Proceedings of the 16th Italian Association of Equine Veterinarians Congress

Carrara, Italy
January 29-31, 2010

Next SIVE Meeting:
Feb. 4-6, 2011 – Montesilvano, Pescara, Italy

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ABSTRACT

Neonatal foals deteriorate rapidly with disease and debilitation. Appropriate emergency intervention can make a huge difference to ultimate outcome, duration and costs of treatment. Cardiopulmonary resuscitation (CPR) is clinically worthwhile and technically possible in newborn foals. The priority is provision of an airway, and ventilation at a 10-20 breaths per minute. Fewer foals require thoracic compressions at 90-120 compressions per minute.

Foals may be hypovolaemic without showing many clinical signs. Foals that have not nursed for 4-6 hours are highly likely to be hypovolaemic. Hypovolaemia should be reversed with boluses of one litre (20 ml/kg) of lactated Ringer’s solution. Most obviously hypovolaemic foals require at least 2 boluses, and the maximum to be given acutely is 4 litres.

Foals may be hyperglycaemic, hypoglycaemic or normoglycaemic, and not all require glucose support. 50% glucose should be used to provide emergency glucose support in preference to 5% glucose.

Referral should be considered for any foal that is not up and nursing after treatment, is less than 2 weeks old and severely lame, or is colic and does not respond to an enema.

INTRODUCTION

Neonatal foals deteriorate rapidly with disease and debilitation. This rapid deterioration demands early identification and treatment of compromised foals. This lecture aims to outline emergency resuscitation in the foal, including cardiopulmonary cerebral resuscitation, rapid restoration of circulating volume with emergency fluid therapy, respiratory support with oxygen therapy and nutritional support with glucose supplementation.

CARDIOPULMONARY CEREBRAL RESUSCITATION (CPCR) OF THE FOAL

Newborn foals can arrest as a result of the birthing process, without any specific underlying pathophysiology. This makes them good candidates for resuscitation, in contrast to critically ill foals and horses that arrest as a result of a disease process.

RECOGNITION OF RESPIRATORY OR CARDIAC ARREST

Normal stage two labour should take less than 20 minutes. Regular breathing should start within 30 seconds of birth. The heart rate should be regular and around 70 bpm. Foals have pain and sensory awareness at birth and develop a righting reflex within 5 minutes. Respiratory, rather than cardiac, arrest is virtually always primary in the newborn foal. The arrest is usually a result of asphyxia, caused by premature placental separation, early severance or twisting of the umbilical cord, prolonged dystocia or airway obstruction by foetal membranes. Some foals will not start breathing spontaneously without any apparent birthing misadventure.

Foals requiring resuscitation are those that gasp for longer than 30 seconds or have obvi-
oss dyspnoea, foals with no respiratory movements or no heart beat, and those with a heart rate less than 40 bpm. Foals at risk of arresting should be identified prior to foaling, so that a veterinarian may be present. Risk factors include vaginal discharge during pregnancy, precocious udder development, placental thickening, illness of the dam during pregnancy, and delivery by C-section. The key to successful cardiopulmonary resuscitation (CPR) is a disciplined, ordered approach. The mantra “Airway, Breathing, Circulation” is especially important in resuscitation of the newborn foal, because respiratory arrest (rather than cardiac arrest) is almost always the primary problem.

THE FIRST 20 SECONDS

The first thing to do is to decide whether CPR is appropriate for this foal. Thereafter, this short time is dedicated to preparing the foal for CPR.

The foal should be placed in lateral recumbency on a hard flat surface. If any of the ribs are broken, the side with the broken ribs should be placed against the ground. If ribs are broken on both sides, the side with more of the cranial ribs (3, 4 and 5) broken should be placed on the ground. The head should be extended, so that the nose is in a straight line with the trachea. In the newborn foal, the nares and mouth should be cleared of membranes as a priority during this time. Vigorous towel drying should also be started, which acts as a strong stimulus to the foal to start breathing.

