

# CPR: what are the current recommendations?

Kathryn Marguerite Pratschke MVB MVM CertSAS DipIECVS MRCVS  
Small Animal Surgical Specialist, Stirling, UK

## SYNOPSIS

In December 2005, the American Heart Association (AHA) published new guidelines for cardiopulmonary resuscitation (CPR) in humans for the first time in five years. Key points in the new guidelines included a greater emphasis on chest compressions, avoidance of excessive ventilation rates and immediate resumption of compressions after a single defibrillation. Many of the recommendations were based on small animal research, suggesting they may be pertinent to the veterinary patient. This article reviews the current evidence-based recommendations for CPR in veterinary patients.

## INTRODUCTION

Recently, the Reassessment Campaign on Veterinary Resuscitation (RECOVER) completed a critical review of the information available on resuscitation medicine with a focus on how this could apply to clinical veterinary medicine. From this, they generated a set of (reasonably) evidence-based, updated consensus guidelines for CPR in veterinary patients in 2012 (*Journal of Veterinary Emergency and Critical Care*, volume 22).

Figures for survival to discharge from hospital following cardiopulmonary arrest (CPA) in veterinary patients range from 3-6 per cent in dogs and 2-10 per cent in cats.<sup>1</sup> A patient who arrests under anaesthesia is thought to have an improved chance of survival,<sup>1</sup> although the main supporting evidence does come from a referral hospital population where patients were comprehensively instrumented and monitored.

## BE PREPARED

Quick recognition of an arrest is crucial. Any patient that becomes acutely non-responsive should be rapidly assessed for airway, breathing and circulation (ABC). Pulse palpation is not a reliable method of confirming CPA and should not be relied on.<sup>2,3</sup> If there is any doubt whether a patient has arrested, current recommendations are that CPR should be started without delay.<sup>4</sup>

Training should be provided for every member of staff that might have to participate in CPR. Experience shows that regular revision is necessary to reinforce the basics and ensure that updates are incorporated. Some form of hands-on practice is strongly recommended for key skills, including chest compressions, passing an endotracheal tube and starting an electrocardiogram (ECG).

Responsibility for checking/maintaining the crash cart/

box should be clearly assigned, taking account of holidays/ absences. The frequency with which the crash cart/box needs to be checked depends on the individual practice, as well as how quickly the vets learn not to raid the crash cart when they want something!

It is a good idea to have a practice policy of confirming resuscitation status at admission, particularly if the owner wants a 'DNR' (do not resuscitate) status. This is especially important where the patient has a condition that can predispose to arrest, such as sepsis, cardiac failure, coagulopathies, toxicities or traumatic brain injury and a quick decision may need to be made.



**Figure 1: The crash cart should be mobile and well-stocked with everything clearly labelled and easily accessible in order to reduce time-wasting during a CPR event.**

## BASIC LIFE SUPPORT

This involves effective chest compressions, providing ventilation, and includes the use of external defibrillation. It is very important to do these basics well – they are the only aspects of CPR proven to be effective for treatment of a CPA.<sup>3</sup> So which to prioritise? Ventilation in the absence of blood flow, ie. ventilation without chest compression, is of little benefit to the patient, and the greater the delay in starting chest compressions, the worse the outcome in general. However, chest compressions without ventilation mean hypoxia and hypercapnia, both of which decrease the chance of successful return of spontaneous circulation (ROSC). Although human CPR algorithms tend to prioritise compressions over ventilation, the majority of dogs and cats suffer non-cardiac origin CPA, so RECOVER guidelines

suggest that early intubation and ventilation should be an essential early part of the process.<sup>5</sup>

## AIRWAY AND BREATHING

First, ensure there is no obstruction from vomitus, blood or froth, and suction/swab clear if necessary. A laryngoscope should be used to place an endotracheal (ET) tube, as excessive manipulation of the epiglottis from repeated efforts to intubate can induce vagal bradycardia.<sup>6</sup> Once the tube is in, confirm correct placement, ie. airways, not digestive tract.

