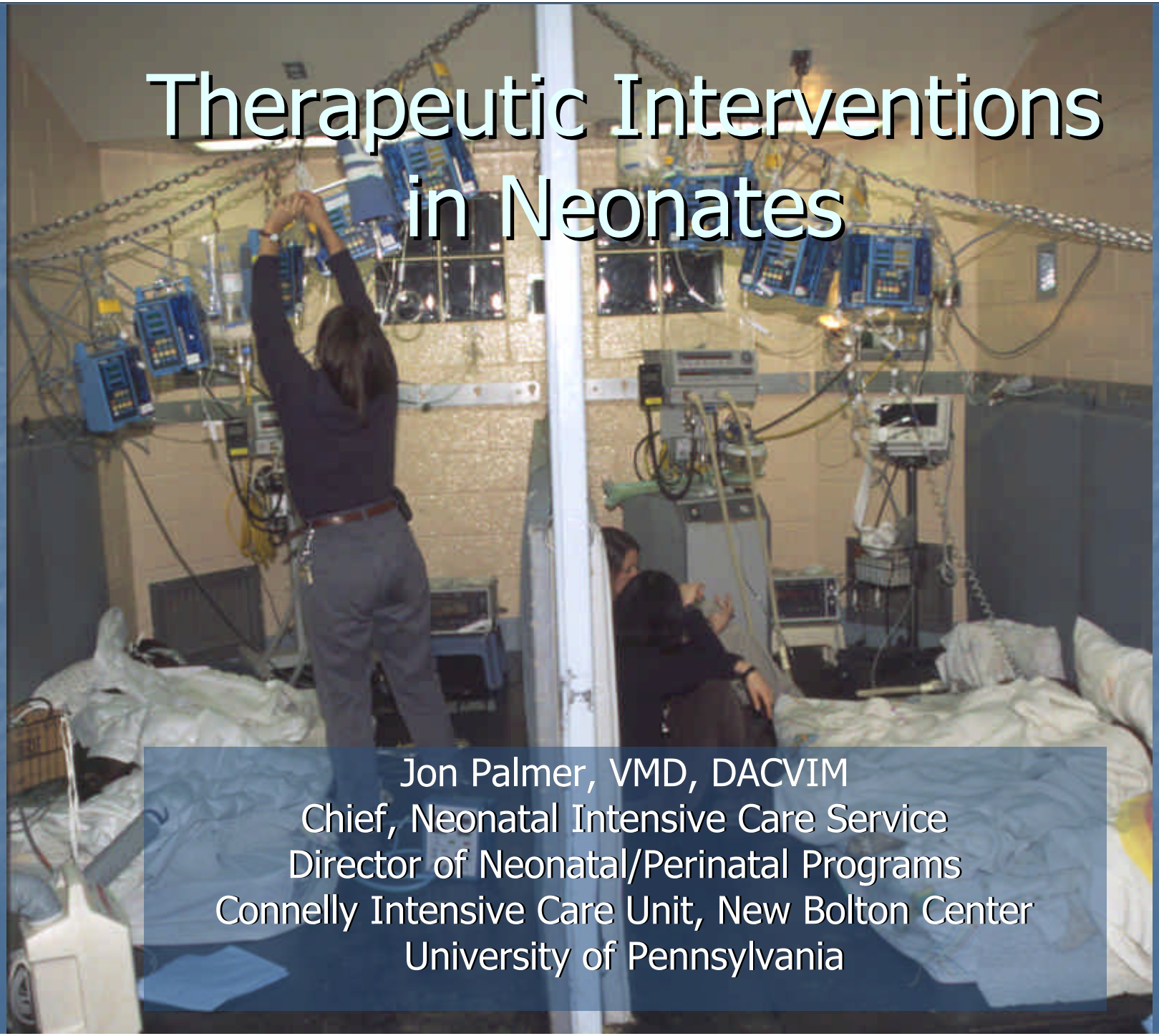
Therapeutic Interventions in Neonates



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Resuscitation of the Seriously Compromised Foal

- Rapid intervention
- Intensive intervention
- On Farm
- At referral center
  - Rapid transport
    - In a car
  - Short travel time
    - < 2 hours – don't treat - send
    - > 2 hours – begin treatment

Resuscitation on the Farm

- Delay in transportation
- Delay in decision making
- Lack of referral center availability
- Economic constraints
- Level of care on farm depends on
  - Environment/Facilities available
  - Experience/Energy of the help
  - Time constraints on the clinician
  - Availability of equipment

Resuscitation of the Seriously Compromised Foal

- Treat sepsis
- Stabilize blood glucose
- Respiratory support
- Insure tissue perfusion
  - Fluid therapy
- Deliver cerebral support
  - Control seizures
- Aid thermogenesis
- Correct metabolic abnormalities
- Spare renal work
- Deliver nutrition – oral/parenteral
- Give general supportive care

