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Enhancing post–cardiac arrest care for dogs and cats: a veterinary technician's guide to the RECOVER Initiative

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Abstract

Cardiopulmonary arrest, the acute cessation of ventilation and perfusion, can result from various clinical conditions in veterinary patients. Advances in CPR have improved the return of spontaneous circulation rates, but most patients rearrest within 24 hours, known as the post–cardiac arrest period. Ideal postresuscitation care, as seen in human medicine, reduces morbidity and mortality by focusing on cardiopulmonary stabilization, identifying the cause of cardiopulmonary arrest, neuroprotection, and preventing recurrence. In this narrative review, we will look at how care has evolved prior to and within the Reassessment Campaign on Veterinary Resuscitation guidelines while providing a clinical overview of the post–cardiac arrest period.

Keywords: ROSC, RECOVER, CPR, PCA, veterinary technician

Cardiopulmonary arrest (CPA) is defined as the acute cessation of ventilation and perfusion, leading to tissue oxygen deprivation and death.[1] Common causes in dogs and cats include trauma, critical illness, anesthetic complications, and respiratory distress.[1] While 30% to 40% of human patients survive after CPA and CPR, only 6% of dogs and 19% of cats survive to discharge.[2]

Around 40% to 50% of dogs and cats undergoing CPR experience a return of spontaneous circulation (ROSC), but most will experience a subsequent arrest, with even fewer surviving to discharge.[2] To improve survival rates, the first veterinary CPR guidelines were published in 2012 and updated in 2024 by the Reassessment Campaign on Veterinary Resuscitation (RECOVER) Initiative. These guidelines were based on a literature review and expert consensus to standardize CPR training and practice.[1]

Post–cardiac arrest (PCA) syndrome refers to the physiologic effects following successful resuscitation, including anoxic brain injury, myocardial dysfunction, and reperfusion injury.[3] The RECOVER PCA algorithm **(Figure 1)** outlines a systematic approach to evaluating ROSC patients, focusing on ventilation, hemodynamic support, and neuroprotection.[4]

A main goal in the PCA period is preventing rearrest. Patients should be hospitalized in facilities where frequent and close monitoring capabilities are available, as well as a team comfortable with caring for PCA patients. Continuous ECG should be utilized, as it may identify tachycardia, bradycardia, or arrhythmias that may precede CPA.[5] Frequent or continuous blood pressure (BP) monitoring—ideally, direct arterial BP (ABP) monitoring—should be utilized in these patients. Indirect measurements of BP are acceptable if direct monitoring is not possible.[5] These patients often require one-on-one nursing care due to hemodynamic, neurologic, ventilation, and oxygenation alterations.

Ventilation

Patients with ROSC after CPA often have alterations in ventilation. The quality and quantity of a patient's ventilation can affect cardiac output, hemodynamic stability, and brain health. Inadequate ventilation, and resulting hypercapnia, will contribute toward acidemia and may worsen hemodynamic instability. In addition, even minor changes to a patient's ventilation status will have a significant impact on their cerebral blood flow.[6] Hypercapnia may increase cerebral blood flow by causing cerebral arterial dilation, leading to increased intracranial pressure (ICP) in an already vulnerable brain.[6] Immediately after ROSC, the patient should be evaluated for adequate spontaneous ventilation, which should be based on effective chest excursions during ventilation and/or an acceptable carbon dioxide concentration. The effectiveness of ventilation should be assessed using the partial pressure of carbon dioxide in arterial blood ($PaCO_2$), partial pressure of carbon dioxide in venous blood ($P\bar{v}CO_2$), and/or end-tidal

