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Birth Resuscitation

The transition that occurs at birth is often thought of in terms of the shift from placental to pulmonary gas exchange and the circulatory changes that accompany this remarkable transformation. The full transformation from fetal life is much more complicated and wide ranging. It begins weeks before birth and continues weeks after birth. There are endocrine changes with a slow rise in cortisol levels accompanied by a simultaneous increase in numbers and sensitivity of cortisol receptors. There are fluid shifts associated with progressive increases in blood pressure and redistribution of perfusion at the organ level such as the transition of renal perfusion from the juxtamedullary area to the subcapsular nephrogenic zone and the redistribution of intestinal perfusion to the villus capillary beds. There are metabolic changes such as the appearance of glucogenic enzymes and insulin responsiveness. Gastrointestinal motility and function dramatically change. These are just a few of the many transitions which need to occur for successful birth.

An essential and surprisingly complex step is the initiation of consistent rhythmic breathing. Episodic breathing occurs throughout late gestation, stimulated during active sleep or by changing CO_2 levels. It is inhibited at the brain stem by endogenous opioids, PG and adenosine. Hypoxemia completely inhibits breathing whereas severe asphyxia can induce gasping movements (associated with aspiration of meconium late term or at birth). During parturition breathing is completely inhibit, but for a successful birth transition, breathing must begin within seconds after birth. **First breaths** are normally triggered by the combination of removal of placental humoral inhibitory factors, cooling of the neonate, tactile stimulation, the catecholamine surge's induction of substances important for breathing (e.g. substance P) and rising CO_2. The reasons for **apnea at birth** are many and include birth asphyxia, CNS depression from maternal drugs, CNS injury, septicemia, anemia, primary muscular or neurological disease, obstructing congenital malformations or other mechanical obstruction of the airway. High oxygen levels used during resuscitation may actually delay the time to first spontaneous breaths because the set point of the peripheral chemoreceptors for Pao_2 is low after birth, and takes 2 to 3 days before it increases to adult levels.

Although the delay or failure of any of the transitions may adversely affect outcome, there are a few steps which if unsuccessful, such as the initiation of breathing, will result in a rapidly fatal outcome. Birth resuscitation is focused on monitoring the progress of these critical steps with a well thought out plan for rapid intervention to prevent a negative outcome.

Preparation for Resuscitation

The key to successful birth resuscitation is anticipation. High risk situations may be obvious from the pregnancy history (complications, drug therapy, nutritional state) or intrapartum course (prolonged stage I or II, unusual circumstances) but not all neonates requiring birth resuscitation have a suspicious history. At least half of the neonates requiring birth resuscitation have no indication that they are in a high risk situation. Since the need for birth resuscitation can't always be anticipated those attending must always be prepared. An important part of preparation is a well thought out plan and readily available adequate equipment. It is most important to have equipment for securing an airway and assisting ventilation. In foals, where nasotracheal intubation is preferred, this consists of proper size endotracheal tubes (55 cm long, 9 mm internal diameter; 8 mm, 7 mm, 10 mm tubes may also be useful depending on breed and maturity of the foal) and a self-inflating bag. For other species, it may also include a laryngeal scope and proper size blades as well as stylets. Also, a grip with resuscitation drugs, catheters, intraosseous needles and other supplies can be very useful.

EXIT Procedures

The explosive nature of parturition in the mare and the rapid release of the fetal membranes, places the foal at greater risk of not surviving even a short dystocia than other species. The **use of EXIT procedures** (EXutero Intranatal Treatment procedures) can increase the likelihood of survival. The most important EXIT procedure is intubation and ventilation of the foal before delivery. If there is a head and neck extended anterior presentation and the nose is within reach, the foal can be intubated and ventilated while the dystocia is corrected. Monitoring exhaled CO_2 is a simple, noninvasive procedure which can be very useful during any resuscitation attempt. This EXIT procedure will prove whether or not the foal is alive and with that knowledge and with successful ventilation the urgency to correct the dystocia will be gone, allowing for a reassessment of the situation and more time for manipulations. Ventilation of the foal will not only insure oxygenation, it will also decrease placental perfusion, preventing further transfer of drugs or other deleterious substances from the dam. Also ventilation will help clear volatile anesthetics. Once a foal is delivered after successful EXIT procedures, they are often active and very responsive unless they have suffered server asphyxia before ventilation was begun.