Treat Sepsis

- Plasma transfusion therapy
- Antimicrobial
  - Based on likely sensitivity
  - Community isolates vs. nosocomial isolates
  - Avoid
    - Commonly used antimicrobials
    - Toxic effects

Community Acquired Isolates

- 22% *E coli*
- 19% *Enterococcus*
- 19% *Pantoea agglomerans*
- 5% *Klebsiella*
- 5% *Streptococcus*
- Others
  - *Acinetobacter* , *Aeromonas,* Alpha *Strep*
  - *Burkholderia, Listeria, Mannheimia*
  - *Comamonas, Salmonella, Staphylococcus*
- 60% Gram-negative and 40% Gram-positive

Nosocomial Bacterial Isolates

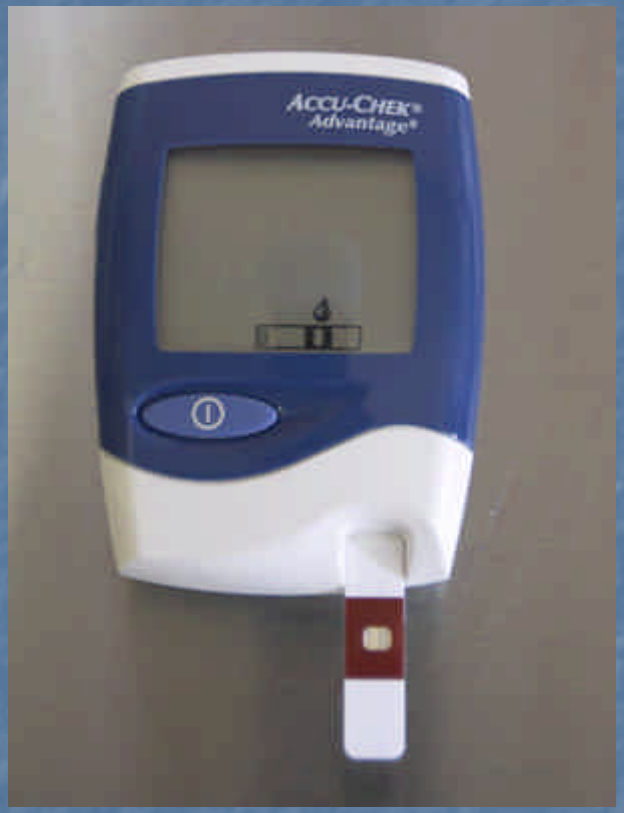
- 23% *Enterococcus*
- 18% *E coli*
- 11% *Enterobacter cloacae*
- 9% *Acinetobacter baumannii , Salmonella*
- 7% *Pantoea agglomerans, Pseudomonas*
- 5% Coag neg *Staphylococcus*
- 4% *Klebsiella pneumonia, Streptococcus*
- Others
- 68% Gram-negative and 32% Gram-positive

Antimicrobial Choices

- Community acquired infection
  - Ambulatory patient, controlled sepsis
    - Cefuroxime
    - TMS - IV
  - Critically ill neonate, uncontrolled sepsis
    - Ceftiofur Na - IV
      - 10 mg/kg IV QID
      - Continuous rate infusion (CRI)
- Nosocomial infection
  - Penicillin and amikacin – IV
  - Ticarcillin with clavulancic acid - IV

Glucose Therapy

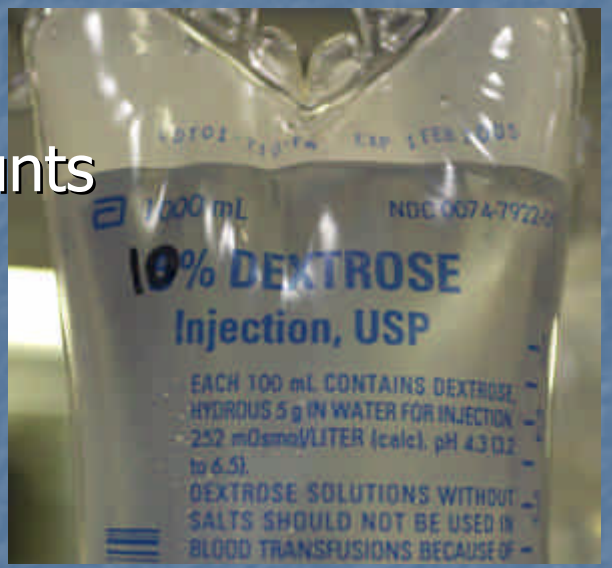
- Bedside monitoring – Glucometers
- Levels reflect homeostasis
  - Not availability
- Normal values
  - Birth – ½ maternal glucose
    - 1.87-2.78 mmol/l, 30-50 mg/dl,
  - Drop - low point 2 hrs after birth
  - Increase with time/nursing



- High glucose levels at birth (> 3.89 mmol/l, 70 mg/dl)
- Low glucose levels at birth (< 1.11 mmol/l, 20 mg/dl)

