Cardiopulmonary arrest (CPA) is an unfortunate but common occurrence in veterinary medicine. CPA is defined as sudden cessation of spontaneous and effective respiration and heartbeat. Veterinary staff should prepare for CPA by regularly reviewing techniques of cardiopulmonary cerebral resuscitation (CPCR; see box on page 781).

Reported survival rates after CPCR in humans are 5% to 20%. In-hospital survival rates are usually higher than out-of-hospital rates. In veterinary medicine, most reports have been focused on in-hospital CPCR, in which survival rates are even lower (i.e., 5% to 10%). One of the confounding factors in comparing studies of CPCR is the definition of survival. In many experimental studies, successful CPCR is described as return of spontaneous circulation (ROSC). In general, these success rates are much higher than in clinical trials, in which the definition of success is return to normal neurologic function or survival to discharge. Because the re-arrest rate of resuscitated animals can be as high as 60% to 70% and neurologic dysfunction may be seen as a complication of resuscitation, successful CPCR should be defined as survival to discharge with intact neurologic function.

Patient selection is one of the principal factors affecting the likelihood of successful resuscitation from CPA. Patients with end-stage disease, regardless of the organ system involved, are not good candidates for CPCR. Patient selection should be based on prior health of the animal and the reason for arrest (i.e., anesthesia versus multi-organ failure). In cases of critical illness, the possibility of arrest should be discussed with owners and advanced directives considered, such as “do not attempt resuscitation” (DNAR) orders.

ABSTRACT:
Cardiopulmonary arrest is a risk for any critically ill patient. Reported survival rates in humans and animals are poor. However, the veterinary team must understand the approach and techniques most likely to help patients survive. This article reviews the techniques of basic cardiopulmonary cerebral resuscitation.
PREPARATION

Successful resuscitation relies on teamwork, efficiency, and developing a CPCR protocol in the practice. Emergency equipment and drugs must be in an easily accessible location. Larger institutions may have a “crash cart” for their emergency equipment, but every practice should have an emergency kit with at least the basic supplies (Table 1). The emergency kit should be kept stocked at all times, and staff should be familiar with its contents. The kit should be mobile so that it can be used anywhere in the hospital, or a separate kit should be available in inaccessible locations. All staff members should frequently review the standard “ABCs” (i.e., airway, breathing, circulation) of CPCR. Recognizing animals at risk of CPA is vital to prevent a delay in resuscitation procedures. As may be expected, survival rates improve when ROSC is rapid.\(^2,7\)

Airway

When an animal is not breathing, the first priority is to assess the airway. Placing an appropriately sized, cuffed endotracheal tube is often the quickest and easiest way to establish and maintain a patent airway. Intubation not only permits delivery of a high concentration of oxygen but also reduces the risk of aspiration; in addition, when intravenous access is not already in place, the intratracheal route can be used to administer medications.

Practices should have a supply of endotracheal tubes in a variety of sizes and ancillary equipment, such as laryngoscopes, stylets, syringes, and gauze, in easily accessible locations. Suction should be available to clear pharyngeal or tracheal secretions, vomitus, blood, or edema fluid from the airway. When there is an upper respiratory obstruction, tracheostomy may be warranted. There are many methods of oxygen supplementation, but endotracheal and tracheostomy tubes have the advantage of connecting directly to an Ambu bag or anesthetic circuit to provide positive-pressure ventilation.

Correct endotracheal tube placement may be confirmed by directly visualizing the tube between the vocal cords or using an end-tidal carbon dioxide (ETCO\(_2\)) monitor. The presence of carbon dioxide (CO\(_2\)) in an exhaled breath (whether it is a spontaneous or positive-pressure breath) correlates with correct placement of the tube in the trachea.\(^8,9\)

Breathing

Once the airway has been secured, artificial ventilation can commence. An Ambu bag or anesthetic machine with a rebreathing bag may be used. Mechanical ventilators should not be used because they may be affected by pressure and volume changes caused by chest compressions during CPCR. One hundred percent oxygen delivery is recommended; however, delivery of room air (with a fraction of inspired oxygen of 21%) is acceptable when an oxygen supply is unavailable.\(^10\) If an anesthetic machine is used, the line must be purged of all anesthetic gases before connecting it to the patient. Continuous oxygen delivery is effective at maintaining hemoglobin saturation; however, after 5 to 6 minutes without ventilation, dogs have been shown to develop hypercapnia and severe respiratory acidosis. Chest compressions have been shown to provide adequate ventilation to prevent hypercapnia in experimental studies of canine CPA.\(^11\)