AIRWAY

If an endotracheal tube is available, it should be placed as soon as possible. As a rule, no more than 2 attempts should be made at nasotracheal intubation, before orotracheal intubation is attempted. These techniques are described above. The endotracheal tube should be attached to a self-inflating resuscitation bag (Ambubag®).

PUMP AND MASK

If an endotracheal tube is not available, or the expertise to use it is not available, the next most preferred option is a pump and mask. The mask should be fitted over the foal’s muzzle, to include the whole of both nostrils and form a reasonable seal. If possible, a second person should gently occlude the proximal oesophagus, to prevent air being forced into the stomach, which will hinder movement of the diaphragm. The oesophagus is best occluded just dorsal to the trachea (which can be felt as a tube with semi-rigid rings of cartilage), cranially and ventrally on the neck, just caudal to the larynx.

MOUTH TO NOSE RESUSCITATION

If neither an endotracheal tube nor a pump and mask are available, it is possible to perform mouth to nose resuscitation. One hand should be used to cup the chin and occlude the down nostril. The other hand should gently occlude the proximal oesophagus, as described above. The head should be dorsi-flexed as far as possible to straighten the airway, but the head should not be lifted. The resuscitator should watch to check that the thorax rises as they blow into the foal’s nostril.

BREATHING

The breathing rate should be 10-20 breaths per minute. This is the same, whichever method is used to provide air into the lungs. If possible, the resuscitation bag or pump should be connected to an oxygen supply. However, this is not essential, and CPR should not be delayed on interrupted to connect the oxygen.

CIRCULATION

If, after 30-60 seconds of supported ventilation, the foal has a heart rate less than 40, th
Thoracic compressions should be started. If the heart rate is between 40 and 60, supported ventilation should be continued for another 30-60 seconds, and the heart rate rechecked. If it has decreased or not changed during this period, thoracic compressions should be started. The foal should be on a hard surface in lateral recumbency for support of circulation. The best position for the resuscitator to adopt when performing thoracic compressions is as follows: Kneeling up, with the knees very close to the foal’s backbone at the level of the thorax. The resuscitator should place their hands at the highest part of the thorax, about one hand’s width behind the triceps mass. The hands are best placed flat on the thorax, with one hand on top of the other. The resuscitator should lean forwards, so that their shoulders are directly over their hands. They should then transfer their weight forward, to put firm even pressure on their hands, and aim to compress the chest. The hands should travel through approximately 3-5 cm as they compress the chest. The aim is for fast compressions, in the region of 90-120 compressions per minute. An effort should be made not to put any pressure on the thorax in the brief gap between compressions. Thoracic compressions are extremely tiring, and if more than one person is available, they should take turns at thoracic compressions (with minimum gap as they switch) every 2-3 minutes. If only one person is available, they should perform 15 thoracic compressions, followed by 2 breaths. If two people are performing the CPR, the 2 resuscitators should perform breathing and compressions independently, at the appropriate rates. In this case, thoracic compressions should not be interrupted for a breath.

**DRUGS**

Epinephrine (adrenaline) is the major drug for resuscitation of the foal. It should be given if the heart rate remains very low (<40 bpm) or absent after 2 minutes of full CPR (thoracic compressions and breathing). The dose is 0.01-0.02 mg/kg i.v. This is 0.5-1.0 ml per 50 kg bodyweight, when using standard 1 mg/ml (1:1000) epinephrine. This dose should be repeated every 3-5 minutes until a regular heart rate has returned, or it has been decided that CPR has been unsuccessful. If venous access is not possible, epinephrine can be injected into the trachea (below the cuff of the endotracheal tube, if present). The dose for intratracheal epinephrine is 0.1-0.2 mg/kg: 5-10 ml per 50 kg bodyweight. Intra-cardiac injection should be avoided.

Other drugs have a much more minor role in CPR of foals, and are rarely used in acute resuscitation. Fluid therapy (bolus of 10 ml/kg crystalloids) may be helpful in restoring the circulation. The following drugs are ineffective or dangerous in resuscitation of the newborn foal: Atropine, calcium and doxapram.