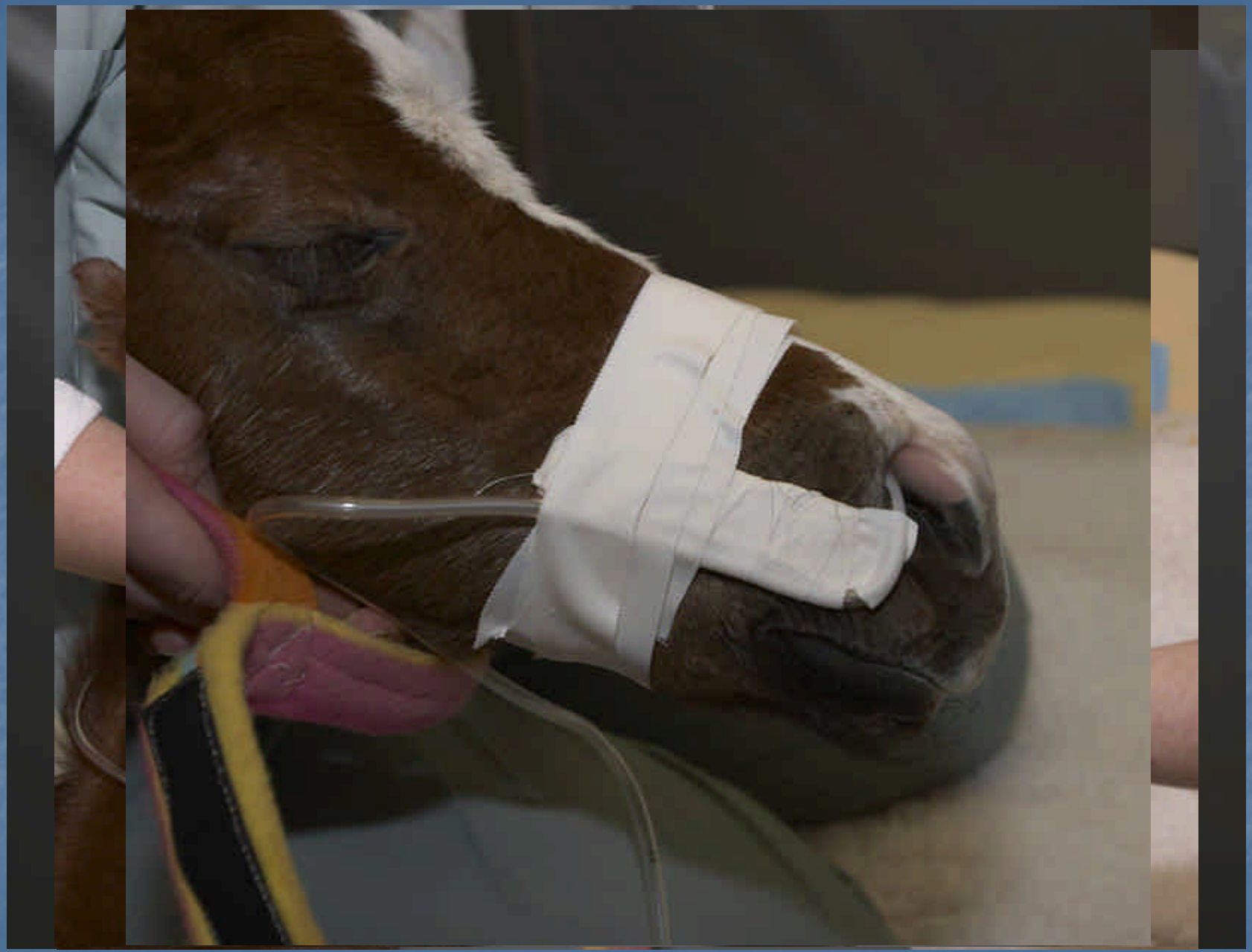
Glucose Therapy

- All compromised neonates
  - Will benefit from glucose therapy
- Placental glucose transport
  - Equine delivers 3.78 mmol/kg/min
  - Range between 2.2 – 4.4 mmol/kg/min
- Neonatal liver produces similar amounts
- Glucose therapy
  - Begin 2.2 mmol/kg/min
  - Goal of 4.4 mmol/kg/min
- Hyperglycemia - insulin therapy
- Hypoglycemia – hypermetabolism
- Glucose boluses
  - Metabolic anarchy
  - Often more harmful than continued hypoglycemia



