1. Introduction
Cardiopulmonary resuscitation (CPR) is universally accepted in human medicine and has been applied to many veterinary species. In adult horses, the mass of the animal makes CPR insufficient for sustaining life. However, CPR is technically possible and has been successful in foals.1–3 The success of CPR in foals depends on early intervention and the cause of arrest. Anticipation of which foals may require resuscitation and adequate training and preparation of staff are crucial for maximizing chances of a successful outcome.

CPR is indicated in cases of cardiopulmonary arrest, which is defined as sudden cessation of spontaneous and effective respiration and heartbeat. Early identification of foals at risk for cardiopulmonary arrest may aid in adequate preparation. There are numerous maternal and foal factors that may make arrest more likely (Table 1). Newborn foals, particularly those from mares with high-risk pregnancies, are at risk of arrest during the immediate peri-partum period, because they may fail to make the physiological transition from fetal to neonatal life. Common clinical scenarios resulting in arrest include dystocia, premature delivery, premature placental separation, and delivery by caesarean section. In some cases, the foal may have already arrested before birth. Arrest in foals can also occur outside of the immediate perinatal period secondary to systemic disease that results in respiratory or cardiac failure and the subsequent development of hypoxic acidemia. Acidemia may then cause respiratory arrest, resulting in bradycardia and eventually, asystole. Perinatal hypoxia can damage the areas of the brain that control respiration, which causes hypoventilation. Septic shock can alter the oxygen content of the blood and cardiac output, resulting in cardiopulmonary arrest. Primary lung disease, airway obstruction, and failure of the respiratory muscles (diaphragm and intercostal) can result in progressive respiratory failure. In these foals, respi-
ratory arrest may be preceded by an increased respiratory rate and effort before fatigue of the muscles and respiratory acidosis caused by high carbon-dioxide levels in the blood. Cardiac arrest can also be caused by pulseless, non-perfusing arrhythmias, including ventricular fibrillation, ventricular tachycardia, asystole, or pulseless electrical activity (PEA). Primary cardiac failure is uncommon in neonatal foals, whereas secondary cardiac failure can be the result of congenital cardiac defects, hypoxic-ischemic damage, and severe systemic inflammatory disease. 1-3

Although CPR may successfully revive the patient, their ultimate survival depends on identifying and correcting the cause of arrest. Otherwise, they are likely to arrest again, and subsequent resuscitations are less likely to succeed. It is also important to keep in mind that if the underlying problem cannot be corrected, such as with foals having congenital defects, CPR is not indicated.

2. Materials and Methods

Preparation

There are few situations in which time is as important as during an arrest. Having a systematic plan of action and being prepared with well-organized supplies are crucial to a successful outcome. Because a veterinarian may not be present for all foalings, it is their responsibility to educate clients and prepare them for providing basic CPR. The needed supplies will partially depend on if you will be working in a farm or hospital situation, but the basics are the same. All supplies should be organized in a dedicated, easily accessible, and easily portable container or “crash cart” (Table 2; Fig. 1). It is often helpful to keep an easily readable, laminated copy of a CPR flow chart (Fig. 2) and emergency drug doses on the crash cart.

Performing CPR

A: Assessment/Airway

A rapid initial assessment of the foal should be made in the first seconds after birth. After a few initial gasps, a normal foal should be breathing regularly with a respiratory rate of 60–80 breaths per min (bpm) within 30 s. The heart rate should be regular at a rate of 60–70 bpm. Some normal foals have