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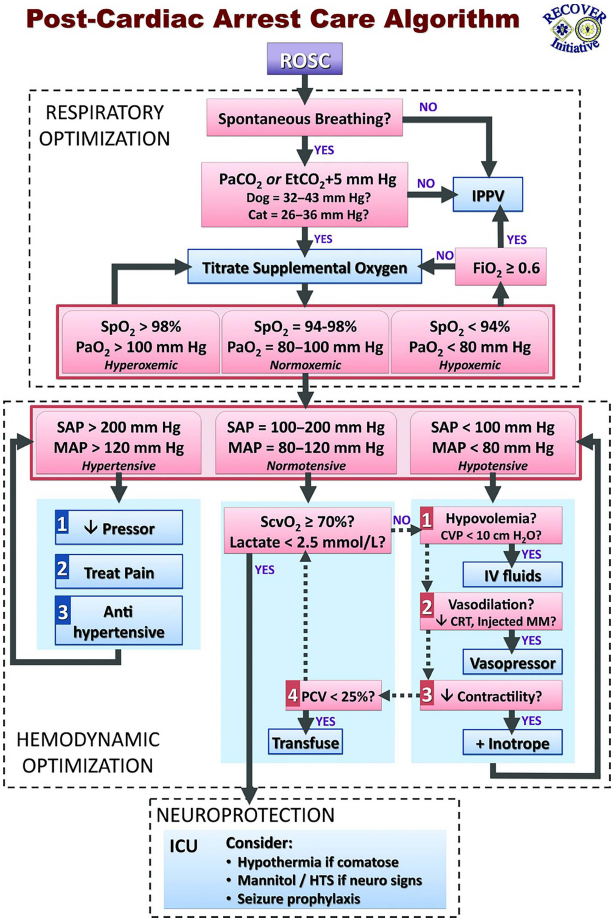

Figure 1—Reassessment Campaign on Veterinary Resuscitation post–cardiac arrest care algorithm that outlines a systemic approach to the return of spontaneous circulation (ROSC) patient and should be available to all veterinary professionals, ideally stored in the crash chart.[4] CRT = Capillary refill time. CVP = Central venous pressure. $ETCO_2$ = End-tidal carbon dioxide. FiO_2 = Fraction of inspired oxygen. HTS = Hypertonic saline. IPPV = Intermittent positive pressure ventilation. MAP = Mean arterial pressure. MM = Mucous membranes. SAP = Systolic arterial pressure. $ScvO_2$ = Central venous oxygen saturation. SpO_2 = Peripheral oxygen saturation of hemoglobin as measured by pulse oximetry. (Reproduced from 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 (San Antonio)*. 2012; 22(suppl 1):S102S131. doi:10.1111/j.1476-4431.2012.00757.x. Reprinted with permission.)[4]

carbon dioxide ($ETCO_2$). Assisted ventilation via intermittent positive pressure ventilation (IPPV) is indicated if these values are not being maintained within a normal range.

A normal $ETCO_2$ for a healthy dog and cat is typically 35 to 45 mm Hg. A normal $PaCO_2$ in dogs is 32 to 43 mm Hg, and in cats it is 26 to 36 mm Hg.[6] An arterial sample is the preferred method to measure carbon dioxide concentration and the animal's ventilatory efficacy, but if an arterial sample cannot be obtained, a venous sample can be used. It should be noted that a patient's $P\bar{v}CO_2$ and $ETCO_2$ are usually about 2 to 5 mm Hg higher than their $PaCO_2$. The *Reassessment Campaign on Veterinary Resuscitation Guidelines, Part 7: Clinical Guidelines* recommend keeping the patient at normocapnia during the PCA period. If the patient is breathing spontaneously but not meeting these guidelines or if apnea occurs, intervention is necessary. While the $PaCO_2$ or $P\bar{v}CO_2$ can only be intermittently monitored, the $ETCO_2$ should be continuously monitored in intubated patients to maintain normocapnia. A capnograph is arguably the most vital tool in a veterinary technician's tool belt during CPR and the PCA period.

Hyperventilation

If a patient's CO_2 levels ($PaCO_2$, $P\bar{v}CO_2$, or $ETCO_2$) fall below the reference range, it indicates hyperventilation. This condition is often a result of tachypnea but can also occur because of an increased tidal volume delivered during assisted ventilation. When hyperventilation occurs, there is a decreased amount of CO_2 in the tissue beds. This causes vasoconstriction, which leads to a decreased amount of blood and oxygen being delivered to vital organs, including the brain.[4] During CPR, blood flow and oxygenation to the brain are markedly compromised. While vasoconstriction occurs as a protective measure during the PCA period, vasoconstriction secondary to hyperventilation can be detrimental to the ROSC patient, leading to cerebral ischemia and decreased cardiac output.[7]

Pain, anxiety, anemia, hypoxemia, and metabolic acidosis can cause hyperventilation in the spontaneously ventilating patient. The underlying causes of metabolic acidosis should be identified and treated. Severe metabolic acidosis (pH < 7.0) can be treated with sodium bicarbonate,[6] although sodium bicarbonate is rarely used because of documented worse outcomes and metabolic derangements associated with its use.[5] Treatments of pain, anxiety, and anemia are discussed later in this review.