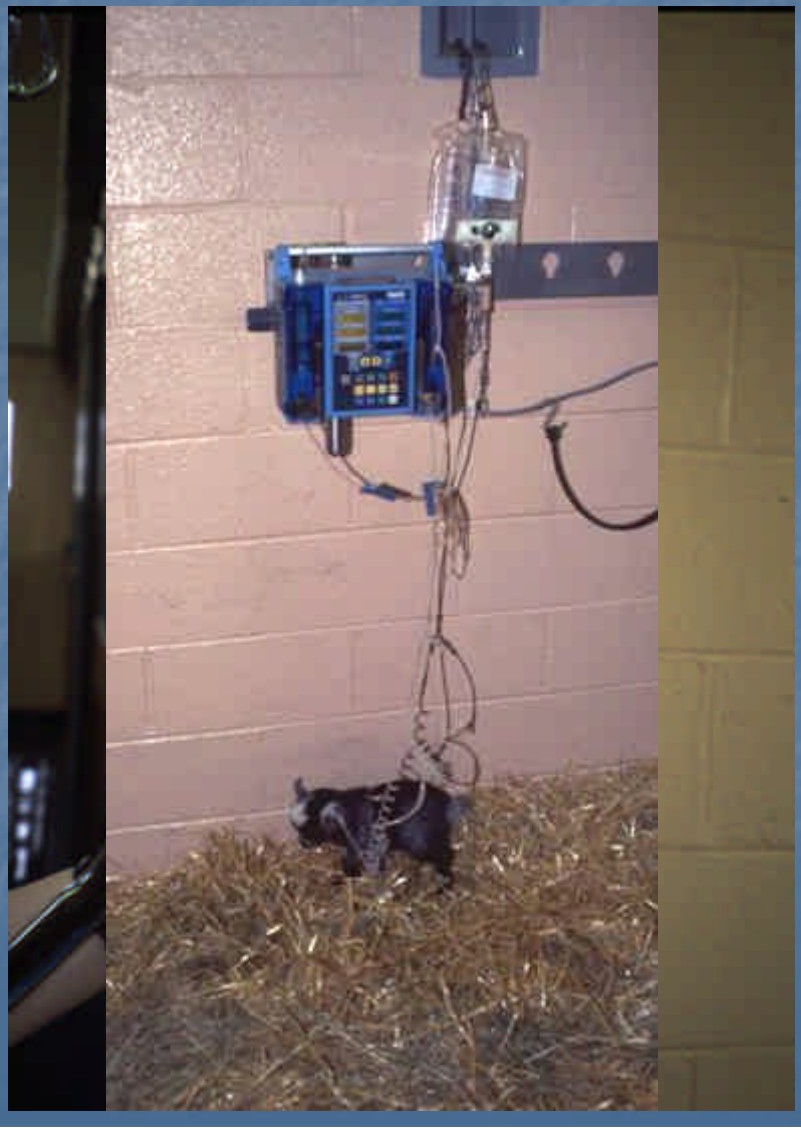
Respiratory Support

- Frequently hypoxemic
  - Ventilation perfusion mismatching
- Intranasal oxygen insufflation
  - Pa_{O2} < 60 torr (< 8 pKa)
  - SaO_2 < 90%
  - Goal
    - Pa_{O2} 80 - 110 torr (10.7-14.7 pKa)
    - SaO_2 > 92%
  - Nasal cannula
    - Flow rate of 6-10 lpm (2 to 15 lpm)
    - Preconditioned - water filled humidifier
- Central respiratory depression
  - Caffeine (10 mg/kg PO or PR)
  - Positive pressure ventilation.