Table 2. Resuscitation Kit Supplies

<table>
<thead>
<tr>
<th>Essential</th>
<th>Optional</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nasotracheal tubes (7–10 × 55 cm²)</td>
<td>Oxygen tank and flow meter</td>
</tr>
<tr>
<td>Air syringe</td>
<td>Heat lamp</td>
</tr>
<tr>
<td>Self-inflating resuscitation bagb</td>
<td>Other drugs (Table 3)</td>
</tr>
<tr>
<td>Bulb syringe</td>
<td>ECG monitor</td>
</tr>
<tr>
<td>Stethoscope</td>
<td>ETCO₂ monitor</td>
</tr>
<tr>
<td>Pen light</td>
<td>Electrical defibrillator</td>
</tr>
<tr>
<td>Epinephrine</td>
<td>Mask resuscitator</td>
</tr>
<tr>
<td>Syringes and needles</td>
<td></td>
</tr>
<tr>
<td>IV catheters (14–16 gauge)</td>
<td></td>
</tr>
<tr>
<td>Fluids</td>
<td>PE tubing or red rubber catheter</td>
</tr>
</tbody>
</table>
arrhythmias for up to 15 min after birth. These arrhythmias usually do not require treatment. Foals that continue gasping for >30 s or are not breathing, have a heart rate <50 bpm, or do not have a heartbeat require immediate resuscitation.

After a normal delivery, the foal can simply be observed for the first 20 s to ensure that it is breathing spontaneously. After a dystocia or caesarian section, the foal should be dried vigorously, and a clear airway should be established by clearing the fetal membranes and fluid from the nose and mouth. Drying the foal with the head lowered can aid in clearing fluid from the airways. However, aggressive postural damage, such as suspending the foal by the hindlimbs, is contraindicated. If meconium staining is evident, the airways should be suctioned with a bulb syringe or mechanical suction unit. If a mechanical unit is used, suctioning should not be performed for >5–10 s at a time, because there is a risk of stimulation of the vagus nerve, which can cause bradycardia.

Intubation is the best way to provide a patent airway. Nasotracheal intubation is preferable to orotracheal intubation, because there is less chance that the tube will be damaged as the foal wakes up. To intubate, the foal should be in sternal or lateral recumbency. Lateral recumbency may be preferable, because thoracic compressions can be started during intubation. With the neck extended to make a straight line from the nasopharynx into the trachea, the tube should be held with the curve downward and pushed with one hand ventromedially (“ventral and central”) into the ventral meatus while the other hand advances the tube. After the tip is in the nasopharynx, it can be rotated and advanced slowly through the arytenoids and into the trachea. If the tube is placed correctly, expired air should be felt at the proximal tube as the thoracic is compressed, and the first breath should expand the thoracic. The cranial ventral neck, just behind the throat latch, should be palpated, because the tube is easily palpable if it is in the esophagus. Applying gentle lateral pressure in this region while passing the tube can help prevent it from entering the esophagus. After the tube is in place, it should be advanced as far as possible to decrease dead-space ventilation. The cuff should then be inflated, and the tube should be secured in place behind the foal’s ears. If nasotracheal intubation is unsuccessful after the first few attempts, orotracheal intubation should be attempted.

Mask resuscitators are inferior to intubation, because there is a risk of aerophagia; additionally, it is more difficult to effectively ventilate the patient. However, they are a good alternative if trained personnel are not available to intubate the foal. Administration of doxapram to stimulate respiration increases oxygen consumption and requirement, but it should not be used as a substitute to ventilation during CPR.

B: Breathing
After the foal is intubated, a self-inflating resuscitation bag, such as a self-inflating resuscitation bag, should be attached to the nasotracheal tube. Oxygen should be used if it is available, but room air is sufficient; some studies in human neonates suggest an advantage to room air over 100% oxygen. Placing the foal on a firm, dry surface with the bag on the ground allows the person ventilating to use their upper body and the floor to their advantage to squeeze the bag, which delays fatigue.

The ideal rate of ventilation for foals is unknown, but rates of approximately 10 bpm are typically ef-
Hyperventilation should be avoided, because this has been associated with a decreased survival rate in people and animal models. Each breath should visibly expand the chest wall. It is possible to damage the lungs with too much volume or pressure of air; therefore, care should be taken not to be too aggressive. Some self-inflating resuscitation bag models provide measurement of airway pressure. A pressure of ≈20 cmH₂O is recommended.