Hypoventilation

Hypoventilation occurs when CO_2 levels exceed the normal reference range. In post-ROSC patients, it can be harmful, increasing the risk of ischemic injury and worsening acidemia. Additionally, hypoventilation may lead to vasodilation and increased ICP. Hypoventilation is commonly seen when the respiratory rate is decreased but can also occur with lower tidal volumes. It is common for ROSC patients to have either absent or dysfunctional cerebral autoregulation, leading to hypoventilation.[8,9] Other causes of hypoventilation in the PCA period include respiratory depression secondary to sedatives or opioids, atelectasis, and pain from underlying disease or rib fractures.

Once hypoventilation is recognized, steps should be taken to enhance ventilation. Reversing sedatives or analgesics should be considered. Positioning the patient in sternal recumbency allows full lung expansion, reducing the risk of atelectasis. If $PaCO_2$, $P\bar{v}CO_2$, and/or $ETCO_2$ levels do not improve, assisted ventilation should be initiated. A study[10] published in 2005 revealed that cats that achieved ROSC and required

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IPPV had poor survival rates. Although more studies and data are still needed, the routine use of IPPV during the PCA period should be avoided unless clinically indicated. The use of assisted ventilation is appropriate for patients who are hypoventilating, are experiencing hypoxemia, require high oxygen concentrations to maintain normoxemia, or are at risk of respiratory arrest.[10] Since assisted ventilation should be used only when necessary, veterinary technicians should take other actions to improve the patient's ventilation status first.

Oxygenation

Adequate oxygenation, specifically normoxemia, is the ideal goal during the PCA period. Normoxemia is defined as peripheral oxygen saturation of hemoglobin as measured by pulse oximetry (SpO_2) of 94% to 98% or PaO_2 of 80 to 100 mm Hg. While monitoring and adjusting the patient's ventilation, it is vital to monitor the SpO_2 and/or the PaO_2 simultaneously. Peripheral oxygen saturation of hemoglobin as measured by pulse oximetry or PaO_2 readings higher than 98% or 100 mm Hg, respectively, indicate hyperoxemia, and oxygen supplementation should be decreased to achieve normoxemia.[11] The 2024 RECOVER guidelines recommend the use of SpO_2 monitoring with continuous or periodic monitoring of other perfusion parameters **(Table 1)**.[5] Peripheral oxygen saturation of hemoglobin as measured by pulse oximetry monitoring in veterinary patients can often be challenging because of multiple factors, such as skin or mucous membrane color, the presence or absence of fur, the presence of hypotension/poor perfusion, and arrhythmias. To achieve an accurate depiction of oxygenation, SpO_2 should be monitored in conjunction with perfusion parameters. An accurate SpO_2 reading will coincide with a matching heart rate when a pulse oximeter is utilized.

Hyperoxemia

Too much oxygen supplemented during the PCA period can be detrimental to the ROSC patient and potentiate PCA syndrome.[2,3] After ischemia occurs during CPR and in the PCA period, a large amount of oxygen free radicals is produced. Oxygen free radicals are unstable molecules that are very reactive and cause cellular damage and death. In human patients, hyperoxia during the early stages of ROSC has been observed to cause damage to postischemic neurons.[5] Although data are limited in veterinary patients, similar risks may exist and warrant caution.

Table 1—Normal perfusion parameters in dogs and cats.