If an ET tube is not available, and the airways are not obstructed, then mouth-to-snout ventilation (much loved by the media) can be employed. To do this, the patient's mouth is held firmly shut with one hand, the head and neck extended to open the airways as much as possible, the ventilation volunteer makes a seal over the nares with their mouth and blows firmly to inflate the chest. Chest compressions cannot be performed simultaneously with mouth-to-snout so 30 compressions should be delivered, followed immediately by two short breaths, then a two-minute cycle alternating compressions and ventilations at a 30:2 ratio. Mouth-to-snout is mentioned in one sentence in a case report of a dog with traumatic cervical injury: "The dog had been given artificial respiration by use of a mouth-to-snout technique immediately after the trauma until it was brought to a local emergency hospital,"<sup>7</sup> but there are no other studies evaluating or confirming the efficacy. As it gives you no protection against aspiration, and requires intermittent cessation of chest compressions, mouth-to-snout should only be used if intubation is not possible due to lack of equipment or suitably trained personnel.

Doxapram has been used in the past as a respiratory stimulant. This is no longer recommended as it simultaneously increases cerebral oxygen requirements while decreasing the cerebral blood flow necessary to feed the demand, particularly in neonates.<sup>8</sup> The Jen Chung (GV26) point has been suggested for patients in respiratory, but not cardiac, arrest; this involves twirling a 25g 5/8-inch needle inserted into the bone in the nasal philtrum at the ventral aspect of the nares.<sup>9</sup> Information about this technique is largely anecdotal.

Ventilations should be provided at a rate of 10-12 breaths/minute at airway pressures  $\leq 20$ cmH<sub>2</sub>O (tidal volume 10ml/kg), with a one-second inspiratory time. Each breath should cause visible rising of the chest wall, followed by normal relaxation. In patients with pre-existing hypoxia or severe pulmonary disease, it may be beneficial to increase to 12-15 breaths per minute, but previous practices involving 20-24 breaths/minute are no longer recommended as they may adversely affect cardiac venous return and coronary perfusion.

## CIRCULATION

The aims of chest compressions are:

1. Restore blood flow to the lungs – CO<sub>2</sub> elimination and oxygen uptake
2. Deliver oxygen to tissues – restore organ function and

metabolism.

Manual CPR was developed so as to actively pump blood from the chest and vital organs during chest compressions; a period of relaxation between compressions allows venous return. In this way, chest compressions can ensure adequate myocardial, as well as cerebral, blood flow.

*External chest compressions:* continuous, uninterrupted compressions are essential, as a lack of chest compressions is positively associated with increasing mortality.<sup>6,10</sup> Recent studies in humans suggested that interruptions for attempted defibrillation, securing/checking the airway, placing IV catheters, drug administration and ECG evaluation mean chest compressions cease for up to 40-50 per cent of the overall CPR time without the rescuers realising.<sup>11-13</sup> External chest compressions work via the thoracic pump theory in humans and medium-large dogs, and cardiac pump theory in smaller patients (Table 1) – this dictates correct placement of your hands during effective CPR (Table 2). Currently, there is evidence neither for, nor against, the efficacy of interposed abdominal compressions in veterinary patients. Correct posture is important for the person carrying out chest compressions – shoulders directly above the hands with the elbows locked. This posture engages the core muscles rather than relying solely on biceps and triceps that will fatigue more readily. It also reduces the risk of leaning on the patient's chest between compressions. If possible, switch the compressor every two minutes to maintain adequate force and rate – if you are doing them right, then chest compressions are tiring!<sup>10,16</sup> The appropriate rate for veterinary patients is 80-100 compressions per minute at a 1:1 ratio between compression and relaxation.

*Internal cardiac massage:* previously, internal cardiac massage was suggested for penetrating chest wounds, thoracic trauma with rib fractures, pleural space disease, diaphragmatic rupture, pericardial effusion, haemoperitoneum, intraoperative sudden arrest and failure to achieve adequate circulation within two to five minutes of external compressions, especially in dogs above 20kg bodyweight.<sup>17-19</sup> However, there is no good evidence supporting the use of open chest resuscitation and current recommendations are to only consider it where there is significant intra-thoracic pathology (eg. tension pneumothorax) but not for routine CPR.<sup>5</sup>

## ADVANCED LIFE SUPPORT

Advanced life support includes monitoring, drug therapy and electrical defibrillation.

