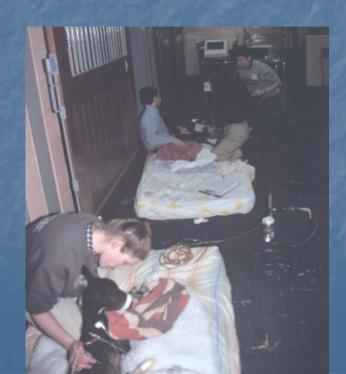
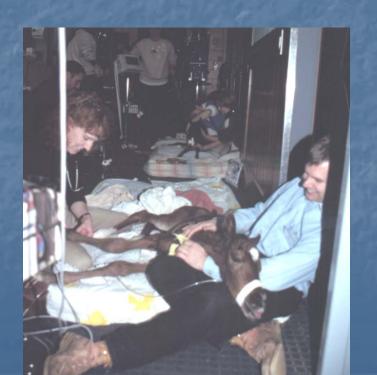


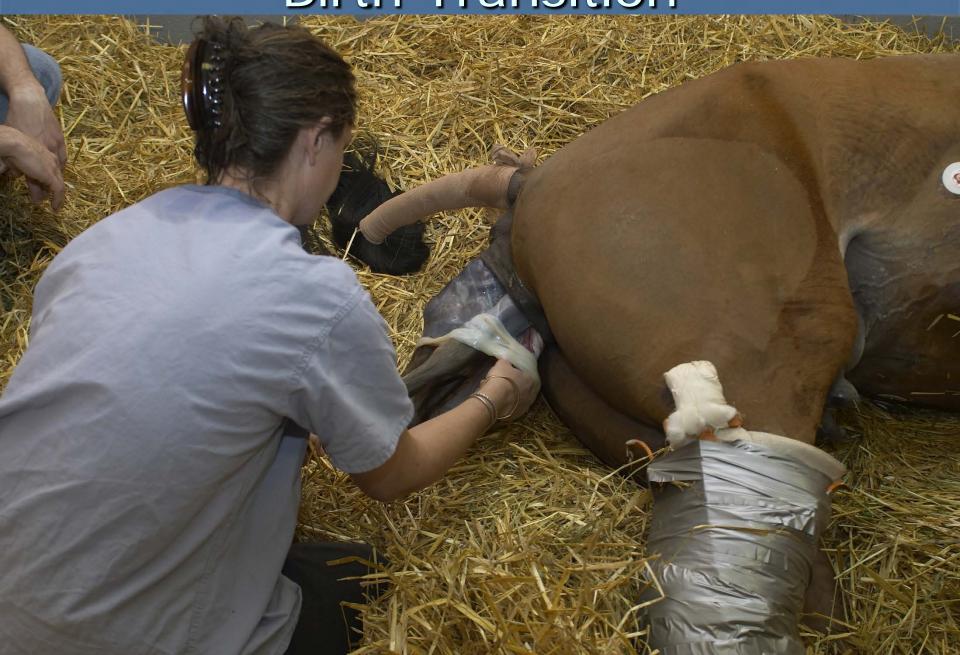
Resuscitation

- Fetal Resuscitation
- Intranatal Resuscitation
 - **EXIT** Procedures
- Birth Resuscitation





Birth Transition



Vital Birth Transitions

- Cardiovascular responsiveness
- Systemic blood pressure changes
 - Transition from fetal circulation
- Establishment of respiration
- CNS responsiveness

Breathing at Birth

- Fetal breathing
- Stimulate sustained rhythmic respiration
 - Catecholamine surge
 - Induction of substances important for breathing
 - Substance P
 - Removal of placental
 - Humoral inhibitory factors
 - Cooling
 - Tactile stimulation
 - Rising CO₂

Apnea at Birth

- Birth asphyxia
- Maternal drugs
- CNS injury
- Septicemia
- Muscular or neurological disease
- Obstructing congenital malformations
- Other mechanical obstruction



Neonate Not Breathing at Birth

Monitor heart rate

- Birth bradycardia normal
- Is it accelerating?
- Is the bradycardia persistent?