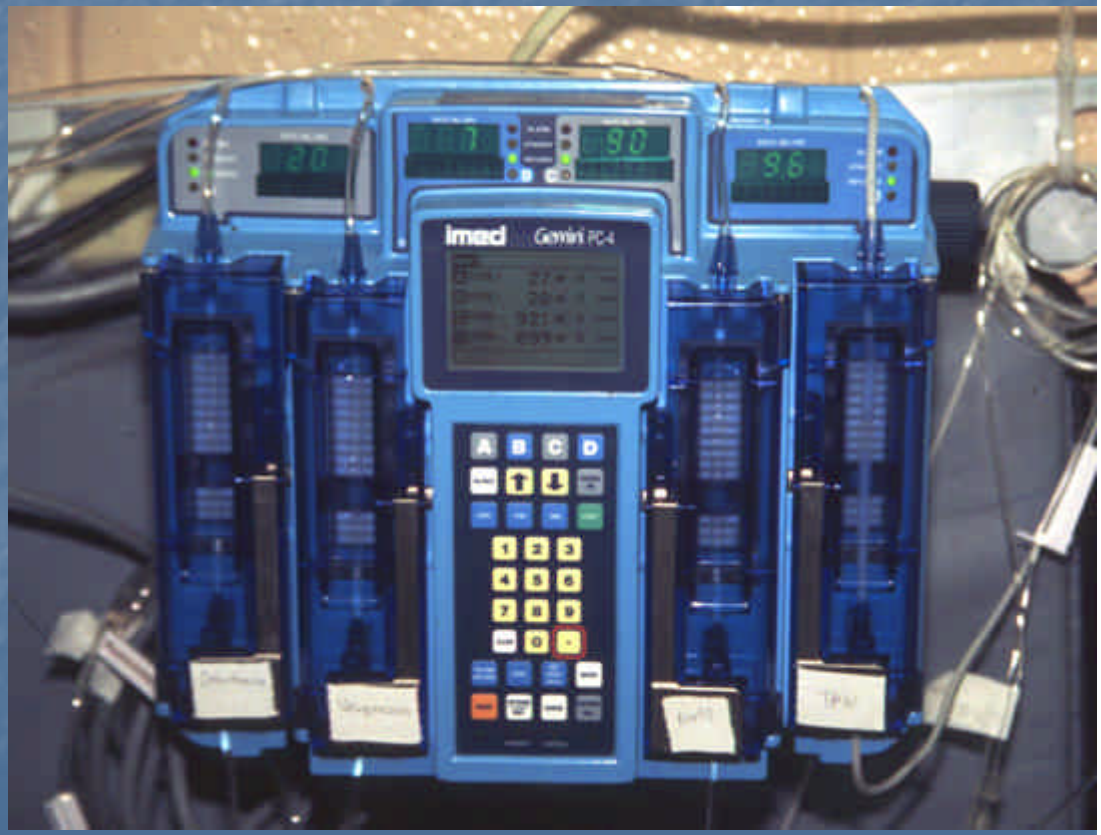
Fluid Therapy

- Hypoperfusion
  - Hypovolemia due to poor vascular tone
  - Almost never dehydrated
    - Hyperhydrated but hypovolemic
- Correct the hypovolemia
  - 20 ml/kg blouses over 10 to 20 minutes
- Maintenance fluids
  - 100 ml/kg/day for the 1st 10 kg weight
  - 50 ml/kg/day for the 2nd 10 kg weight
  - 25 mg/kg/day for each kg above 20 kg



Inopressor Therapy

- Inotrope and pressor therapy
  - Dopamine
  - Dobutamine
  - Epinephrine
  - Norepinephrine
  - Vasopressin
- Accurate CRI pumps

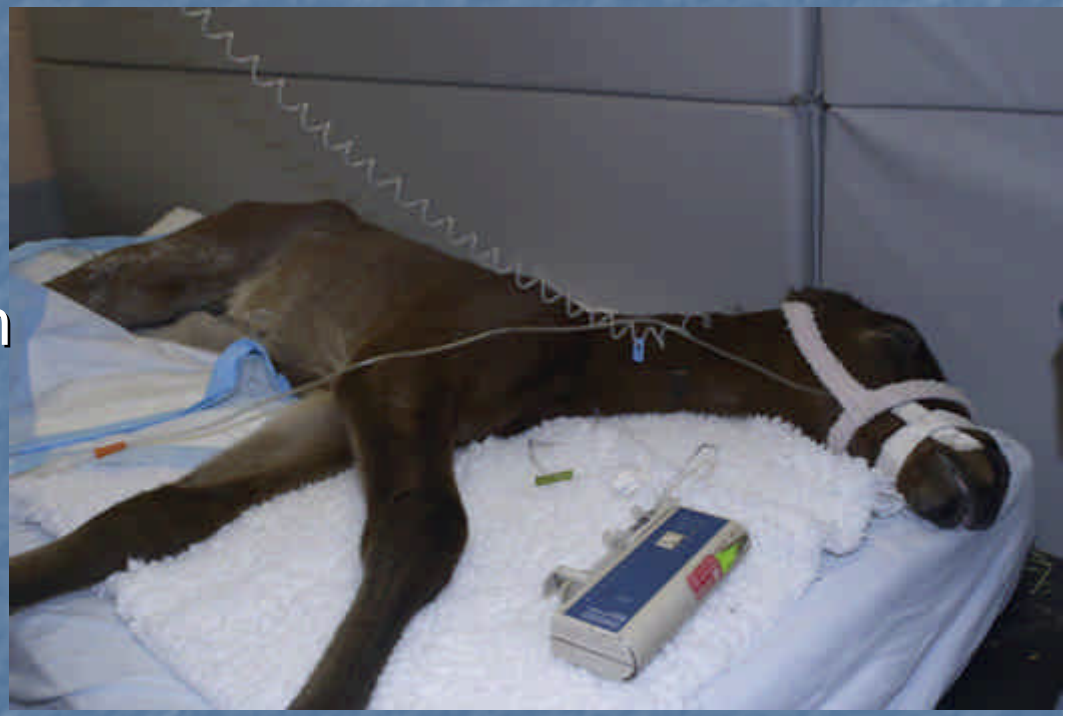


Thermogenesis

- Thermogenesis
  - Successful resuscitation
- Active warming
  - Contraindicated early treatment
  - Hot air blanket