### MONITORING DURING CPR

Many commonly used monitoring devices are of limited use during CPR because of their susceptibility to motion artefact and the likelihood that decreased perfusion and altered pulse quality will compromise accuracy. Pulse oximetry and indirect blood pressure monitors, including Doppler and oscillometric devices, are not useful until you have ROSC. The two most useful tools during CPR are an ECG and capnograph.<sup>3</sup> The ECG will be affected by motion

<b>Cardiac pump theory<sup>14</sup></b>	When the ventricles are directly compressed the increased pressure opens the pulmonic and aortic valves, thereby providing blood flow to the lungs and the tissues. The elastic properties of the rib cage allow recoil of the chest between compressions, creating negative intra-thoracic pressure to allow re-filling of the ventricles before the next compression.
<b>Thoracic pump theory<sup>15</sup></b>	External chest compressions increase overall intra-thoracic pressure, which forces blood from intra-thoracic vessels into the systemic circulation; the heart acts as a passive conduit.

**Table 1: Cardiac and thoracic pump theory in external chest compressions.**

Patient type	Patient position	Hand placement
Medium-large dogs (thoracic pump theory)	Lateral recumbency	Place your hands over the widest part of the chest, one hand placed on top of the other and both hands parallel so that even pressure is applied to the chest wall.
Dogs with deep, narrow chest conformation, eg. greyhounds, lurchers (cardiac pump theory)	Lateral recumbency	Hands as before, but placed directly over the heart.
Smaller patients, 7-10kg or less (cardiac pump theory)	Lateral recumbency	Depending on patient size either use two hands as described for greyhounds/lurchers, or one hand squeezing the chest from opposing sides.
Brachycephalic breeds, eg. Bulldogs	Dorsal recumbency	Place your hands over the sternum at the level of heart, similar to humans. NB: these breeds tend to have poor chest compliance so considerable compression force may be required for effective compressions.

**Table 2: Hand placement and patient position in external chest compression.**

artifact and cannot be interpreted properly during ongoing chest compressions, but an accurate rhythm diagnosis is essential to help decide drug and defibrillation therapy. There are four rhythms commonly associated with cardiac arrest: asystole, ventricular tachycardia (VT), ventricular fibrillation (VF) and pulseless electrical activity (PEA). Capnography (ETCO<sub>2</sub>) gives an indirect measure of cardiac output by reflecting pulmonary perfusion and gas exchange.

## DRUG THERAPY

Ideally, a central line should be used for administering medications during CPR. In reality, this will rarely be feasible in veterinary patients, so a peripheral IV catheter is next best, followed by intraosseous (IO) and tracheal administration last.<sup>3,20</sup> Medications given via peripheral catheter should be given as a bolus injection followed by a fluid bolus and, if possible, raise the extremity for 10-20 seconds. Continue chest compressions uninterrupted for at least two minutes after you give any medication before stopping to check the ECG, as it will take one to

two minutes for medications to reach general circulation. Sites for IO administration include the tibial crest, humeral tubercle, and femoral trochanteric fossa. With the right equipment this can be a good option, but may not always be practical.

## SPECIFIC MEDICATIONS

### Vasopressors (epinephrine, vasopressin)

Even with effective chest compressions, only 25-30 per cent of the normal cardiac output is likely to be achieved. In order to prioritise cardiac and cerebral blood flow in the face of this reduced output, high peripheral vascular resistance is necessary.<sup>5</sup> Vasopressors are therefore recommended regardless of the arrest rhythm, as they're not being used for the arrhythmia per se, but to help maintain central blood flow. With epinephrine, low doses are recommended initially: 0.01mg/kg IV or IO every second cycle of CPR (one cycle = two minutes chest compressions). With prolonged CPR, the dosage can be increased to 0.1mg/kg IV or IO every second cycle. Epinephrine can be administered via the tracheal route, at 2-2.5 times the above dosages, but the efficacy is less certain and it must be diluted at least 1:1 with sterile water/isotonic saline. Vasopressin is an alternative pressor peptide that can be used interchangeably with epinephrine every second cycle of CPR. It has some potential benefits over epinephrine in terms of cerebral perfusion and preferential shunting of blood to the central nervous system (CNS) and heart. It also maintains efficacy in an acidic environment, such as follows CPA, where epinephrine and other catecholamines can be adversely affected. The dosage rate is 0.2-0.8U/kg IV/IO or 0.4-1.2U/kg per trachea.