**MONITORING CPR**

The effectiveness of CPR can be monitored in several ways. A positive (normal) pupillary light response suggests that an adequate circulation is being maintained. A dilated, fixed pupil is a poor prognostic sign. The strength and consistency of a peripheral pulse can also be felt to monitor thoracic compressions, but this is difficult during resuscitation. If available, end tidal carbon dioxide tension represents the best way to monitor the effectiveness of cardiopulmonary resuscitation. Tensions greater than 15 mmHg indicate good perfusion and portend a good prognosis, whereas tensions persistently lower than 10 mmHg indicate ineffective CPR and a poor prognosis.

**WHEN TO STOP**

If started, thoracic compressions should be stopped when there is a regular heart beat of greater than 60 bpm. Ventilatory support should be stopped when there is regular, spontaneous breathing at a rate greater than 15 breaths/min. It is important not to stop ventilatory support too early, and intermittent support may be required before it can be completely withdrawn. Whereas a spontaneous heartbeat should start instantly when thoracic compressions are...
stopped, there may be a lag between ventilatory support stopping and spontaneous breathing, because blood carbon dioxide tensions have been reduced. This lag period should not be greater than 30 seconds, unless ventilatory support has been above the recommended rate of 10-20 breaths per minute. If there is no return of spontaneous circulation after 15 minutes of full CPR, the outlook is virtually hopeless.

**EMERGENCY FLUID RESUSCITATION**

Prompt, adequate fluid therapy is one of the easiest and most effective ways to maximize a foal’s chance of survival. However, determining which foals require emergency fluids can be difficult, as many of the clinical signs of hypovolaemia that are familiar in the mature horse are inconsistently present in the foal. The clinical signs of hypovolaemia in the mature horse are: tachycardia, weak pulses, poor filling of the jugular vein, tachypnoea and cold extremities. When any of these clinical signs occur in the foal, hypovolaemia should be suspected.

**FLUID THERAPY FOR HYPOVOLEMA**

Balanced electrolyte formulas, designed for resuscitation, such as Hartmann’s solution, Lactated Ringer’s solution or Normosol-R, are the best fluids to use for reversing hypovolaemia. These fluids contain approximately the same concentration of electrolytes as plasma. They are therefore the safest fluids to use if electrolytes cannot be measured. If blood electrolyte concentrations are available prior to beginning fluid therapy, the most common choice of fluid should still be balanced electrolyte solutions. It is inadvisable to attempt major electrolyte replacement prior to restoring a circulating volume. One exception to this rule is hyperkalaemia, hyponatraemia and hypochloraemia, which are seen in a percentage of cases of ruptured bladder. For these foals, 0.9% or 1.8% sodium chloride solution is often the best resuscitation fluid. Sodium chloride has traditionally been used as a resuscitation fluid in human medicine. Sodium chloride is an acidifying fluid and therefore may not be the best choice for acute resuscitation in foals, as most of these foals are acidic, due to lactic acidosis. Hypertonic saline (7 to 7.5% sodium chloride) has no role in resuscitation of neonates. Hypertonic saline may cause a rapid change in plasma osmolarity, resulting in brain shrinkage and subsequent vascular rupture with cerebral bleeding, subarachnoid hemorrhage and permanent neurological damage or death, of which neonates are particularly susceptible. The change in plasma osmolarity is more severe in animals with renal insufficiency, a common finding in critically ill foals. Colloids are solutions that contain large protein or starch molecules, as opposed to crystalloids, which contain only electrolytes and water or glucose and water. The role of colloid solutions such as modified gelatins (Haemacel and Gelofusine) and hydroxyethyl starches (tetraastarch, pentastarch and hetastarch) for acute resuscitation of the foal is unclear. The theoretical advantage is that they expand the plasma volume by a greater amount than the balanced electrolyte formulas, and persist in the circulation longer, prolonging their positive effect. They also increase the plasma oncotic pressure, in contrast to crystalloids, which decrease it. However, there are no clearcut benefits of colloids for neonatal foals in clinical practice. The total daily dose of pentastarch should not exceed 15 ml/kg. At higher doses, pentastarch may interfere with coagulation and may cause clinical bleeding. Pentastarch is probably indicated for resuscitation in foals with a plasma total solids concentration of less than 35 g/L. The initial plasma expansion is greater with lower molecular weight colloids and higher molecular weight colloids persist longer in the circulation. The average molecular weight of modified gelatins is small (30-35kD) when compared to albumin (69kD) and pentastarch (200kD). Modified gelatins solutions are therefore preferred for initial resuscitation of markedly hypovolaemic foals, and hydroxyethyl starches.
may be better for long-term maintenance of plasma colloidal oncotic pressure. Plasma is a colloid solution often used for supplementing passive immunity in foals. Plasma needs to be defrosted (if stored) or collected from a donor, and is therefore rarely available for acute fluid resuscitation. Furthermore, because some foals may have anaphylactoid reactions to plasma transfusions, it is good practice to initially infuse plasma slowly, and check for a reaction. Again, this detracts from the use of plasma for fluid resuscitation.