A respiratory rate of 8 to 12 breaths/min is indicated in most patients.\(^6,12\) Smaller animals may require higher respiratory rates. Some protocols have suggested rapid respiratory rates, applied simultaneously with each compression (i.e., 1:1 simultaneous compression ventilation) to increase intrathoracic pressure.\(^6,13,14\) However, 1:1 simultaneous compression ventilation tends to increase

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**After 5 to 6 minutes without ventilation, dogs have been shown to develop hypercapnia and severe respiratory acidosis.**
right atrial pressure more than aortic diastolic pressure, so coronary perfusion pressure and myocardial blood flow may actually decrease.\textsuperscript{15,16} Minute ventilation is increased by thoracic compression alone, without any change in the respiratory rate, and the risk of barotrauma with overventilation favors using lower respiratory rates.\textsuperscript{17}

A tidal volume of 10 to 15 ml/kg should be used and may be assessed visually as the point at which an animal’s chest first expands. Overinflation of the lungs during CPR may lead to barotrauma, pulmonary hemorrhage, and pneumothorax. Diseased lungs are easily overinflated. In animals with pulmonary or pleural compromise, it may be beneficial to use smaller tidal volumes and slightly increased respiratory rates.\textsuperscript{4,12} Pro-

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longed inspiratory times or maintaining high levels of positive end-expiratory pressures above 10 cm water (0.7 mm Hg) causes a sustained increase in intrathoracic pressure and leads to a significant reduction in venous return. In turn, cardiac output may decline and arterial blood pressure may fall. However, small amounts of positive end-expiratory pressure (i.e., 3 to 5 cm water [2.1 to 3.5 mm Hg]) prevent collapse of small airways and should improve oxygenation.\textsuperscript{12}

In veterinary medicine, respiratory arrest often precedes cardiac arrest. This is in contrast to humans, in which primary cardiac arrest is common because of the high incidence of myocardial infarction and cardiovascular disease. Respiratory arrest has a more favorable prognosis than does cardiac arrest.\textsuperscript{1} Because supplemen-

<table>
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<th>Table 1. CPCR Emergency Kit</th>
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<tr>
<td><strong>Equipment</strong></td>
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<tr>
<td><strong>Basic Kit</strong></td>
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<tr>
<td>Endotracheal tubes (all sizes)</td>
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<tr>
<td>Laryngoscope with various blade sizes and functioning light</td>
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<tr>
<td>Gauze tie</td>
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<tr>
<td>Ambu bag or easily accessible anesthetic machine with rebreathing bag; oxygen supply</td>
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<tr>
<td>Intravenous catheters (all sizes)</td>
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<td>Needles and syringes</td>
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<tr>
<td>Feeding tubes or long urinary catheters</td>
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<tr>
<td>Three-way stopcocks; extension tubing</td>
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<td>Scalpel blades; sterile surgical kit</td>
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<td>Epinephrine</td>
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<td>Atropine</td>
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<td>Naloxone</td>
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<tr>
<td>Electrical defibrillator</td>
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<td>Central venous catheters</td>
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<tr>
<td>Intraosseous catheters or bone marrow needles</td>
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<tr>
<td>Angiocatheters</td>
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<tr>
<td>Thoracostomy tubes</td>
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<td>Amiodarone</td>
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<td>Calcium gluconate</td>
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<td>Dopamine–dobutamine</td>
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<td>Lidocaine</td>
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<td>Magnesium chloride</td>
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<td>Sodium bicarbonate</td>
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<td>Vasopressin</td>
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<td>Isotonic crystalloid fluids</td>
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<td>Synthetic colloid</td>
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<td>Hypertonic saline</td>
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tal oxygen and positive-pressure ventilation may be sufficient for resuscitation. However, respiratory arrest can lead to cardiac arrest because prolonged hypoxemia and a combination of respiratory and metabolic acidosis result in cardiac dysfunction.

**Circulation**

Chest compressions are fundamental to CPCR. The goal of compressions is to generate adequate blood flow to reach the target organs, which are the myocardium via the coronary arteries and the brain via the cerebral arteries. Chest compressions use either the direct “cardiac pump” or the indirect “thoracic pump” technique to promote forward blood flow. The goal in humans is to achieve a compression rate of 80 to 100 bpm. In veterinary patients, the operator should maintain 80 to 120 compressions per minute, with equal time for compression and decompression (i.e., a 50% compression–decompression cycle). Because ROSC rates decline when chest compressions are interrupted, it is not necessary to stop compressions to deliver a positive-pressure breath.