C: Circulation
Thoracic compressions are not required in all cases of cardiopulmonary arrest. After 30 s of ventilation, the foal should be reassessed to determine if thoracic compressions are necessary. If there is no heartbeat, if the heartbeat is <40 bpm, if the heartbeat is <60 bpm and not increasing, or if a non-perfusing rhythm is present, then thoracic compressions are indicated. The ideal rate of compressions is not known, but rates of 80–120/min are typical, and a ratio of compression to relaxation is usually 1:1. All efforts should be made to avoid interruption to thoracic compression. The resuscitator will quickly become fatigued at this rate and should be relieved every 2–3 min. Ventilation should be continued throughout the compressions and do not need to be coordinated with compressions. If there is only one person attempting to both ventilate the foal and perform compressions, one breath can be given for every 10 thoracic compressions.

With the foal on a firm, dry surface, the resuscitator should be positioned at the foal’s spine, kneeling or standing. The hands should be placed on top of each other just behind the foal’s triceps, at the highest point of the thorax. With the shoulders directly over the foal and the elbows locked, the entire upper body can be used to compress the thorax (Fig. 3). Keep in mind that the goal is to compress the entire thorax, not just the heart. Rolled towels or sandbags should be placed under the foal to support the thorax and aide in compressions. If the foal has palpable broken ribs on one side, that side should be placed down. Although there is a risk of fracturing ribs during the procedure or lacerating the lungs with previously fractured ribs, this is not a reason to avoid performing thoracic compressions.

D: Drugs
Ventilation and thoracic compressions should always be the first steps during CPR. If there is no heartbeat after 1 min of thoracic compressions and ventilation, drug therapy can be considered. There have been no studies on CPR on foals, and all information regarding the use of drugs has been extrapolated from other species. In other species, drug treatment regimens for CPR are controversial, be-
cause there is little evidence of a clear survival benefit in randomized clinical trials.

Epinephrine is an endogenous catecholamine. The α2-adrenergic effects are believed to be most important during resuscitation, because they cause peripheral vasoconstriction and increase aortic tone during dias-
tole. The increased aortic pressure during diastole combined with thoracic compressions can promote cor-

donary perfusion, which is essential for return to a normal heart rhythm.

If vascular access is available, epinephrine should be given intravenously at a dose of 0.01–0.02 mg/kg. Epinephrine is supplied as 1 mg/ml; therefore, the dose for a typical 50-kg foal is 0.5–1.0 ml. Epineph-

rine has a short half-life; therefore, if there is no response after the first dose, additional doses can be given every 3 min. Although high-dose epineph-

rine regimens may be more likely to result in spontane-

taneous circulation in people, survival is less likely compared with low-dose epinephrine because of the toxic effects. Side effects of epinephrine include increased myocardial oxygen demand resulting in myocar-
dial necrosis and post-resuscitation tachyar-

rhhythmias and hypertension.

If IV access is not available, epinephrine can be given intratracheally. With the nasotracheal tube in place, the syringe can be attached to a long red rubber catheter or polyethylene (PE) tubing and placed down the tube. Higher doses (0.05–0.1 mg/kg of epinephrine) must be used for intratra-

cheal administration because of lower absorption using this route. Regardless of the route of admin-

istration, the drug must circulate to have an effect. Therefore, thoracic compressions should be contin-

ued to try to promote circulation.

Although epinephrine is typically considered the first line of treatment for cardiac arrest, randomized clinical studies showing a clear benefit over placebo are lacking. According to the American Heart Asso-

cation’s 2005 guidelines for cardiopulmonary cerebral resuscitation in humans,8 vasopressin can be used in place of or in conjunction with epinephrine for treatment of PEA, ventricular fibrillation, and ventricular tachycardia. Vasopressin (antidiuretic hormone) is an endogenous stress hormone that causes peripheral vasoconstriction. Vasopressin can be given at 0.8 IU/kg, and the dose can be repeated every 3–5 min or alternated with epinephrine every 3–5 min.9 Although vasopressin causes reflex bradycardia in healthy animals, which minimizes the pressor effect, the pressor effect is profound during arrest. Vasopressin is unlikely to cause arrhythmias or hypertension after resuscitation.