**FLUID RATE**

There are two ways to think about the treatment of hypovolaemia, which both result in similar treatment patterns. Hypovolaemic foals typically require 20-80 ml/kg of crystalloid fluids acutely.

**Shock Dose**

The ‘Shock Dose’ concept is borrowed from small animal medicine, and so is familiar to many. The shock dose for a neonatal foal is 50-80 ml/kg of crystalloid fluids. Depending on the perceived degree of hypovolaemia, a quarter to one half of the shock dose is given as rapidly as possible (over less than 20 minutes) and the foal is reassessed. If the foal requires further fluid, another quarter of the shock dose is given and again the foal is reassessed. The final quarter of the shock dose is only given to severely hypovolaemic foals.

**Fluid boluses**

The incremental ‘fluid bolus’ concept is borrowed from human medicine. It is actually a much more practical method, except when an electronic infusion pump is available. The caveat is that it assumes a similar bodyweight between all patients, and for this reason it has not been adopted in small animal medicine. The bolus method is simply to give a bolus of 1 litre of crystalloids (i.e. approximately 20 ml/kg bwt for a 50 kg foal), and reassess. Up to three further boluses may be given, reassessing the foal after each. Most obviously hypovolaemic foals require at least two boluses.

In foals where their bodyweight is obviously different from 50 kg, the method needs to be adjusted so that the bolus is approximately 20 ml/kg. In pony foals and very premature thoroughbred foals, boluses of 500 ml are usually appropriate. In large draft foals, the first bolus should be 2 litres.

**How much to give**

Whether using the ‘shock dose’ method, or the fluid bolus method, the animal is reassessed during acute fluid therapy to judge if further fluids are required. Foals with a strong pulse, improved mentation and that are urinating probably do not require any further resuscitation fluids.

These foals are likely to still require fluids to correct dehydration and electrolyte imbalances, and to provide for maintenance and ongoing losses (see below). Foals with continued weak pulses (or low blood pressure), depressed mentation and that have not urinated may require further acute fluids, up to the maximum of 80 ml/kg or 4 litres.

It is advisable to auscultate the lungs and trachea before and during aggressive fluid therapy, because pulmonary oedema is an important theoretical complication. Fortunately, pulmonary oedema appears to be extremely rare in critically ill foals aggressively resuscitated with crystalloids. Crackles, classically associated with pulmonary oedema, are more likely to represent opening and closing of collapsed alveoli, rather than oedema in foals. Severe pulmonary oedema will result in wet sounds in the trachea, and a frothy pink fluid from the nares or mouth. If oedema does occur, furosemide (0.25 to 1 mg/kg i.v.) should be administered and further fluid therapy carefully titrated, preferably by means of central venous pressure or pulmonary pressures.