Birth arrhythmias are very common

- Perfusing? monitor
- Nonperfusing? resuscitate

Birth Bradycardia

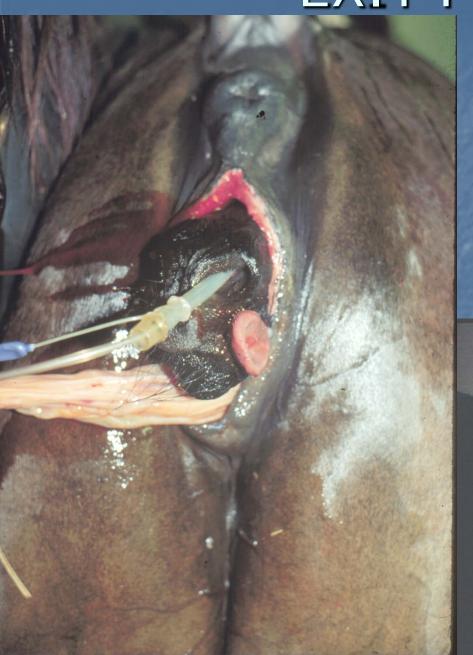


Unsuccessful Birth Transition

- Moderate to severe asphyxia
 Maternal systemic, placental, fetal, or intrapartum disease
- Fetal response reinforcing fetal circulation
- Neonatal response reverting to fetal pattern
- Development of primary then secondary apnea
- If not breathing at birth assume secondary apnea

Preparation for Resuscitation

- Anticipation
- High risk situations
 - Obvious from history
 - Intrapartum course
- Unexpected
 - 50% of neonates requiring birth resuscitation
- Must always be prepared
 - Well thought out plan
 - Readily available equipment



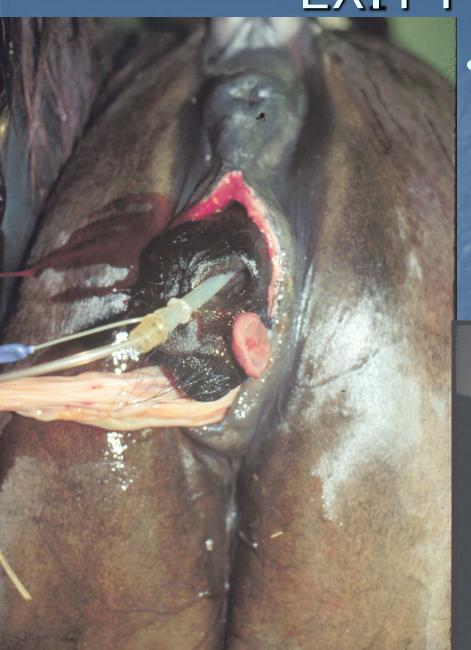
Ex-utero Intrapartum Treatment





- Resuscitation during parturition
 - •Oxygen therapy for the mare
 - •Fetal ECG
 - •Resuscitation during parturition





- Resuscitation during parturition
 - Intubate if nose is available
 - Use capnograph
 - Expect initial poor lung perfusion





- Resuscitation during parturition
 - Cannot in all cases
 - If can, then the pressure is off
 - Can make the difference







EXIT

- Luxury of time to correct the dystocia
- Assess fetal viability
- Rescue foals during dystocia
- Increase successful referral radius



Pitfalls of EXIT Procedure

- Latrogenic hyperventilation
 - ETCO₂ will decrease
 - Decrease cerebral perfusion
- Thoracic pump effect
 - Foal is dead but ETCO₂ > 0
 - If not dead but CO failing CPR
- Look beyond monitor to patient





Elements of Birth Resuscitation

- Initial Assessment
 - Apgar score
- Clearing the Airway
- Tactile Stimulation
- Thermal management
- Free Flow Oxygen
- Positive Pressure Ventilation
- Chest Compressions
- Medication

Initial Assessment



APGAR Score

Current Researches in Anesthesia and Analgesia-July-August, 1953

A Proposal for a New Method of Evaluation of the Newborn Infant.*

Virginia Apgar, M.D., New York, N. Y.

Department of Anesthesiology, Columbia University, College of Physicians and Surgeons and the Anesthesia Service, The Presbyterian Hospital



ESUSCITATION OF INFANTS at birth has been the subject of many articles. Seldom have there been such imaginative ideas, such enthusiasms, and dislikes, and such unscientific observations and study about one clinical picture. There are outstanding exceptions to these state-