Many other drugs have been used during CPR on humans and veterinary species, and some may be indicated under specific circumstances (Table 3). Atropine sulfate, an anticholinergic parasympatholytic drug, is indicated for the treatment of vagally mediated bradycardia and asystole. In foals, bradycardia and asystole are most often caused by hypoxia rather than vagal mediation and therefore, should be treated with ventilation. Inappropriate use of atropine may be detrimental because of the increased oxygen demand by the heart in the face of hypoxia.3 Ami-
darone, a class III antiarrhythmic agent (potassium-channel blocker), is indicated for the treatment of ventricular tachycardia and ventricular fibrillation that is unresponsive to defibrillation and vasopressin.7 Lipid caine, a class 1b antiarrhythmic agent, stabilizes membranes by sodium-channel blockade and may be helpful in treatment of ventricular arrhythmias by decreasing automaticity.3 There has been no proven efficacy of lidocaine in cardiac arrest in humans. Current guidelines8 recommend lidocaine for ventricu-
lar fibrillation only if amiodarone is unavailable and electrical defibrillation is not planned because it may increase the defibrillation threshold. Calcium was originally advocated for use during CPR for its ability

**Table 3. Emergency Drug Dosages**

<table>
<thead>
<tr>
<th>Drug</th>
<th>Indications</th>
<th>Concentration</th>
<th>Dose</th>
<th>IV Dose for 50-kg Foal (ml)</th>
<th>Intratracheal Dose for 50-kg Foal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Epinephrine</td>
<td>Asystole, pulseless electrical activity, ventricular tachycardia, VF refractory to shock</td>
<td>1 mg/ml</td>
<td>0.02 mg/kg</td>
<td>1</td>
<td>2 ml in 10 ml of saline</td>
</tr>
<tr>
<td>Vasopressin</td>
<td>Asystole</td>
<td>20 IU/ml</td>
<td>0.8 IU/kg</td>
<td>2 (four vials)</td>
<td>3 ml in 10 ml of saline</td>
</tr>
<tr>
<td>Lidocaine</td>
<td>Ventricular tachycardia, VF refractory to shock</td>
<td>20 mg/ml</td>
<td>1 mg/kg</td>
<td>2.5</td>
<td>5 ml in 10 ml of saline</td>
</tr>
<tr>
<td>Amiodarone</td>
<td>Ventricular tachycardia, VF refractory to shock</td>
<td>50 mg/ml</td>
<td>5 mg/kg</td>
<td>5</td>
<td>NA</td>
</tr>
<tr>
<td>Atropine</td>
<td>Vagally mediated bradycardia (rare)</td>
<td>15 mg/ml</td>
<td>0.02 mg/kg</td>
<td>0.07</td>
<td>0.15 ml in 10 ml of saline</td>
</tr>
<tr>
<td>CaCl2</td>
<td>Severe hyperkalemia</td>
<td>100 mg/ml</td>
<td>20 mg/kg</td>
<td>10*</td>
<td>NA</td>
</tr>
<tr>
<td>MgSO4</td>
<td>Refractory ventricular arrhythmias</td>
<td>4.06 mEq/ml</td>
<td>0.2 mEq/kg</td>
<td>10*</td>
<td>NA</td>
</tr>
</tbody>
</table>

* Dilute in 100 ml of saline and give over 10 min.
NA, not applicable.
to increase cardiac contractility. However, a demonstrable benefit has not been shown, and the high calcium levels may speed cell death; therefore, it is only recommended for the treatment of ionized hypocalcemia and hyperkalemia. Magnesium sulfate is an essential cofactor for many enzymes involved in cellular metabolism, including the Na⁺,K⁺-ATPase. Hypomagnesemia can alter electrical activity and conductance through the heart. Administration of magnesium sulfate may be beneficial for refractory ventricular arrhythmias such as ventricular fibrillation and torsades de pointes (a life-threatening form of ventricular tachycardia).⁷,⁸

Foals delivered through caesarian section or controlled vaginal delivery under general anesthesia may require a specific reversal agent if an appropriate agent is available. IV fluid boluses are indicated in foals that are hypovolemic before resuscitation. However, newborn foals are rarely hypovolemic, unless hemorrhage has occurred, and aggressive administration of IV fluids to euvolemic foals may result in decreased coronary perfusion. Dextrose-containing fluids are indicated for hypoglycemcic foals, but they should not be administered as a bolus or used for fluid resuscitation during CPR. If ventricular fibrillation is confirmed by electrocardiogram (ECG), electrical defibrillation is indicated if the equipment and trained personnel are available.