Elements of Resuscitation

The series of elements that makes up birth resuscitation are presented as steps but should not be thought of as a linear flow. The progression varies with the circumstances of the case and ongoing evaluation. The initial step may be taken in all cases, but the final steps in very few cases.

Initial Quick Overview – The initial assessment during a dystocia should begin in the birth canal (see EXIT procedures above). With a spontaneous delivery, if the neonate is breathing, getting sternal, looking around, has good body tone, has a normal respiratory pattern, is not meconium stained, mucous membranes are not cyanotic or pale, then no special intervention is needed.

****Apgar score**** – If the initial assessment suggests the neonate may not be normal, the heart rate should be immediately assessed. If a nonperfusing rhythm is present, full CPR should be initiated. If the heart is effectively producing perfusing blood flow, the Apgar score can be very useful in assessing the need for invasive resuscitation. Scores are usually recorded at 1 minute, 5 minutes and 10 minutes. Scores of 4 or less suggest the need for immediate intervention. Scores of 7 or 8 suggest no intervention is needed. Scores of 5 or 6 suggest close observation, stimulation and preparation for intervention.

Apgar score modified for the foal.

Score	0	1	2
Heart Rate	Absent	< 60 irregular	> 60 regular
Respiratory Rate	Absent	Irregular	regular
Muscle Tone	Limp Lateral	Some Flexion	Active Sternal
Reflex Nasal Stimulation Ear Tickle	No Response	Grimace Weak Ear Flick	Sneeze/Cough Ear Flick/Head Shake

Score = 7-8 Normal
Score = 4-6 Mild to moderate asphyxia - stimulate, intranasal O_2
Score = 0-4 Severe asphyxia - begin CPR

Clearing the Airway – Make sure the fetal membranes are not occluding the airway. Suctioning is rarely indicated even in the presence of meconium staining. If the neonate is very depressed and "chunky" meconium is present, suctioning of the airway using an endotracheal tube as a suction tip may be helpful. However, especially in calves and crias, but generally in all neonates, vigorous suctioning and stimulation posterior pharynx may induce bradycardia resulting in a nonperfusing rhythm. Also suctioning will cause collapse of the lungs and acute hypoxemia. Suctioning should be used sparingly and not for longer than 5 second intervals.

****Tactile Stimulation**** – Rubbing the chest and head should stimulate regular respiration and increased heart rate. If initial attempts fail to reverse apnea it is unlikely that prolonged stimulation or more vigorous attempts will be successful. Rather than continuing tactile stimulation with the danger of prolonging anoxia, other more

aggressive attempts to establish respiration and a resolution of bradycardia, such as intubation and ventilation, should be pursued. However, continued tactile stimulation can be useful in cases of irregular respiration with short respiratory pauses.

Thermal management – Hypoxic neonates are susceptible to cold stress (especially small neonates with high surface area to volume ratios). The neonates should be dried and wet bedding removed to avoid evaporative heat loss. Prematurity, asphyxia, hypoxia blunts the normal response to cold. Hypoxic neonates suffer greater than normal drop in core temperature. Recovery from acidosis is delayed by hypothermia.

****Free Flow Oxygen**** – If the neonate has spontaneous respiration but has bradycardia, irregular respiration or is cyanotic, free flow oxygen (intranasal or flow-by) can be useful. There is some debate about whether room air is more appropriate than 100% O_2 in resuscitation, but free flow oxygen usually supplies considerably less than 100% O_2. Placement of an intranasal line is strong tactile stimulus and sometimes results in regular respiration or increase in heart rate before oxygen flow is begun. Flow-by oxygen therapy is useful in small neonates where introduction of a nasal line may cause significant nasal obstruction or any neonate where the stimulus of an intranasal line is counterproductive. All free flow oxygen should be humidified to avoid unnecessary nasotracheal trauma.