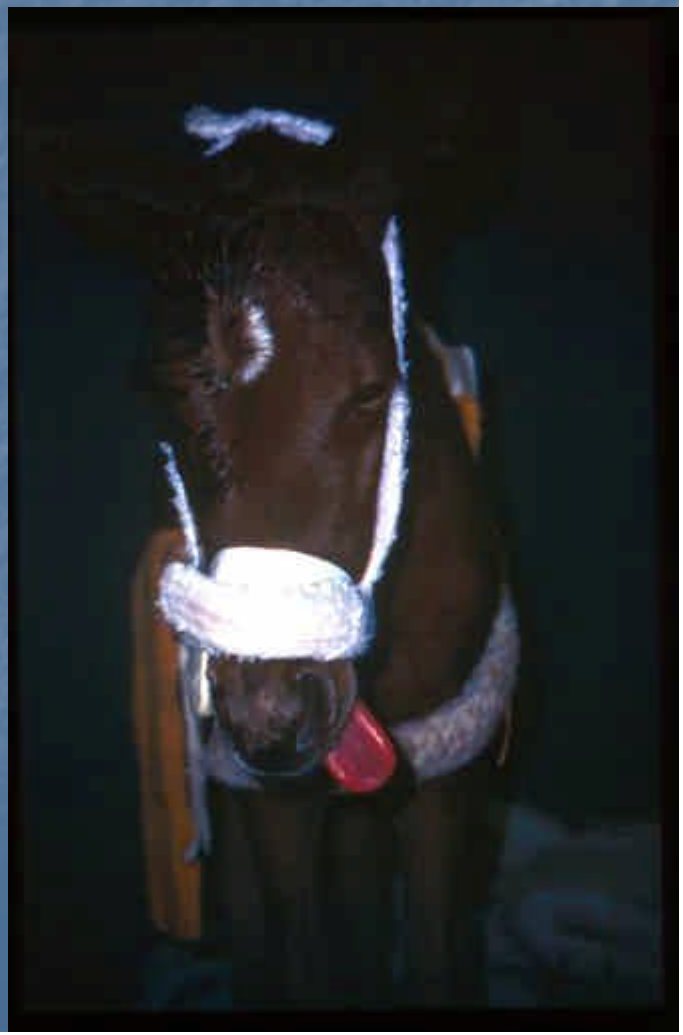
Seizure Control

- Phenobarbital
  - Hypothermia
  - Hypercapnia
  - Hypotension
  - Infused over 15-20 min
  - Half-life of >200 hrs
- Phenytoin
- Others
  - Diazepam
  - Midazolam



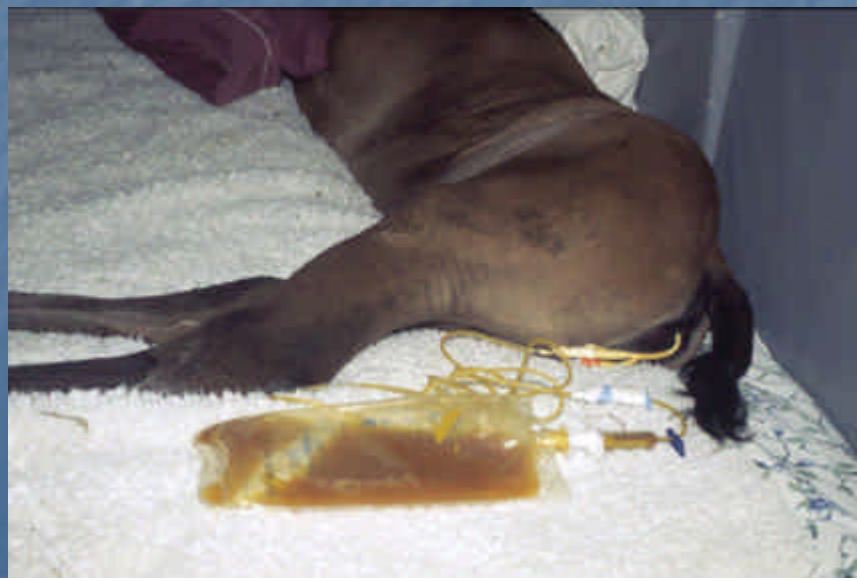
Cerebral Support

- Maintaining cerebral perfusion
  - Fluid replacement
  - Maintaining adequate BP
- Thiamine
- MgSO4
- Not used
  - DMSO
  - Mannitol