### Atropine

Atropine has been extensively studied in CPA situations, but there are only a few studies showing a beneficial effect.<sup>3,21,22</sup> It can be administered at a dose of 0.04mg/kg IV/IO or 0.08mg/kg per trachea, and is most likely to be helpful in bradycardiac arrests or situations of suspected high vagal tone.

### Anti-arrhythmic drugs (amiodarone, lidocaine)

The anti-arrhythmic drugs have been extensively studied in experimental models and clinical trials in people. In a recent meta-analysis of the data, however, only amiodarone showed consistent benefit.<sup>23</sup> Its use is suggested in pulseless ventricular fibrillation (VF)/ventricular tachycardia (VT) that is refractory to electrical defibrillation, using a dose of 2.5- 5.0mg/kg IV/IO over 10 minutes, with one repeat dose at 2.5mg/kg IV after three to five minutes. Allergic reactions, including hypotension and anaphylaxis, have been seen in dogs so very close monitoring is warranted. Whether lidocaine is of benefit is more difficult to determine from the available evidence, with findings both for and against. However, given the poor prognosis for patients in refractory VF/pulseless VT, if amiodarone is not available and electrical defibrillation has either failed or is not available, lidocaine should be considered. This

is administered at 2-4mg/kg slow IV/IO in dogs. It should be used cautiously, if at all, in cats (high risk of toxicity, especially CNS) at a significantly lower dose of 0.2mg/kg IV/IO. There is currently no compelling evidence to support the use of magnesium sulphate and it is not recommended for routine CPR.<sup>5</sup>

#### Reversal agents (*naloxone, atipamezole, flumazenil*)

Of the various reversal agents, only naloxone has actually been assessed in a CPR scenario. Although evidence of efficacy is limited, it can be given at 0.04 mg/kg IV/IO if opioid toxicity is suspected or opioids were recently administered. Other available reversal agents, including flumazenil (0.01 mg/kg IV/IO) for benzodiazepines, and atipamezole (0.1 mg/kg IV/IO) for alpha2-agonists, are of unknown efficacy.<sup>5</sup>

#### Corticosteroids

The potential risks far outweigh the potential benefits; corticosteroids are not recommended for CPR.

#### Sodium bicarbonate

Metabolic acidosis arises with CPA, therefore it may be tempting to administer an alkalinising agent such as sodium bicarbonate. However, the available evidence shows that its use early in CPR is counter-productive, and current recommendations are to reserve it for patients with prolonged CPA (10-15 minutes), documented severe acidemia (pH <7.0) of known metabolic origin, pre-existing severe metabolic acidosis, overdose of tricyclic antidepressants or severe hyperkalaemia.<sup>6</sup> The dose for use is 0.5-1mEq/kg, diluted, given once IV.

#### Calcium

Calcium is an essential electrolyte, and patients with prolonged CPA often develop hypocalcaemia; however, the available evidence shows that routine use of calcium during CPR is not beneficial.<sup>5</sup> In those rare cases requiring treatment of calcium channel blocker toxicity, severe hyperkalaemia or a documented ionised hypocalcaemia, 10 per cent calcium gluconate can be given at 0.5-1.5ml/kg slowly IV. Remember – the serum total calcium is NOT an accurate reflection of ionised calcium levels.<sup>5,6</sup>

#### Intravenous fluid therapy

Contrary to popular belief, aggressive intravenous fluid therapy (IVFT) during CPR is not always beneficial. Indeed, it may be harmful if patients were euvoalaemic before arrest through an adverse effect on coronary artery perfusion pressure.<sup>24,25</sup> Although CPR-specific evidence for veterinary patients is lacking, from first principles it seems logical that hypovolaemic patients will benefit from appropriate fluid resuscitation, tailored to physiological variables rather than a set number, but those that are euvoalaemic should have more cautious IVFT.