Positive Pressure Ventilation – Neonates who don't initiate spontaneous ventilation or who develop a nonperfusing bradycardia should be ventilated. Most neonates are dedicated but not obligate nose breathers. This makes mouth-to-nose ventilation possible if no equipment to secure an airway is readily available. If possible, the airway should be secured with an endotracheal tube and the neonate ventilated with a self-inflating bag equipped with a pressure relief (pop-off) valve which can be temporarily occluded when needed. When ventilating a neonate who has never established spontaneous ventilation, not only is it necessary to deliver an appropriate tidal volume, but also a normal FRC should be established. With spontaneous ventilation in a neonate with normal physiology, a normal FRC is usually established during the first breath. An opening pressure greater than 10 cm water is rarely needed for the first breath because air enters the lung as soon as the intrathoracic pressure begins to fall. Also, slight glottis resistance during expiration helps force more small airways open resulting in more even distribution of ventilation. With positive pressure ventilation, the FRC established may be much smaller. This has lead to the suggestion that the inspiratory phase of the first assisted breath be prolonged 5 seconds to help establish the FRC. But it may be more effectively establish with higher pressures during the first 4 breaths. Excessive pressure resulting in large tidal volumes, especially when repeated can quickly cause lung damage through volutrauma. The best ****guide to adequate but not excessive pressure/tidal volume**** is the gentile rise and fall of the neonate's chest. As the FRC is established and surfactant released, compliance will rise so the pressure required to deliver the appropriate tidal volume decreases. By adjusting the pressure used to deliver the breath as gauged by the rise of the chest, excessive volume (and resulting volutrauma) can be avoided but an adequate tidal volume delivered. An awareness of the rapid rise in compliance resulting

in a drop in pressure needed to deliver the appropriate tidal volume and willingness to act on these changes is needed to avoid lung injury.

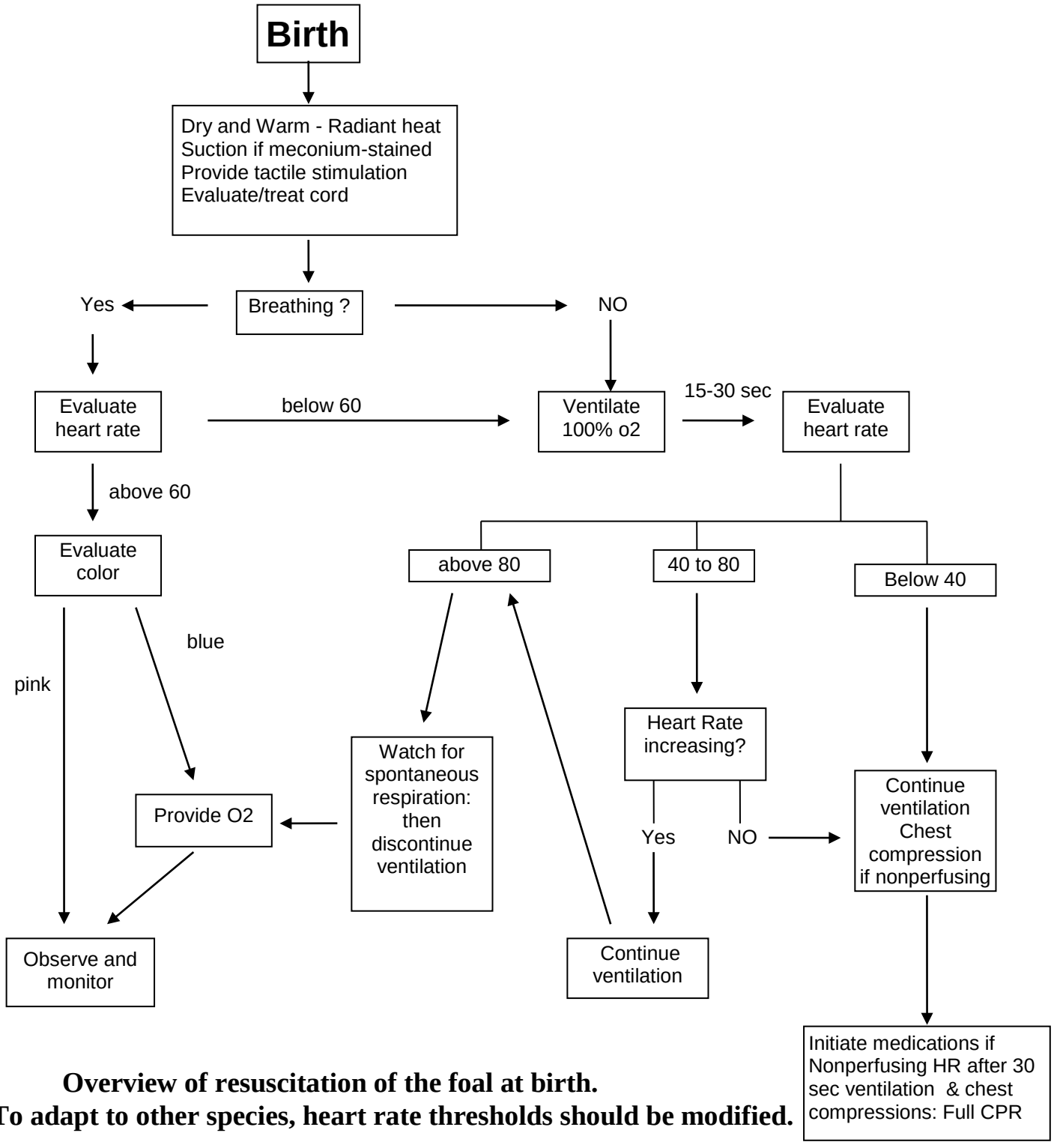
Chest Compressions – If the neonate has an extreme bradycardia or other nonperfusing cardiac rhythm, cardiac compressions should be initiated. The foal should be placed on a firm surface with its withers against a wall so that it does not move during forceful compressions. Place the palm of the hand with the fist closed over the heart. Place the other hand to reinforce the compressing hand. Compressions should originate from motion of the waste of the attendant not the elbows (the upper body weight powering the compression resulting in increased endurance). To maximize cardiac output half of the duty cycle should be compression and half relaxation. This is easiest to achieve with a rapid compression rate of 100-120 per minute. The resuscitator should not be overly ambitious in setting a rate. Too rapid a rate will result in early operator fatigue. If an airway is secured, coordination between ventilation and chest compression is not essential. Cardiac output is enhanced by ventilation superimposed on chest compression but there is evidence that cardiac perfusion may decrease during the simultaneous ventilation and compression. Cardiac output is heavily dependent on cardiac filling between compressions which is impeded by positive thoracic pressure such as occurs during positive pressure ventilation. Pausing active chest compressions for more than 3 seconds as would occur with interposed ventilation with compressions significantly increases the likelihood of a negative outcome. Routine coordination of chest compression and ventilation can result in increased cerebral pressure which is clearly contraindicated in cases with neonatal encephalopathy and should be avoided in neonates with possible perinatal hypoxia. There is recent evidence that rapid respiratory rates during CPR are detrimental and more effective tissue oxygen delivery will occur at a rate of 1 ventilation for every 30 to 60 compressions. Although many of these issues have not been adequately explored in neonates, based on current evidence I recommend delivering 100 chest compressions per minute with only 6 to 8 breaths per minute without stopping the compressions to deliver the positive pressure ventilation. If chest compressions do not increase heart rate within 30 sec resulting in a perfusing spontaneous rhythm, medication is indicated.