Treatment of hypovolaemia takes precedence over any concerns about possibly causing cerebral oedema and thus worsening Perinatal Asphyxia Syndrome. Inadequate cerebral perfusion, due to hypovolaemia, prolongs the ischemic event and is thus extremely detrimental to these foals. This is far more important than theoretical concerns over cerebral oedema.
IMPORTANT EXCEPTION TO AGGRESSIVE FLUID THERAPY

Aggressive fluid therapy should be avoided in uncontrolled hemorrhage, because it may increase bleeding. This is uncommon in neonatal foals, but may occur with trauma resulting in internal abdominal bleeds or rupture of an inaccessible artery. In humans and experimental animals, aggressive fluid therapy in uncontrolled haemorrhage has been demonstrated to increase mortality. If blood pressure can be measured, fluid therapy should be titrated to maintain the mean arterial pressure as close to 60 mmHg as possible, without increasing the systolic pressure over 90 mmHg. If blood pressure cannot be measured, then a fluid rate of 2-3 ml/kg/hr should be used, until haemorrhage can be stopped. Foals with neonatal isoerythrolysis are not commonly hypovolaemic, unless they have become so debilitated that they have stopped nursing for 4 hours or more. In foals with isoerythrolysis and hypovolaemia, aggressive fluid therapy is not counter-indicated. Although fluid therapy will decrease the hematocrit, it will not decrease the number of circulating erythrocytes and may improve their distribution to the tissues. However, in foals with isoerythrolysis and hypovolaemia, aggressive fluid therapy is not counter-indicated. Although fluid therapy will decrease the hematocrit, it will not decrease the number of circulating erythrocytes and may improve their distribution to the tissues. However, in foals with low packed cell volumes, restoring blood oxygen carrying capacity is a priority, and donor blood, washed mare’s blood or haemoglobin substitutes (Oxyglobin) should be given as soon as possible.

EMERGENCY GLUCOSE SUPPORT

Intravenous glucose therapy is often part of emergency treatment in foals. This is because the glycogen stores at birth are only sufficient for approximately two hours energy requirements in the unfed foal, and that fat stores are also very low at birth. Therefore, foals that are not nursing are very prone to hypoglycaemia. Septicaemia may also result in hypoglycaemia, possibly as a result of lack of glyco- gen reserves and poor nursing in septic foals. However, foals may also be hyperglycaemic at hospital admission, presumably as part of the physiological response to cortisol release or associated with unregulated glucose metabolism with disease processes. In a series of 515 referred foals at hospital admission, 17% were hypoglycaemic (blood glucose <4.2 mmol/L) with 15% severely hypoglycaemic (<2.8 mmol/L). 47% were hyperglycaemic (>7.3 mmol/L), but only 12% were severely hyperglycaemic (>10 mmol/L).

Both hypoglycaemia and hyperglycaemia may be harmful. Following cerebral hypoperfusion (a feature both of hypovolaemia, and of perinatal asphyxia syndrome), hyperglycaemia may be more detrimental than hypoglycaemia. For this reason, it is advisable to monitor the blood glucose frequently in foals. Hyperglycaemia is treated with infusions of normal insulin (0.05-1.0 u/kg/hr). Hypoglycaemia is treated with glucose containing fluids, or parenteral nutrition.

FLUIDS FOR SUPPORTING GLUCOSE CONCENTRATION

5% glucose solutions
One litre of 5% glucose provides approximately 190 kcal (796 kJ). They are not a great source of energy for the foal. To meet resting energy requirement (44 kcal/kg/day (184 kJ/kg/day) of a 50 kg foal, 11.5 litres per day would need to given. This is over double the foal’s maintenance fluid requirements and would cause considerable electrolyte disturbances. 5% glucose has been suggested as a resuscitation fluid for foals, because it provides both volume and energy. However, it is not a good fluid for treating hypovolaemia. After 30 minutes, only 10% of volume given is left in the circulation, and each litre of 5% glucose will drop the plasma sodium concentration by 4-5 mmol/L in a 50 kg foal.