## DEFIBRILLATION

There is a large body of available literature regarding acute

cardiac arrest caused by VF which strongly supports early electrical defibrillation as the best option.<sup>26</sup> The earlier VF is detected, the more responsive it is to defibrillation. If the duration of VF is four minutes or less, chest compressions should be continued while the defibrillator is charged, and the patient defibrillated as soon as the defibrillator is ready. If, however, the duration of VF is more than four minutes, one full cycle of CPR should be performed first to allow time for some recovery in myocardial cell function.<sup>27</sup>

Defibrillators may be monophasic (delivers a current in one direction) or biphasic (delivers a current first in one direction, then the opposite direction). Biphasic defibrillators are preferred because: (1) they are more effective; and (2) a lower current is required, meaning less myocardial injury<sup>28</sup> (Table 3).

<b>Monophasic defibrillator</b>	<b>Initial dose 4-6J/kg, second dose may be increased by 50 per cent, but subsequent doses should not be further increased.</b>
<b>Biphasic defibrillator</b>	<b>Initial dose 2-4J/kg, second dose may be increased by 50 per cent, but subsequent doses should not be further increased.</b>

**Table 3: Dosage for monophasic and biphasic electrical defibrillators.**

Alcohol, ultrasound gel, etc. should not be used in an effort to improve contact, as they are non-conductive; in fact, alcohol on the fur is a fire hazard.<sup>5</sup> Ideally, the patient should be in dorsal recumbency with the paddles on each side of the chest wall, level with the heart. If using defibrillator patches rather than paddles, fur must be clipped off first.<sup>5</sup>

It is essential to ensure no harm comes to the personnel involved in CPR. The person administering the shock should not be in direct contact with anything connected to the patient, including the table and ECG leads. They should clearly announce their intent to defibrillate ('clear') and then check visually that everyone really is clear before shocking. Older guidelines suggested giving three stacked shocks but current advice is to administer a single shock followed by immediate resumption of chest compressions for at least two minutes before you give a second shock.

#### Precordial thump

The precordial thump, a film and TV favourite, was first described in 1969.<sup>29</sup> More recent studies evaluating the technique have not found any strong supportive evidence,<sup>30-32</sup> so current guidelines are to only consider this where electrical defibrillation is not available<sup>5</sup> (which, in reality, is the situation in many veterinary practices).

## POST-RESUSCITATION CARE

Unfortunately, successful resuscitation does not stop at successful CPR. A sepsis-like syndrome can follow successful resuscitation in people, caused by global ischaemia and reperfusion injury. Approximately two-thirds of those initially successfully resuscitated will subsequently die.<sup>33</sup> One study in the veterinary literature suggested that 83 per cent of patients initially resuscitated may not survive to hospital discharge.<sup>1</sup>

There is no 'standard' protocol for these patients, because there is such wide variation in the degree of anoxic brain injury, post-ischaemic myocardial dysfunction, systemic response to ischaemia and reperfusion, and precipitating abnormalities.<sup>34</sup> Precipitating diseases must still be addressed, general supportive care and monitoring provided. Adequacy of ventilation can be monitored with arterial blood gas analysis or ETCO<sub>2</sub>, while pulse oximetry can be used to monitor oxygen saturation, aiming for 94-96 per cent.<sup>6</sup> Haemodynamic status should be supported through the use of appropriate IVFT and the judicious use of drug therapy. The brain is particularly sensitive to anoxic damage, so neuroprotective measures are important, including prompt treatment of seizures, avoiding hyperthermia, and avoiding anything that may increase intracranial pressure (sneezing from nasal canulae, for example).

### PROGNOSIS

Objective prognostic indicators for CPR patients are poorly studied in the veterinary literature, so we do not currently have an evidence-based answer to this question. For the time being, it will be necessary to continue assessing each case on its own merits and make an informed clinical judgement regarding prognosis.