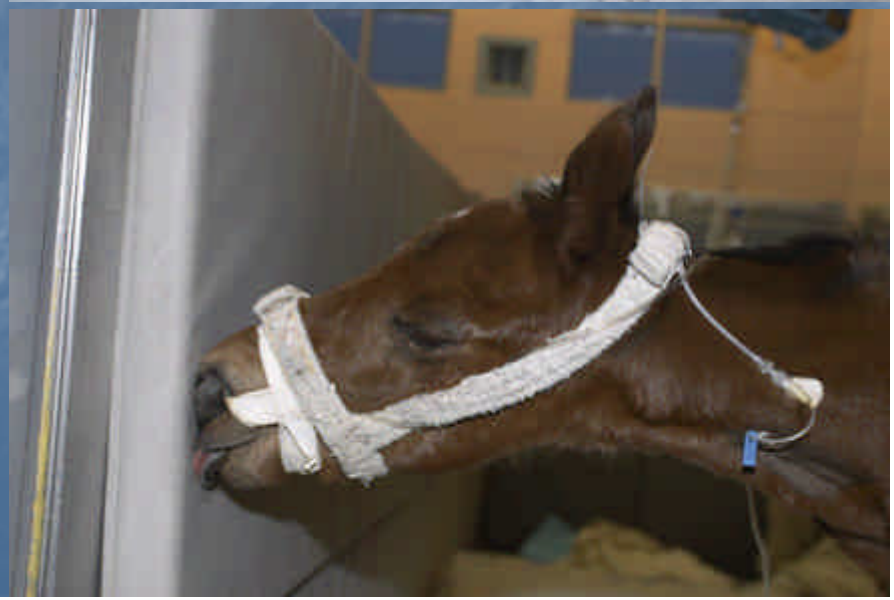
Renal Function

- Neonatal diseases target
- Normal neonatal kidney
  - Fluid handling
  - Sodium regulation
- Goal - minimize renal work
  - Regulating fluid balance
  - Regulating sodium balance
- Fluid and Na overload
  - Inappropriate weight gains
  - Development of edema
- Drugs to avoid
  - Flunixin meglumine
  - Aminoglycoside antimicrobials
    - Unless blood levels are measured



Oral Nutrition

- Colostrum
  - Large volumes
- Critical neonate
  - Hypoxemia, hypoperfusion
  - Hypoglycemia, hypothermia
  - Can't support enterocytes
- Criteria for feeding
  - Pao_2
  - Blood glucose
  - Perfusion
  - Core temperature is >37.8 C
  - Borborygmi present
  - Meconium is being passed



Oral Nutrition

Oral Nutrition
What should be fed?

Indwelling Enteral Feeding Tube

Ulcer Prophylaxis
Reasons not to suppress acid

- Sick neonates produced little acid
- Acid blockers have a decreased efficacy
- Gastric ulcer pathogenesis
  - Acid plays a minor role
- Acid is protective against nosocomials
  - Should not be suppressed or neutralized
- Ulcer prophylaxis not affect incidence of ulcers
- Occurrence decreasing
  - More effective supportive therapy for neonates

Summary

- Treat sepsis
- Maintain blood glucose homeostasis
- Maintain fluid balance
- Keep the patient warm
- Give respiratory support
- Maintain tissue perfusion
- Control seizures and support cerebral perfusion
- Maintain renal function
- Conservatively approach oral nutrition
- Deliver general supportive nursing care.

Avoid

- Excessive fluid
- Excessive sodium
- Aggressive warming
- Large volumes oral feeding
- NSAIDs (flunixin meglumine)
- DMSO
- Gastric acid blocking therapy

Therapeutic Interventions in Neonates

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Successful resuscitation of the seriously compromised foal requires rapid and frequently intensive intervention. Although this intervention can be successful on the farm, conditions and support on the farm make success difficult at best. The highest success rate occurs with rapid transport to a referral center with minimal intervention on the farm. If the travel time is short (< 2 hours) and those in attendance agreed to transport the neonate in a car (not waiting for traditional transportation) then the effort should be concentrated in getting the foal to the referral center. If the trip must be made in a horse trailer/Van, then an effort should be made to ensure that there is not further heat loss. If stabilization or definitive treatment at the farm is necessary because of the delay in transportation, delay in decision making, lack of referral center availability or economic constraints then there are several principles that should be kept in mind while instituting a therapeutic plan. The level of care that can be dispensed on the farm certainly varies depending on the environment/facilities available, the experience/energy of the help, time constraints on the clinician and availability of equipment. Even at the most advanced referral center, often compromises in what is ideal and what is possible must be made. The following outlines things to consider during neonatal foal resuscitation, with the realization that not all goals may be achievable.

Sepsis: Recognition and early treatment of sepsis is of paramount importance. Sepsis is the biggest killer of neonatal foals. It is commonly involved in prematurity having a role in placentitis. All compromised foals have increased susceptibility to secondary infections. Early treatment is very important in supporting the compromised neonatal foal. Treatment should include plasma transfusion therapy (even if the IgG level is considered adequate) and appropriate antimicrobials.

Antimicrobial choice should be based on likely sensitivity of pathogen (whether community or nosocomial pathogen), avoiding antimicrobials commonly used on the farm since the pathogen is more likely to be sensitive to these. Also care should be used to avoid antimicrobials with toxic effects involving compromised organ systems, such as aminoglycoside antimicrobials when renal compromise is suspected. Community acquired isolates from our practice between 2002-2004 include: 22% *E coli* , 19% *Enterococcus spp*, 19% *Pantoea agglomerans* , 5% *Klebsiella*, 5% *Strep* and others (*Acinetobacter lwoffi*, *Aeromonas caviae*, Alpha *Strep* not Gp D, *Burkholderia cepacia*, *Comamonas testosterone*, CDC Enteric Gr 76, *Listeria monocytogenes*, *Mannheimia haemolytica* A, *Salmonella*, *Staph*). Thus, as we have found for the past decade, about 60% of the isolates are Gram-negative and 40% Gram-positive, emphasizing the need for initial broad spectrum antimicrobials. Nosocomial bacteria isolates from our NICU patients during the same period (all isolates from neonates born in our NICU or isolates