Perfusion parameter	Normal reference range
Heart rate (beats/min)	80–120 dogs and 180–220 cats
Pulse quality	Strong distal pulses
Mucous membrane	Pink
Capillary refill time (s)	< 2
Mentation	Alert
Lactate (mmol/L)	< 2.5
Blood pressure (mm Hg systolic)	100–140

Hyperoxemia is defined as SpO_2 > 98% or PaO_2 > 100 mm Hg. Supplemental oxygen should be titrated or discontinued in the PCA period to achieve normoxemia. Oxygen should be titrated slowly while the patient's SpO_2, PaO_2, and perfusion parameters are assessed closely.[4]

Hypoxemia

Hypoxemia is defined as $SpO_2 \leq 94\%$ or $PaO_2 \leq$ 80 mm Hg. If hypoxemia is identified, supplemental oxygen should be increased until an adequate response is achieved. Increased supplemental oxygen can be accomplished by increasing the oxygen flow rate in an oxygen cage to oxygen concentrations of between 40% and 60%. When nasal cannulas are utilized, oxygen flow rates of between 50 and 150 mL/kg/min should be considered. Patient discomfort is often noted when flow rates exceed 100 mL/kg/min. The placement of bilateral cannulas to split the total flow rate can help reduce discomfort. When available, high-flow nasal oxygen should be utilized at 1 to 2 L/kg/min with oxygen concentration titrated between 40% and 100% to achieve the desired SpO_2. However, prolonged exposure to high concentrations of oxygen can lead to oxygen toxicity, which may result in pulmonary damage and should be carefully monitored. Finally, mechanical ventilation should be considered in patients who are unresponsive to conventional oxygen supplementation.

Hemodynamic Support

The hemodynamic system, which regulates the flow of blood through the arteries and veins, contains many elements, including heart rate, stroke volume, BP, cardiac output, and systemic vascular resistance. The RECOVER guidelines recommend having a set of end goals in mind when assessing a patient's hemodynamic status. Primary resuscitation endpoints include central venous oxygen saturation ($ScvO_2$) or lactate. Secondary endpoints include ABP, central venous pressure, PCV, and arterial oxygen saturation.[2]

Blood pressure is a very important hemodynamic monitoring parameter. In human pediatric care, hypotension occurring early after cardiac arrest is a poor prognostic indicator.[12] While evidence of this applying to veterinary medicine is lacking, the extrapolation from human studies should be cautiously considered. Direct ABP monitoring is the most accurate but invasive method to monitor BP. However, BP measurements can also be obtained with indirect methods (Doppler or oscillometric). Normotension should be maintained with a mean arterial pressure (MAP) of 80 to 100 mm Hg or a systolic arterial pressure (SAP) of 100 to 140 mm Hg.[4]

Hypotension

Hypotension, defined as SAP < 100 mm Hg or MAP < 80 mm Hg, in the PCA period is a common and detrimental finding in these patients. Post–cardiac arrest shock, characterized by hypotension and hypoperfusion requiring vasopressor support, occurs in about 70% of human patients with out-of-hospital

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cardiac arrest. Decreased BP leads to decreased tissue oxygen delivery, resulting in worsening ischemic injury. Hypotension is most commonly caused by 1 of 3 physiologic alterations: hypovolemia, vasodilation, or myocardial dysfunction.[12] Animals with hypotension often have other clinical signs of poor perfusion, including but not limited to tachycardia, weak peripheral pulses, pale mucous membranes, prolonged capillary refill time, cold extremities, hyperlactatemia, and altered mentation.

The mainstay for treating hypotension secondary to hypovolemia is fluid resuscitation. A fluid bolus of an isotonic crystalloid at a dose of 10 to 20 mL/kg in dogs and 5 to 10 mL/kg in cats can be delivered over 15 to 20 minutes.[13] Any time a fluid bolus is administered, the patient's perfusion parameters should be reassessed. If there is no response to the fluid therapy, it is likely that the patient is not hypotensive secondary to hypovolemia, and administration of additional fluids is unlikely to be helpful. If normotension is not achieved after fluid resuscitation is initiated, the patient's vascular tone should then be assessed.

Systemic inflammation, metabolic acidosis, and ischemic injuries that occur because of CPA, as well as the patient's underlying disease, may result in peripheral vasodilation, causing poor vascular tone, hypotension, and distributive shock. Patients with vasodilation will require a vasopressor to restore vascular tone and oxygen delivery. Norepinephrine, a catecholamine vasopressor, is the most utilized vasopressor during the PCA period. In a study[14] of human patients, the use of epinephrine in the PCA period was associated with a significantly higher all-cause hospital mortality and cardiovascular-specific mortality compared to the use of norepinephrine. Norepinephrine is short acting and thus administered as a constant rate infusion (CRI) after dilution according to the manufacturer's recommendations. A norepinephrine CRI should be administered at 0.1 to 2 µg/kg/min, IV, starting at the low end of the dose. Blood pressure should be monitored continuously or in 5- to 15-minute increments if direct ABP monitoring is unavailable. The CRI dose is then titrated up until BP improves.