### REFERENCES

- Hofmeister EH, Brainard BM, Egger CM et al. Prognostic indicators for dogs and cats with cardiopulmonary arrest treated by cardiopulmonary cerebral resuscitation at a university teaching hospital. *J Am Vet Med Assoc* 2009; 235(1): 50-7
- Dick WF, Eberle B, Wissner G et al. The carotid pulse check revisited: what if there is no pulse? *Crit Care Med* 2000; 28 (11 Suppl): N183-5
- Fletcher DF, Boller M. Updates in Small Animal Cardiopulmonary Resuscitation. *Vet Clin Small Anim* 2013; 43: 971-987
- Rittenberger JC, Menegazzi JJ, Callaway CW. Association of delay to first intervention with return of spontaneous circulation in a swine model of cardiac arrest. *Resuscitation* 2007; 73(1): 154-60
- Fletcher DJ, Boller M, Brainard BM et al. RECOVER evidence and knowledge gap analysis on veterinary CPR. Part 7: clinical guidelines. *J Vet Emerg Crit Care* 2012; 22(Suppl 1): S102-31
- Plunkett SJ, McMichael M. Cardiopulmonary resuscitation in small animal medicine: an update. *J Vet Intern Med* 2008; 22(1): 9-25
- Smarick SD, Rylander H, Burkitt JM et al. Treatment of traumatic cervical myelopathy with surgery, prolonged positive-pressure ventilation, and physical therapy in a dog. *J Am Vet Med Assoc* 2007; 230(3): 370-374
- Ebihara S, Ogawa H, Sasaki H et al. Doxapram and perception of dyspnea. *Chest* 2002; 121: 1380-1381
- Davies A, Janse J, Reynolds GW. Acupuncture in the relief of respiratory arrest. *NZ Vet J* 1984; 32: 109-110
- Maier GW, Tyson GS, Olsen CO et al. The physiology of external cardiac massage: high-impulse cardiopulmonary resuscitation. *Circulation* 1984; 70(1): 86-101
- ECC Committee, Subcommittees and Task Force of the American Heart Association. American Heart Association guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. *Circulation* 2005; 112: IV1-IV203
- Abella BS, Sandbo N, Vassilatos P et al. Chest compression rates during cardiopulmonary resuscitation are suboptimal: A prospective study during in-hospital cardiac arrest. *Circulation* 2005; 111:428-434
- Valenzuela TD, Kern KB, Clark LL et al. Interruptions of chest compressions during emergency medical systems resuscitation. *Circulation* 2005; 112: 1259-1265
- Kouwenhoven WB, Jude JR, Knickerbocker GG et al. Closed-chest cardiac massage. *J Am Med Assoc* 1960; 173: 1064-7
- Niemann JT, Rosborough J, Hausknecht M et al. Blood flow without cardiac compression during closed chest CPR. *Crit Care Med* 1981; 9(5): 380-1
- Malzer R, Zeiner A, Binder M et al. Hemodynamic effects of active compression-decompression after prolonged CPR. *Resuscitation* 1996; 31: 243-253
- Crowe DT Jr. Cardiopulmonary resuscitation in the dog: A review and proposed new guidelines (Part I). *Semin Vet Med Surg (Small Anim)* 1988; 3: 321-327
- Haskins SC. Internal cardiac compression. *J Am Vet Med Assoc* 1992; 200: 1945-1946