**Cardiac Pump**

The cardiac pump compression technique should be used in cats and small dogs (i.e., <15.4 lb (<7 kg)). The operator’s hand should be placed directly over the apex of the heart, with the thumb on one side of the chest and the fingers on the other (Figure 1). A two-handed technique may also be used (Figure 2). Each thoracic compression should cause a direct cardiac compression. This technique can be performed with the patient in lateral or dorsal recumbency. The operator should concen-
trate on speed of compressions rather than depth. In most cases, the chest wall needs to be compressed only 1 to 2 cm (i.e., one-fourth to one-third the depth of the thoracic cavity).\textsuperscript{23}

**Thoracic Pump**

The thoracic pump technique should be used in animals weighing more than 15.4 lb (7 kg). Compressions should be applied at the widest part of the chest with the patient in lateral recumbency or on the sternum with the patient in dorsal recumbency and to a depth of one-fourth to one-third of the width of the thoracic cavity (Figure 3). It has been suggested that large dogs be placed in dorsal recumbency for CPCR because intrathoracic pressure is maximized when compressions are administered over the sternum.\textsuperscript{18,24} In practice, however, this may be a difficult position to maintain, especially in thin or deep-chested dogs. Excessive movement decreases the force of thoracic compressions.\textsuperscript{24} Recently, there have been suggestions that lateral recumbency is also suitable for CPCR in large dogs.\textsuperscript{4,6,13,14,25}

Chest compressions delivered by the thoracic pump technique increase intrathoracic pressure, which closes the thin-walled intrathoracic veins. When combined with one-way valves, this prevents retrograde blood flow. In contrast, thick-walled arteries tend to remain open, facilitating forward blood flow. When the compression is released and the veins reopen, a pressure gradient develops across the thoracic inlet, increasing vascular flow from extrathoracic to intrathoracic veins and, in turn, enhancing preload. Blood is also moved from the pulmonary vasculature into the heart.\textsuperscript{26–28} The coronary arteries are perfused during diastole, so aortic pressure must remain higher than right atrial pressure to ensure myocardial perfusion. Success of CPCR is greatly affected by myocardial blood flow, which, when decreased, can lead to myocardial ischemia, prolong fibrillation, and impair myocardial performance in the postresuscitative state. Coronary perfusion pressures are defined as the difference between aortic and right atrial pressures in mid-diastole. In dogs, normal values range from 70 to 100 mm Hg. Coronary perfusion pressure less than 15 to 20 mm Hg has been associated with decreased survival in dogs.\textsuperscript{29}

**Abdominal Compressions**

Alternating abdominal compressions with chest compressions may improve forward flow, increase mean
arterial pressures, and enhance coronary artery perfusion.\textsuperscript{30,31} (Figure 3). With this technique, there is a risk of blunt injury to abdominal organs, although this is reportedly minimal in canine models.\textsuperscript{32} Abdominal compressions enhance venous return and thereby increase preload. Compression of the abdominal aorta also helps to provide retrograde aortic flow and increase aortic diastolic pressure. This increases coronary perfusion pressure and myocardial blood flow.\textsuperscript{26,28} Abdominal compressions may be contraindicated in cases of significant abdominal organomegaly, disease, or trauma. Abdominal binding, which involves placing a wrap on the hind legs and abdomen to provide compression, has been described in dogs in an attempt to direct blood flow cranially. However, this simultaneously increases both aortic diastolic pressure and right atrial pressure, so myocardial blood flow does not improve.\textsuperscript{33}

**Internal Cardiac Massage**

One of the earliest decisions to make in the course of CPCR is whether to proceed to internal cardiac massage. In cases of chest wall, pleural space, or pericardial disease or trauma, internal cardiac massage is indicated immediately. In very large or obese animals or those that do not respond to external cardiac compressions, open-chest CPCR is also indicated.\textsuperscript{4} Many authors have suggested that internal cardiac massage is appropriate if the patient has not responded to external compressions within 10 minutes; others have recommended a more aggressive approach and earlier institution of internal cardiac massage.\textsuperscript{1,6,13,14,18,34} If cardiac arrest remains untreated for more than 20 minutes, even open-chest CPCR does not improve survival.\textsuperscript{35} Internal cardiac massage can improve carotid and aortic blood flow and increase cardiac output in CPCR by at least 50% over external compressions.\textsuperscript{34,35} There is a risk of trauma to the heart, lungs, and surrounding tissues, which may cause complications after resuscitation, and the invasiveness of the technique limits its usefulness outside the hospital. No veterinary studies have shown an improved clinical outcome using internal cardiac massage.