Norepinephrine is less effective in acidemic states, so a higher dose may be required if initiated soon after ROSC. Norepinephrine is arrhythmogenic, and the patient should be monitored on ECG for the development of arrhythmias. The drug should ideally be administered through a central venous catheter, although human literature has described successful use of vasopressors through peripheral catheters with low rates of adverse effects.[15] Some human medicine studies[16] have shown that patients who did not achieve ROSC after 15 minutes or more of CPR may benefit from mild hypertension. Therefore, it is recommended to aim for mild hypertension, especially if they endured extended CPA.[16] For these patients, a MAP > 150 mm Hg during the first few hours of PCA could be beneficial and can be accomplished with the help of a vasopressor CRI. Other vasopressors, like vasopressin or dopamine, may be considered if norepinephrine is unavailable or if the patient is unresponsive to norepinephrine.

Myocardial dysfunction is another common reason for hypotension in the PCA period. During CPA, the heart may experience ischemic injury or myocardial damage. Additionally, the patient may have underlying myocardial disease, although this is less common in veterinary patients experiencing CPA than human patients. Myocardial dysfunction that occurs secondary to CPA and CPR can manifest within hours of achieving ROSC, peaking at around 8 hours after ROSC and resolving within 72 hours.[2] To assess myocardial function, a point-of-care ultrasound should be routinely utilized to subjectively evaluate the quality of the heart's contractility through the PCA period. If poor contractility is identified or suspected, the administration of a positive inotrope is recommended, with dobutamine being the preferred positive inotrope.[17,18] Dobutamine is a β-adrenergic agonist that enhances cardiac contractility and is typically administered as a CRI, after appropriate dilution, at a dose of 1 to 20 µg/kg/min in dogs. Dobutamine should not be administered higher than 5 µg/kg/min in cats because of the risk of seizures. Continuous ECG monitoring is essential while the patient is receiving dobutamine, as arrhythmias are a common side effect. As with norepinephrine, dosing should start at the lower end and be titrated up or down based on the patient's BP and repeated point-of-care ultrasound.

Hypertension

While at times mild hypertension may be beneficial, hypertension with SAP > 200 mm Hg or MAP > 120 mm Hg may cause end-organ damage. Hypertension may occur due to pain, anxiety, increased ICP, excessive doses of vasopressors, or the patient's underlying disease. Analgesia should be prioritized in painful patients using a reversible opioid. Additionally, anxiety is commonly observed as patients regain consciousness. If the patient is restless or vocalizing, the administration of an anxiolytic is recommended. If an underlying cause of hypertension cannot be identified or treated, pharmacologics that cause vasodilation should be initiated to control BP. Considerations include the use of amlodipine, hydralazine, nicardipine, or nitroprusside.

Lactate and $ScvO_2$

Blood lactate refers to the amount of lactate present in the bloodstream, a byproduct of anaerobic metabolism that occurs when the body generates energy without adequate oxygen. It is naturally produced in small amounts and metabolized by the liver and kidneys. Elevated levels, known as hyperlactatemia, can occur during intense exercise, oxygen deprivation, and critical conditions, like sepsis, shock, and CPA. A blood lactate > 2.5 mmol/L may indicate poor oxygen delivery to the tissues, although there are causes of hyperlactatemia other than poor oxygen delivery. Blood lactate should ideally be sampled from an arterial or central/mixed venous sample. As previously discussed, sampling

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from an artery in the ROSC patient can be challenging, so venous lactate concentration is commonly assessed clinically. The 2024 RECOVER guidelines recommend that blood lactate levels should be monitored serially to assess lactate clearance as lactate levels should decrease as oxygen delivery improves. The veterinary team should work to decrease blood lactate levels as quickly as possible.[5]