3. Results and Discussion

Determining the Efficacy of CPR and Deciding When to Stop

Frequent reevaluation of the patient during CPR can help modify the techniques being used and determine when CPR can safely be stopped. Palpation of pulses can help monitor efficacy of thoracic compressions. However, the peripheral pulse is not an accurate indication of forward blood flow or coronary perfusion. The heartbeat, if present, can be used to decide when to stop thoracic compression. An ECG is useful during CPR to monitor the cardiac rhythm. However, the ECG is only a reflection of the electrical activity of the heart, which can be present without an effective contraction of the heart muscle (pulseless electrical activity). ECG is, therefore, not adequate for determining the efficacy of CPR.

Pupil size and response to light is a reflection of blood flow to the brain and is a rapid and easy way to assess effective blood flow. If the person performing thoracic compression has a pen light, they can periodically check the foal’s papillary light response. When blood flow is inadequate, the pupils are fixed and dilated. With adequate blood flow, the pupils become a more normal size and respond to light by constricting.

In a hospital setting, an ETCO₂ monitor (capnograph) can be a very useful tool during CPR. The expired tension of CO₂ is a reflection of the amount of CO₂ transported to the lungs by the blood and ventilated, and therefore, it reflects the adequacy of resuscitation. During resuscitation, ETCO₂ values <10 mm Hg reflect inadequate resuscitation. Progressively increasing ETCO₂ during CPR indicates adequate cardiac output and effective CPR.⁹,¹⁰,¹¹

If available, blood-gas analysis can aid in the assessment of the adequacy of ventilation and tissue perfusion. Central venous blood samples can be used to assess tissue oxygenation. The central venous oxygen saturation (S₅O₂, normal > 70%) and partial pressure of oxygen (P₅O₂; normal = ~45 mm Hg) are indicators of the oxygen extraction of tissues (i.e., how well the tissues have removed oxygen from the arterial blood). As oxygen delivery by the arterial blood decreases because of severe anemia and/or poor perfusion, these values decrease if the oxygen requirements of the tissues are not being met. The significance of S₅O₂ and P₅O₂ values in sick foals is unknown at this time. Arterial blood samples, obtained from the dorsal metatarsal or brachial artery, can be used to measure the tension of oxygen (PaO₂) and carbon dioxide (PaCO₂). The PaO₂ is a reflection of the oxygenating capability of the lungs, whereas the PaCO₂ is a measure of ventilatory status.

Sometimes it can be difficult to know how long to continue CPR and when to stop. If CPR has been continued for 15 min with no response, success is unlikely, and CPR should be stopped. If the foal is responding, CPR should be continued. After the foal has a regular heartbeat >60 bpm, thoracic compressions can be stopped. Ventilation can be stopped when the foal is breathing spontaneously with a regular rate of 16 bpm or more and has a regular heart rate >60 bpm. The foal may gasp for the first few breathes when initially disconnected from the bag but should then regain a regular rate and pattern.

Post-Resuscitation Care

After successful resuscitation, foals require intensive care and monitoring. A thorough physical examination should be performed and monitoring of body temperature, glucose levels, hydration status, and oxygenation of blood and tissues with appropriate treatment is warranted. If working in a farm setting, referral to a clinic is appropriate because of the delayed effects of asphyxia. If available, ECG monitoring, blood gas analysis, pulse oximetry, central venous pressure measurement, and echocardiography can aide in assessment of the foal’s cardiovascular status.

References and Footnotes


aMcCullogh Medical, Glenfield, Auckland, New Zealand 0600.
bAmbu SPUR adult disposable resuscitator with no mask, Ambu, Glen Burnie, MD 21060.
cSurgiVet Foal Nasal Endotracheal Tube, Smiths Medical PM, Waukesha, WI 53186.