If the decision is made to proceed with internal cardiac massage, the patient should be placed in right lateral recumbency and the chest wall quickly clipped over the fourth to fifth intercostal space on the left side. This area can be swabbed with iodine solution before incision. A vertical incision from the top of the scapula to 3 to 5 cm above the sternum can be made with a scalpel, incising the skin, subcutaneous tissues, and cutaneous trunci muscle. The intercostal muscles can be further dissected with scissors, taking care to avoid the intercostal vessels and nerve, which run along the caudal aspect of the rib. The pleura may be opened bluntly with closed scissors or the operator’s finger to avoid trauma to the intrathoracic structures, and then the incision can be extended dorsally and ventrally. The internal thoracic vessels should be avoided because they run subpleurally near the sternum.\textsuperscript{36,37} The ribs can be held apart with retractors and the operator’s hand (in sterile gloves) inserted into the chest cavity. The pericardium should be opened ventral to the phrenic nerve and reflected dorsally, allowing access to the heart. Direct cardiac compressions, with massage of the heart from apex to base, can then be instituted at 120 to 140 bpm, using a cupped hand rather than finger pressure, which may damage the heart muscle. Cross-clamping of the aorta has been described but is usually time-consuming and may be difficult in smaller animals.\textsuperscript{38} Digital compression of the aorta against the spine promotes cranial blood flow.\textsuperscript{32} In patients in which CPCR is successful, the thoracic cavity should be flushed with sterile saline and samples collected from the thoracic cavity for aerobic and anaerobic culture. Closure should be performed under aseptic conditions.

**ABC VERSUS CAB**

Recently, there has been a discussion in human medicine regarding whether CAB (circulation, airway, breathing) is a more useful acronym than ABC (airway, breathing, circulation) for CPA. This discussion was initiated by reports that bystander CPCR (i.e., out-of-hospital CPCR performed by untrained personnel) is often delayed because of concerns about contracting an infectious disease while performing mouth-to-mouth breath-
ing.²²,³⁹ Using the CAB protocol, chest compressions are instituted before establishing an airway or providing positive-pressure ventilation. It has been shown that within the first few minutes of CPA, blood is still well oxygenated; thus myocardial oxygen delivery is affected more by blood flow than by oxygen saturation.⁴⁰ Chest compressions provide some air movement in the lungs without additional ventilation; so if a rescuer is initially alone when commencing CPCR, compressions have a greater chance of providing both circulatory and some ventilatory support than does assisted ventilation.⁴¹,⁴² In addition, providing assisted ventilation, such as mouth-to-nose or mouth-to-mouth breathing, without first securing the airway has been associated with gastric insufflation and aspiration pneumonia.¹⁵ Artificial ventilation is indicated within the first few minutes after CPA because myocardial hypoxemia and hypercarbia develop, reducing the likelihood of successful resuscitation.⁴³,⁴⁴ The cause of arrest in veterinary patients often differs from that in humans. Humans more commonly have cardiac disturbances, whereas primary respiratory arrest is often seen in veterinary medicine, and providing artificial ventilation is paramount to successful resuscitation. In veterinary situations in which CPA is witnessed, ventilation and compressions are usually instituted simultaneously.

**MONITORING THE EFFECTIVENESS OF RESUSCITATION**

Once CPCR has been instituted, it becomes necessary to evaluate the effectiveness of the procedure. In many cases, clinicians attempt to palpate a peripheral pulse to assess efficacy of compressions. This method is not sensitive, especially when energetic compressions are being applied to a small animal, and the presence of a femoral pulse does not guarantee forward or arterial flow. There are no valves in the caudal vena cava, so retrograde flow from the vena cava to the femoral vein can occur during CPCR.⁸ Doppler ultrasonography may be used to assess blood flow, but it may also be affected by movement artifact.