Central venous oxygen saturation may also be utilized to assess oxygen delivery. Central venous oxygen saturation refers to the percentage of oxygen bound to hemoglobin in the blood within the central venous circulation. It reflects the balance between oxygen delivery and consumption in the body. Central venous oxygen saturation is influenced by the amount of oxygen carried on RBCs, cardiac output, and the amount of oxygen that is being extracted by tissues. Central venous oxygen saturation is measured from blood drawn from the cranial vena cava or the right atrium, often via a central catheter. An ideal $ScvO_2$ should be ≥ 70%. Lactate concentrations < 2.5 mmol/L and $ScvO_2$ values ≥ 70% suggest hemodynamic stabilization. If these targets are not met, it is essential to assess the patient's volume status, vasomotor tone, cardiac output, and arterial oxygen concentration. Since hemoglobin is carried by RBCs, anemia can result in reduced hemoglobin levels, contributing to hemodynamic instability.[5] According to the 2024 RECOVER guidelines, if the PCV is below 25% and the $ScvO_2$ or lactate levels remain unstable, a transfusion with 10 to 15 mL/kg of packed RBCs or 20 to 30 mL/kg of whole blood is recommended.

Neuroprotection

Cerebral edema and hypoxic-ischemic brain injury can develop during the PCA period due to neuronal excitotoxicity, cerebral acidosis, and reperfusion injury. A high ICP is a key indicator of cerebral edema, with normal ICP typically around 10 mm Hg. When ICP increases, the body initiates compensatory mechanisms to reduce pressure. However, once these compensatory mechanisms are exhausted, ICP will inevitably rise. With an increase in ICP comes a decrease in cerebral blood flow, ultimately leading to cerebral ischemia and worsening brain injuries. Neuroprotection can be provided by administering hyperosmotic solutions (such as mannitol or hypertonic saline), utilizing antiepileptics early on, allowing passive hypothermia, and monitoring blood glucose closely.

Hyperosmotic therapy

In human and veterinary patients, cerebral edema often occurs during the PCA period and is associated with a poor outcome.[2] Clinical signs of cerebral edema include mentation changes, such as coma, stupor, and severe depression; cranial nerve deficits; decerebrate posture; and persistent apnea or hyperventilation, requiring persistent IPPV. There are 2 main hyperosmotic solutions utilized during ROSC: mannitol and hypertonic saline. Both hypertonic saline and mannitol can equally and effectively reduce ICP. Some studies[19] of humans suggest that hypertonic saline may be more effective, has a more sustained effect on ICP, and effectively increases cerebral perfusion pressure.

Mannitol reduces ICP by drawing excess fluid from the cerebral interstitium. Additionally, it acts as a free radical scavenger, which is particularly beneficial during the PCA period because of the significant increase in free radical production.[20,21] Mannitol can be administered to treat acute cerebral edema at a dose of 0.5 to 2 g/kg, IV, over 15 to 20 minutes. All doses of mannitol should be followed by IV crystalloid fluids to prevent dehydration.

Hypertonic saline (3% to 7%) is used for its ability to reduce cerebral edema by generating an osmotic gradient due to its high sodium content. Hypertonic saline provides volume expansion and acts as a positive inotrope. However, volume overload could occur, and there is a risk of hypernatremia. Hypertonic saline is administered at 4 to 6 mL/kg in dogs and 2 to 4 mL/kg in cats, IV, over 15 to 20 minutes.

Seizure prophylaxis

Seizures occur in 5% to 15% of human patients in the PCA period, and the incidence increases drastically to 40% in patients who remain comatose.[2] Patients in the PCA period usually have significant neurologic injury secondary to the hypoxia that occurs with CPA. The development of seizures tends to potentiate those neurologic injuries. Seizures are commonly treated with benzodiazepines or levetiracetam. With the high incidence of seizures in the PCA period, it is reasonable to consider prophylactic seizure control.[22]

Therapeutic/passive hypothermia

During the PCA period, patients may develop hyperthermia due to the substantial release of inflammatory cytokines.[20] Hyperthermia in the PCA period is associated with a poor neurologic outcome and increased mortality.[20] Alternatively, hypothermia inhibits the inflammatory cascade and decreases the release of excitatory neurotransmitters. Therapeutic hypothermia has many benefits in human medicine and has been utilized for centuries. Benefits include mitochondrial protection, reduced cerebral metabolism, inhibition of calcium influx into cells, decreased neuronal excitotoxicity, lower production of reactive oxygen species, and regulation of seizure activity.[18,23]