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19. Rieser TM. Cardiopulmonary resuscitation. Clin Tech Small Anim Pract 2000; 15: 76-81
20. Niemann JT, Stratton SJ, Cruz B et al. Endotracheal drug administration during out-of-hospital resuscitation: where are the survivors? Resuscitation 2002; 53(2): 153-7
21. Blecic S, Chaskis C, Vincent JL. Atropine administration in experimental electromechanical dissociation. Am J Emerg Med 1992; 10(6): 515-8
22. DeBehnke DJ, Swart GL, Spreng D et al. Standard and higher doses of atropine in a canine model of pulseless electrical activity. Acad Emerg Med 1995; 2(12): 1034-41
23. Ong ME, Pellis T, Link MS. The use of antiarrhythmic drugs for adult cardiac arrest: a systematic review. Resuscitation 2011; 82(6): 665-670
24. Voorhees WD 3rd, Ralston SH, Koungias C et al. Fluid loading with whole blood or Ringer's lactate solution during CPR in dogs. Resuscitation 1987; 15(2): 113-23
25. Yannopoulos D, Zviman M, Castro V et al. Intra-cardiopulmonary resuscitation hypothermia with and without volume loading in an ischemic model of cardiac arrest. Circulation 2009; 120(14): 1426-35
26. Link MS, Atkins DL, Passman RS et al. Part 6: electrical therapies: automated external defibrillators, defibrillation, cardioversion, and pacing: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Circulation 2010; 2: 122
27. Weisfeldt ML, Becker LB. Resuscitation after cardiac arrest a 3-phase time-sensitive model. J Am Med Assoc 2002; 288(23): 3035-8
28. Leng CT, Paradis NA, Calkins H et al. Resuscitation after prolonged ventricular fibrillation with use of monophasic and biphasic waveform pulses for external defibrillation. Circulation 2000; 101(25): 2968-7
29. Bornemann C, Scherf D. Electrocardiogram of the month. Paroxysmal ventricular tachycardia abolished by a blow to the precordium. Dis Chest 1969; 56(1): 83-4
30. Amir O, Schliamser JE, Nemer S et al. Ineffectiveness of precordial thump for cardioversion of malignant ventricular tachyarrhythmias. Pacing and Clinical Electrophysiology 2007; 30: 153-156
31. Haman L, Parizek P, Vojacek J. Precordial thump efficacy in termination of induced ventricular arrhythmias. Resuscitation 2009; 80(1): 14-16
32. Madias C, Maron BJ, Alsheikh-Ali AA et al. Precordial thump for cardiac arrest is effective for asystole but not for ventricular fibrillation. Heart Rhythm 2009; 6(10): 1495-1500
33. Peberdy MA, Kaye W, Ornato JP et al. Cardiopulmonary resuscitation of adults in the hospital: a report of 14720 cardiac arrests from the National Registry of Cardiopulmonary Resuscitation. Resuscitation 2003; 58(3): 297-308
34. Neumar RW, Nolan JP, Adrie C et al. Post-cardiac arrest syndrome: epidemiology, pathophysiology, treatment, and prognostication. A consensus statement from the International Liaison Committee on Resuscitation. Circulation 2008; 118(23): 2452-83

## Reader Questions and Answers

### 1: WHICH OF THE FOLLOWING STATEMENTS REGARDING CARDIOPULMONARY ARREST IS TRUE?

- A: Palpating a peripheral pulse is not a reliable way of identifying CPA
- B: Oxygen saturation less than 85 per cent on pulse oximetry is consistent with CPA
- C: CPR should not be started until it is confirmed that a CPA has definitely happened
- D: Cardiac origin arrest is common in dogs and cats, unlike in humans

### 2: WHAT VENTILATION RATE SHOULD BE USED FOR ROUTINE CPR IN VETERINARY PATIENTS?

- A: 20-24 breaths/minute
- B: 12-15 breaths per minute
- C: 10-12 breaths per minute
- D: 18-20 breaths per minute

### 3: WHICH OF THE FOLLOWING RHYTHMS IS COMMONLY ASSOCIATED WITH CARDIOPULMONARY ARREST?

- A: Ventricular premature contractions
- B: Ventricular fibrillation
- C: Atrial tachycardia
- D: Atrial fibrillation

### 4: WHICH OF THE FOLLOWING STATEMENTS IS TRUE IN RELATION TO DRUGS USED AS PART OF ADVANCED LIFE SUPPORT?

- A: Amiodarone can be used interchangeably with vasopressin, given every second cycle of CPR for three doses
- B: Epinephrine can be given via the trachea at up to 0.2mg/kg diluted in Hartmann's at least 1:1.
- C: Sodium bicarbonate can be considered if the patients serum potassium levels are 9mmol/L
- D: Vasopressin can be associated with anaphylaxis in cats so reduced dosage levels should be used

### 5: YOU ARE DEALING WITH A PATIENT THAT HAS BEEN IN VENTRICULAR FIBRILLATION FOR APPROXIMATELY FIVE MINUTES. WHAT IS THE MOST APPROPRIATE COURSE OF ACTION?

- A: Start treatment with lidocaine while the defibrillator is charging, then once ready, shock the patient
- B: Shock the patient once, then administer chest compressions for two minutes uninterrupted, then shock again
- C: Start chest compressions at a rate of 60/minute while the defibrillator is charging then shock the patient
- D: Continue chest compressions for two minutes without stopping then administer electrical defibrillation

ANSWERS: 1. A, 2. C, 3. B, 4. C, 5. D