Electrocardiogram (ECG) monitors should be attached to the patient. Much of the progression of CPA is monitored via ECG changes. However, reading the ECG requires cessation of chest compressions and therefore has an adverse effect on ROSC rates, so additional methods of monitoring should be used. ETCO₂ monitors can be easily attached to endotracheal tubes. The ETCO₂ reading can be useful in monitoring the progression of CPCR because ETCO₂ is linearly related
to stroke volume when ventilation is controlled. In low-flow states, many of the alveoli are not perfused, so CO₂ is unable to diffuse from the bloodstream into expired gas, and ETCO₂ measurements are low. As blood flow improves, more alveoli are perfused and more CO₂ is excreted. Success of CPRC has been correlated with higher ETCO₂ values in human and porcine models.⁴⁵-⁴⁷ Administering epinephrine may disturb the correlation between an increased ETCO₂ level and successful resuscitation; a higher incidence of survival in humans who experience CPA has been seen when the ETCO₂ level decreases after epinephrine administration than when there is no change in the ETCO₂ value. The mechanism for this is unknown.⁴⁸,⁴⁹

Blood gas measurements are a very useful monitoring tool during CPRC, although the values may seem paradoxical. Knowledge of the underlying pathophysiology helps with interpretation. In arrest situations, peripheral tissues continue to produce CO₂, which remains in the peripheral vascular beds because of low cardiac output and venous stasis. During CPRC, there is normal to increased ventilation of poorly perfused lungs (i.e., a high ventilation:perfusion ratio), so CO₂ levels in arterial blood are artificially decreased (respiratory alkalosis). Therefore, in low-flow states, such as CPA, venous blood gas measurements are more reflective of intracellular events in the peripheral tissue beds than are arterial measurements.⁵⁰,⁵¹

With restoration of spontaneous circulation and reperfusion of the peripheral vascular beds, CO₂ is washed out, creating transitory arterial hypercapnia and acidemia. During CPA, the myocardium also produces a disproportionate amount of CO₂.⁵²,⁵³ As a result, arterial blood gas measurements with low partial pressure of oxygen, low pH, and increased CO₂ during CPRC or immediately after resuscitation may actually reflect increased cardiac output and improved pulmonary perfusion.⁵¹,⁵⁴

CONCLUSION

Basic resuscitation techniques are fundamental to CPRC. All veterinary staff should be familiar with the ABCs of CPRC, and veterinary practices should have an emergency kit ready at all times. The likelihood of successful resuscitation after CPA is enhanced if animals at increased risk are identified early and the veterinary team is prepared to perform CPRC if required.

REFERENCES


4. Which statement regarding external chest compressions is incorrect?
   a. The chest should be compressed one-fourth to one-third of its depth.
   b. To promote maximal lung inflation and oxygen delivery, operators should stop compressions when a breath is being delivered.
   c. The operator should aim for 80 to 120 chest compressions per minute.
   d. There should be a 50% compression–decompression cycle.

5. In the initial phase of CPCR, commencing chest compressions may be more useful than establishing an airway because
   a. oxygen delivery is affected more by blood flow than by oxygen saturation during the first few minutes of arrest.
   b. intermittent mouth-to-nose ventilation is adequate for oxygen delivery.
   c. compressions have no effect on pulmonary ventilation.
   d. cardiac arrhythmias are more commonly the cause of arrest in veterinary patients than in humans.

6. ETCO$_2$ measurements can be used to evaluate the efficacy of resuscitation because
   a. the alveolar gas contains more CO$_2$ when blood flow from peripheral capillary beds is decreased.
   b. arterial blood contains more CO$_2$ when blood flow from the peripheral capillary beds is decreased.
   c. venous stasis increases the amount of CO$_2$ delivered to the lungs.
   d. ROSC increases the amount of CO$_2$ delivered to the alveoli.

7. Which statement regarding abdominal compressions is incorrect?
   a. Intermittent abdominal compressions combined with chest compressions increase coronary perfusion pressure.
   b. Intermittent abdominal compressions combined with chest compressions increase ROSC.
   c. Intermittent abdominal compressions increase aortic systolic pressure.
   d. Intermittent abdominal compressions increase venous return.

8. On which fundamental principle is CAB based?
   a. Improving circulation is the primary goal in veterinary CPR, and ventilation is a lower priority.
   b. Most veterinary patients that experience CPA have primary cardiac disease.
   c. Chest compressions provide some air movement, and when the first responder is alone, this may be the ideal technique for providing ventilation and circulation.
   d. Caregivers are at risk of infectious disease exposure when performing mouth-to-mouth resuscitation.

9. Evaluating the ECG during CPCR is difficult because
   a. of artifactual arrhythmias.
   b. of arrhythmias due to myocardial trauma.
   c. reading the ECG requires cessation of thoracic compressions.
   d. ETCO$_2$ monitors interfere with the ECG.

10. Which of the following is not an important aspect of the thoracic pump mechanism?
    a. Increases in intrathoracic pressure promote cardiac output.
    b. Flow is improved by direct cardiac compression.
    c. When a compression is released, there is an increase in blood flow from extrathoracic to intrathoracic veins.
    d. Increased myocardial blood flow improves survival.