The 2012 RECOVER guidelines recommend therapeutic hypothermia in patients that remain unconscious in the PCA period. The target temperature should be maintained between 32 and 34 °C, with hypothermia sustained for 24 to 48 hours.[4] Achieving and maintaining a mild therapeutic hypothermic state requires advanced critical care expertise and the capability to provide mechanical ventilation. If mechanical ventilation cannot be performed, then passive hypothermia should be utilized with a rewarming rate of 0.25 to 0.5 °C/h.[4] Many veterinary patients become hypothermic during CPR, which will assist in passive hypothermia

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during the PCA period. Avoid placing the patient on a cool metal examination table, as although it may promote passive hypothermia, it can cause significant discomfort. Instead, place the patient on a cool, padded surface covered with a thin, soft layer to ensure comfort while managing temperature. The patient's temperature should be monitored continuously to ensure a consistent temperature. Rectal thermometers are commonly used to monitor a patient's temperature, but for the most accurate measurement, a core body temperature using an esophageal thermometer is recommended if tolerated by the patient. If the patient's temperature fluctuates, warming agents or light towels/blankets can be applied or removed as necessary to help regulate their temperature. Risks of hypothermia include decreased cardiac output, decreased respiratory rate, impaired wound healing, and increased risk of infection as well as decreased metabolism, which could increase the risk of drug overdoses.[4,23,24]

Glucose management

In critically ill human, adult, and pediatric patients, as well as PCA patients, hyperglycemia or hypoglycemia is associated with poor outcomes.[25] The 2012 RECOVER guidelines suggest that normoglycemia aids in improved discharge rates.[2] While there is no definitive blood glucose value to aim for during the PCA period, blood glucose should be evaluated shortly after ROSC is achieved and monitored until discharge. The treatment of hypoglycemia should be quickly addressed with the administration of dextrose and then monitored closely so that hyperglycemia does not occur. Unfortunately, at this time there are no clear recommendations to treat hyperglycemia, but if treatments are pursued, iatrogenic hypoglycemia could occur.[5]

Other Nursing Care

Additional nursing care includes prioritizing nutrition, often via a nasogastric or nasoesophageal tube. Due to frequent fluid, medication, and laboratory monitoring needs, central venous catheter placement is recommended. Both procedures should be performed with caution as to not cause any additional increase in ICP. Post–cardiac arrest patients are often obtunded and immobile, requiring recumbency care to prevent pressure sores and muscle atrophy. Rotate patients every 2 to 4 hours, and consider passive range of motion if patients are recumbent for over 24 to 48 hours. Provide oral care every 4 to 6 hours in intubated patients to reduce the risk of aspiration pneumonia.[26] Apply eye lubricant every 2 to 4 hours in sedated or mentally altered patients while staining the eyes daily to check for corneal ulcers.

Potassium imbalances can either contribute to cardiac arrest or develop during the post-cardiac arrest (PCA) period as a result of hypoxia.[5] Hypoxia decreases ATP production, which disrupts the sodium-potassium pump and leads to potassium buildup.[27] Therefore, electrolytes should be closely monitored in the PCA period. The 2024 RECOVER guidelines also recommend measuring serum creatinine shortly after ROSC and at least every 24 hours.[5] In humans, 40% of patients develop acute kidney injury after CPA.[27]

Conclusions

Implementing the RECOVER PCA algorithm with a dedicated veterinary team can improve CPR outcomes. The goal is not just ROSC but survival to discharge. Cardiopulmonary resuscitation is physically demanding for both patients and staff, and the PCA period brings further challenges **(Table 2)**. Veterinary technicians should be prepared to address these challenges and continue providing exceptional care.

Table 2—Summary of considerations.

Summary of considerations in the post–cardiac arrest period in dogs and cats
Aim for normal carbon dioxide concentrations
Maintain normoxemia
Maintain normotension
Consider osmotic agents
Monitor glucose, lactate, and electrolytes
Treat seizures
Provide supportive care

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