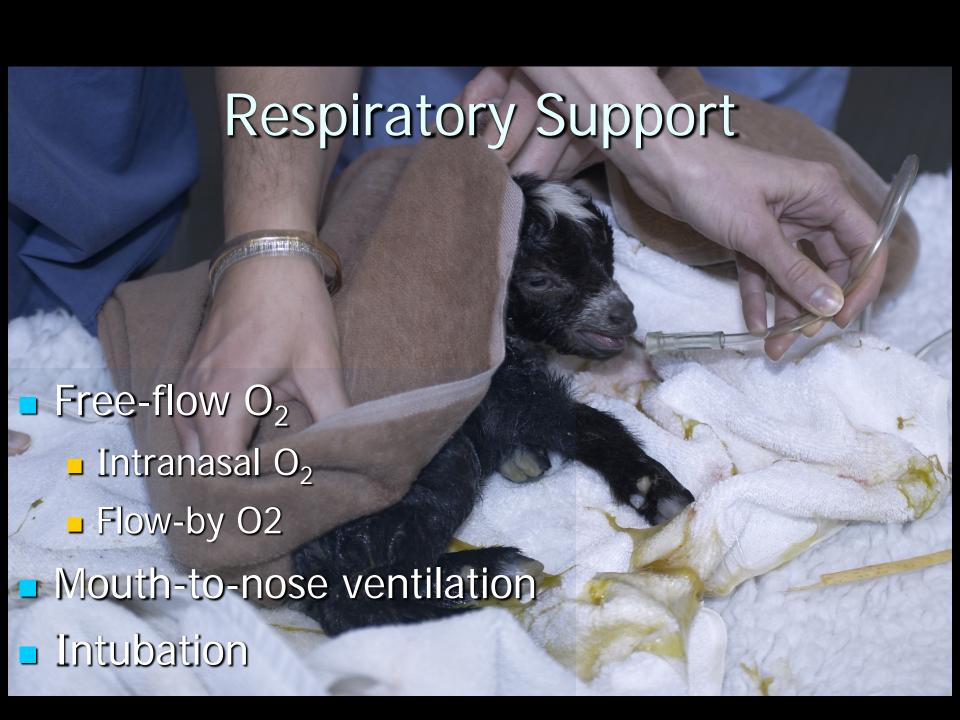
ments, but the poor quality and lack of precise data of the majority of papers concerned with infant resuscitation are interesting.

There are several excellent review articles¹ ² but the main emphasis in the past has been on treatment of the asphyxiated or apneic newborn infant. The purpose of this paper is the reestablishment of simple, clear classification or "grading" of newborn infants which can be used as a basis for discussion and comparison of the results of obstetric practices, types of maternal pain relief and the effects of resuscitation.

The principle of giving a "score" to a patient as a sum total of several objective findings is not new and has been used recently in judging the treatment of drug addiction.³ The endpoints which have

APGAR Score Foal

Score	0	1	2
Heart Rate	Absent	< 60	> 60
		Irregular	regular
Respiratory Rate	Absent	irregular	regular
Muscle Tone	Limp	Some	Active
Muscle Tone	Limp Lateral	Some Flexion	Active Sternal
Muscle Tone Reflex			
	Lateral	Flexion	Sternal



Mouth-to-Nose Ventilation



If the foal does not breath spontaneously

Ventilation

- Self-inflating bag valve device (O₂ Rx?)
- Never spontaneous ventilation
 - Establish FRC
 - Prolonged inspiration phase 1st breath 5 sec
 - Appropriate tidal volume
- Then 20-30/min hyperventilate?
 - Unless require CPR (< 10/min, rapid)</p>
 - Avoid more than mild hyperventilation
- If early in asphyxia
 - 30 sec ventilation will increase HR
- If late myocardium failing
 - Need chest compression

Birth Resuscitation CPR



Just because we can...





doesn't mean we should!

CPR Steps

- Immediate recognition
- Emergency response team
- Advanced life support
 - Correct ventilation 8-10/min
 - Airway, Ambu
 - Effective chest compressions
 - Drugs
 - Venous access
 - Monitoring
 - ECG, capnography
 - Defibrillation
- Post resuscitation care







Effectiveness of Chest Compression Cardiac Output

Feel central arterial pulse

Monitor pupil size

Measure end-tidal CO2

Intratracheal Drug Administration

- Epinephrine
- Lidocaine
- Naloxone
- Reservoir effect



Intraosseous Route Drugs and Fluids

- Easy rapid vascular access
 - Especially in kids, lambs, cria
- Requires some practice in larger neonates
- More reliable drug delivery than IT
- Special needle
 - Foal, calf
 - Kid, lamb, cria or premature not required
- Difficult to stabilize

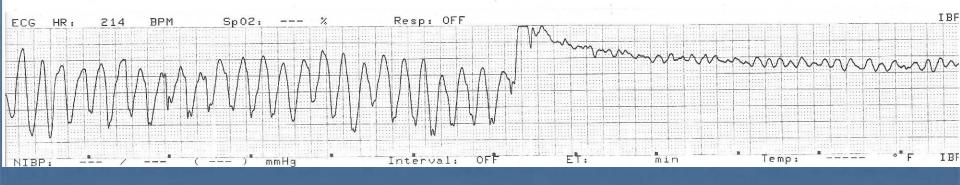


CPR Case Series

- Not breathing and non-perfusing rhythm
 - CPR attempt > 5 min
 - Not EXIT unless continued CPR after birth
- Total episodes 137 in neonates
- At birth 93 high risk births
 - 6% of 1568 attended births
- After birth 44
 - 1.6% of 2644 neonatal admissions

