate: 0.5 mEq/kg IV.

DEFIBRILLATION

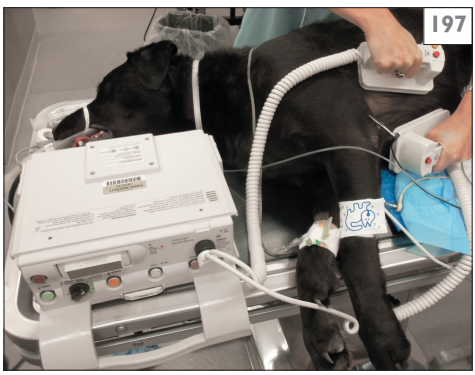
- Ventricular fibrillation should be identified and the patient defibrillated as early as possible. A defibrillator is needed (**196**). With the patient in right or left lateral recumbency, one paddle can be placed on the sternal side of the chest wall and the other paddle can be used on the upper side of the chest wall, and gel applied to both paddles (**197**).



195 The person performing cardiac compression should stand against the back of the animal to prevent the animal sliding away from the center of the compression. The compression is likely to be most effective with the heel of the palm against the animal's chest directly above the heart.

196 A defibrillator. Two external paddles are shown, one to be used on the sternum (marked sternum) and the other on the apex (the metal side facing) of the animal. The metal portion of the paddle comes in contact with the animal and the conduction gel should be applied to the metal portion. Note the power button (green color) and the energy selection dial (5–360 joules) on the defibrillator. The fire button used to discharge the energy is located on the paddles. When firing the electrical shock to the patient, both buttons must be pressed simutaneously.

- How much energy should be used? 50 joules for small dogs and cats; 100 joules for medium dog; and 200 joules for large dogs. (**Note:** One joule is defined as the amount of work or energy exerted when a force of one Newton is applied over a displacement of one meter. One joule is the equivalent energy of one watt of power radiated or dissipated for one second.)
- One shock should be administered rather than the three successive shocks previously recommended, with chest compressions immediately resumed for 2 minutes before reassessing the cardiac rhythm and administering any additional shocks.
- The energy may be doubled if the first shock is not effective in converting the fibrillation.
- When the defibrillator is discharged, a warning of 'clear' must be announced to stop contact with the patient (and anything connected to the patient) and protect personnel from being shocked.



197 With the patient in right or left lateral recumbency, one defibrillator paddle can be placed on the sternal side of the chest wall and the other paddle on the upper side of the chest wall. Gel is applied to the paddles. All personnel should stay clear from the animal and the table while the defibrillator is being discharged to avoid being shocked. Note the fire button (red color) located on the top of each paddle.

SOME CONSIDERATIONS REGARDING CARDIOPULMONARY RESUSCITATION

- One person alone cannot perform successful CPR.
- Venous access should be established as soon as possible, but not before airway management, ventilation, and chest compressions.
- A 'shock dose' of crystalloids (90 ml/kg/hour for dogs and 45 ml/kg/hour for cats) is only recommended if the patient is hypovolemic before cardiopulmonary arrest. In a euvolemic arrested patient, the recommended dose (based on a 2008 publication) is a 20 ml/kg bolus for dogs and a 10 ml/kg bolus for cats using crystalloid fluids. Excessive IV fluids during CPR to euvolemic patients decreases coronary perfusion pressure.
- The ABC (airway/breathing/compressions) protocol of CPR has been followed for many years, but CPR success rates remain low. Controversy exists over the best way to perform CPR and this subject will remain open to debate until outcomes are dramatically changed by the way it is performed. There is some evidence to suggest that cardiac compressions should be done first, rather than airway establishment.
- The patient must constantly be assessed during the procedure. Helpful monitoring equipment includes ECG, capnography, blood pressure monitors, and pulse oximetry.
- Remember that a capnometer may not be useful for assessing intubation in an arrested patient, as circulation is required to present CO_2 to the lung. The presence of $ETCO_2$ (>15 mmHg) is an indication of effective cardiac compressions.
- Most patients will require some form of supportive care following arrest, depending on the severity of the arrest and the success of the resuscitation.

Further reading

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