50% glucose solutions
These fluids may be preferable to the 5% solution. Each millilitre of 50% glucose is equivalent to 1.9 kcal (8 KJ). It may be used in two ways. In the hospital setting, the solution should be administered via an electronic pump, separately from the resuscitation fluids. The starting rate will depend on the degree of
hypoglycaemia. As a rule of thumb, a starting rate of 20 ml/hr is appropriate for mild hypoglycaemia (2.8-4 mmol/L) and 50ml/hr for severe hypoglycaemia (<2.8 mmol/L). In the field, it is probably best to add the 50% glucose solution to the resuscitation fluids. In this situation, 10 to 20 ml of the 50% solution should be added per litre of resuscitation fluid. If blood glucose can be measured, the amount of 50% solution added to the resuscitation fluids should be varied based on the measured blood glucose, to deliver approximately 20 ml/hr for mild hypoglycaemia and 50 ml/hr for severe hypoglycaemia. Glucose is not suitable as long-term nutritional support for foals, and if enteral feeding is not possible or desirable after the first 12 hours of therapy, parenteral nutrition with solutions containing dextrose or glucose, amino acids, vitamins and trace minerals and, in many foals, lipids, should be instituted.

**EMERGENCY OXYGEN THERAPY**

Oxygen therapy is extremely useful for support of foals. It should be considered in all foals following resuscitation and dystocia. Other foals that are likely to benefit from oxygen are those that are dyspneic, cyanotic, meconium stained after birth or recumbent. In the hospital setting, oxygen therapy should be based on the oxygen tension (PaO₂) in an arterial blood sample. Oxygen should be supplemented if the arterial tension is less than 65-70 mmHg (8.7-9.3 kPa). The most convenient places for sampling arterial blood in the foal are the dorsal metatarsal artery and the median artery. Small-bore flexible rubber feeding tubes are useful as oxygen cannulas for intra-nasal oxygen therapy in the foal. The length to be inserted into the nares should be measured as the distance from the nares to the medial canthus of the eye. The tube is then inserted into the ventral meatus of the nose. There are a variety of ways of fixing the tube in place. One method is to attach the tube to a tongue depressor, which has been previously wrapped in tape. The oxygen tube is taped along one edge of the tongue depressor, and then curled around one end, so that it heads back in the direction it is coming from. It is not taped to the bottom edge of the depressor. The tube and depressor are then attached to the foals muzzle using tape or elastikon (elastoplast), taking care not to prevent the foal from opening its mouth. An alternative method is to stitch the cannula to the foal’s skin, at the point it enters the nares. Oxygen may also be delivered in the short-term by facemask. If given for extended periods (greater than one hour), oxygen should be humidified prior to delivery to the foal. The simplest way of achieving this is to bubble it through sterile water. Easily sterilized bottles, designed for humidification, are available commercially. Oxygen therapy should be started at 9 to 10 L/min and titrated according to the response of the patient and, if available, arterial oxygen tensions. If measuring arterial oxygen tension, the oxygen flow rate should be decreased if the tension is greater than 120 mmHg (16 kPa).

Oxygen is not a completely benign therapy. Inspired oxygen fractions of greater than 60%, for more than 48 hours result in pulmonary pathology. This results in tracheobronchitis, leading to ARDS and subsequently to pulmonary interstitial fibrosis. This is probably mediated through oxygen free-radical formation (increased free-radical formation with increased inspired oxygen overwhelms scavenging). There may also be non free-radical mediated injury through cellular metabolic alteration or by enzyme inhibition. Fortunately, it is almost impossible to generate inspired oxygen fractions of greater than 60% with intranasal oxygen therapy.

**CONCLUSION**

Early recognition of foals requiring emergency support is the key to success. This is achieved through assessment of the history to anticipate which foals are likely to require intervention, rapid clinical assessment of foals, and a high index of suspicion. Cardiopulmonary cerebral resuscitation, fluid resuscitation and glucose and oxygen supplementation, applied judiciously, can both reduce mortality and morbidity.