Foals

Rhythm	Episodes	ROSC	Survival
All episodes	84	41 (49%)	15 (20%)
Cardiac Arrest	49 (63%)	17 (35%)	5 (11%)
Non—perfusing Brady	29 (37%)	24 (83%)	10 (42%)
At Birth	46 (56%)	25 (54%)	12 (31%)
After Birth	38 (45%)	17 (45%)	4 (11%)



At Birth Resuscitation Initial Rhythm

Rhythm	Cases	ROSC	Survival
All	43 (46)	25 (54%)	12 (33%)
Brady	22 (51%)	19 (79%)	10 (59%)
V-fib	3 (7%)	1 (33%)	0
V-tach	3 (7%)	1 (33%)	1 (33%)
Shockable	6 (14%)	2 (33%)	1 (20%)
Asystole	11 (26%)	1 (9%)	0
PEA	4 (9%)	2 (50%)	1 (33%)





Birth Resuscitation

Jon Palmer, VMD, DACVIM Neonatal Intensive Care Service New Bolton Center, University of Pennsylvania Kennett Square, PA, USA

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 days to weeks (depending on the species) 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 subcapsular nephrogenic zone to the juxtamedullary area 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 CO₂ 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 CO₂. 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 Pao₂ 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, which 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, 9 mm internal diameter; 8 mm, 7 mm, 10 mm tubes may also be useful depending on breed and maturity) and a self-inflating bag. 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. The use of EXIT procedures (EXtrauterine Intrapartum Treatment procedures) can dramatically 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 CO₂ (ETCO₂) 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.

If EXIT procedures are attempted, the resuscitator should be well aware of the pitfalls. The most common iatrogenic error in performing EXIT is hyperventilation which decreases the $ETCO_2$ and poses the danger of decreasing cerebral perfusion. The inexperienced resuscitator may interpret the decrease in $ETCO_2$ as an indication of failing cardiac output and may continue aggressive ventilation. It is much safer to decrease ventilation in the face of a falling $ETCO_2$. If the $ETCO_2$ increases with slowed ventilation, hyperventilation is the problem and has been corrected. If not, the more aggressive resuscitation such as intratracheal epinephrine can be used in an attempt to increase cardiac output. There are also cases where quite high $ETCO_2$ values may be obtained from dead foals. This can occur when ventilation results in a thoracic pump, resulting in blood circulation expelling the high level of CO_2 from the dead foal's blood. The induction of the thoracic pump by ventilation may also be helpful if it occurs before the foal has died but when the foal's cardiac output begins to fail.

Elements of Resuscitation

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 it is nonperfusing, full CPR should be initiated. If the heart rate is perfusing, 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
Response to Stimulus	No Response	Grimace	Sneeze/Cough
		Weak Ear Flick	Ear Flick/Head Shake

Score: 7-8 = Normal; 5-6 = Mild to moderate asphyxia - stimulate, intranasal O₂; 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. 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 not be used except in very unusual situations.

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.

Free Flow Oxygen – If the neonate has spontaneous respiration but has bradycardia, irregular respiration or is cyanotic, free flow oxygen (intranasal) can be useful. I feel that 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. Routine use oxygen therapy is not indicated and may be harmful.

Positive Pressure Ventilation - Neonates who don't initiate spontaneous ventilation or who develop a nonperfusing bradycardia should be ventilated. Foals are 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. 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 led 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. But 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 – In a series of 1568 high risk births I have attended, 93 (6%) required CPR. 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 not the elbows of the attendant (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 needed. 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 hypoxic-ischemic encephalopathy and should be avoided in neonates with possible perinatal hypoxia. There is evidence that rapid respiratory rates during CPR are detrimental and more effective tissue oxygen delivery will occur at a rate of 1 breath 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 4 to 6 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.

OUTCOME

Foals who require CPR at birth have a better chance of survival to hospital discharge than foals requiring CPR later in the neonatal period. In our experience 54% of foals receiving CPR at birth (6% of high risk births) will return to spontaneous circulation and 31% will survive, whereas although 45% of foals receiving CPR not associated with birth will have return to spontaneous circulation only 11% will survive to hospital discharge.

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 and vasopressin can also be helpful in supporting cardiac output.