Medication – Despite some down sides, the most valuable pharmacologic aid in resuscitation continues to be **epinephrine**. During chest compression coronary blood flow is restricted to the diastolic period. Diastolic aortic pressure determines coronary perfusion, because during cardiac arrest there is no coronary capillary resistance and central venous pressure is low due to minimal venous return. Epinephrine increases diastolic aortic pressure by simultaneously preventing run off into peripheral tissues (by peripheral arterial constriction) and by increasing aortic tone. The combination of effective chest compression and the action of epinephrine results in a return of coronary perfusion, which is the most important step in resolving cardiac arrest no matter what the cause. Without coronary perfusion there is no hope of return to a normal cardiac rhythm. **Fluid volume loading** is not indicated in neonatal resuscitation unless there is an obvious fluid loss as could occur with significant hemorrhage (umbilical bleeding or bleeding secondary to a fracture).

A neonate born to a mare who has received drugs may be drug-depressed and benefit from reversal agents such as naloxone, flumazenil, atipamezule or yohimbine. I prefer to avoid tolazoline because of adverse reactions which occasionally can be fatal in neonates. If the foal is depressed secondary to inhalation anesthetics, ventilation is the best method to enhance clearance.

Post resuscitation care

Once spontaneous ventilation and a perfusion cardiac rhythm is established, intensive supportive care may be indicated. Assisted ventilation may be needed especially if the transition from fetal circulation is incomplete. Glucose therapy is helpful in replenishing glycogen stores, speed resolution of acidosis and as general metabolic support. Fluid therapy is indicated to support cardiac output. Use of inotropes such as dopamine or dobutamine can also be helpful in supporting cardiac output.



Overview of resuscitation of the foal at birth.
To adapt to other species, heart rate thresholds should be modified.