from samples after the first 48 of hospitalization; primarily blood cultures but also umbilical, corneal, catheter, endotracheal tube cultures; excludes fecal cultures): 23% *Enterococcus*, 18% *E coli*, 11% *Enterobacter cloacae*, 9% *Acinetobacter baumannii*, 9% *Salmonella*, 7% *Pantoea agglomerans*, 7% *Pseudomonas aeruginosa*, 5% Coag neg *Staph*, 4% *Klebsiella pneumonia*, 4% *Strep* and others (CDC VE type 2, *Moraxella osloensis*, *Proteus vulgaris*). This represents 68% Gram-negative isolates and 32% Gram-positive isolates. These percentages are somewhat biased by a focal, limited *Salmonella* nosocomial outbreak.

Based on sensitivity patterns of our community acquired pathogens my first line therapy in ambulatory patients with evidence of controlled sepsis is a second generation cephalosporin (cefuroxime axetil 30 mg/kg/day PO divided BID/QID, Cefuroxime Na 50-100 mg/kg/day IV divided TID/QID) or IV TMS with or without a first generation cephalosporin. In the critically ill neonate (recumbent with evidence of uncontrolled community acquired sepsis) my first line therapy is high levels of a third generation cephalosporin (IV ceftiofur Na using much higher doses and frequency than commonly accepted; 10 mg/kg IV QID or continuous rate infusion using 1.5 mg/kg/hr). When faced with a nosocomial infection, again based on current sensitivity, I use penicillin and amikacin (K or Na Penicillin at 20,000-50,000 U/kg IV QID; amikacin in foals < 1 wk old at a dose of 30-35 mg/kg IV SID tailoring the dose to result in a 30 minute peak level of >60 µg/ml and a trough < 2µg/ml) or ticarcillin with clavulancic acid (50 - 100 mg/kg IV QID or 2-4 mg/kg/hr IV as a continuous rate infusion). Further discussion of choices, pharmacokinetics and variations from adult therapy is beyond the scope of this article.

Blood Glucose: Glucometers made for diabetic people make stall side measurements of whole blood glucose levels simple and inexpensive. At birth the whole blood glucose (WBG) is approximately half of the maternal glucose (30-50 mg/dl, 1.87-2.78 mmol/l) and will initially drop as the foal makes the transition to glucogenesis with a low point at about 2 hours after birth. This is followed by a rise, often enhanced by nursing, and stabilization in the normal range. Foal's born with WBG levels > 70 mg/dl (3.89 mmol/l) may be born to a hyperglycemic dam, may be responding to extreme stress or may have had intrauterine gluconeogenesis secondary to catabolism (often secondary to placentitis or IUGR). A concurrent measurement of the mare's WBG will help rule out the first possibility. Foals born with WBG values < 20 mg/dl (1.11 mmol/l) are often compromise and should be watched closely. Those which drop below 10 mg/dl (0.56 mmol/l) should be supplemented with intravenous glucose infusion, although often they are surprisingly asymptomatic.

Blood glucose levels reflect glucose homeostasis and not availability. All compromised neonates will benefit from glucose therapy, no matter what their WBG value. If an external source of energy is not available the neonate will utilize glycogen reserves and then become catabolic, utilizing already limited resources. If hypoglycemic, the neonate is not mobilizing adequate glucose rapidly enough to meet needs and usually will adapt to exogenous glucose delivery immediately. But normoglycemic foals that are producing glucose at the required rate and hyperglycemic foals with poor glucose control may require some time to adjust to exogenous glucose infusion. Both will benefit if they

adapt to the infusion since receiving external glucose will spare their own resources. I try to spare the caloric drain by delivering amounts of glucose equal to what is usually received from the placenta or produced by the neonatal liver. The equine placenta delivers 6.8 mg/kg/min (3.78 mmol/kg/min) of glucose with a range in most species between 4 to 8 mg/kg/min (2.2 – 4.4 mmol/kg/min) and the neonatal liver similar amounts. I begin therapy by delivering 4 mg/kg/min (2.2 mmol/kg/min; 50 kg foal 240 ml/hr of 5% glucose) with the goal if tolerated of 8 mg/kg/min (4.4 mmol/kg/min; 50 kg foal 240 ml/hr of 10% glucose). Some foals require time to adjust since they do not stop endogenous glucose production despite receiving significant exogenous amounts. This will result in hyperglycemia. This usually corrects itself given time if the glucose delivery rate is initially begun at the lower rate and not abruptly begun at the high rate before the foal can adapt to the infusion. Some foals, especially if severely compromised, may require insulin therapy. Foals that will tolerate glucose delivery usually will readily adapt to partial parenteral nutrition if required. Glucose containing fluids must be delivered continuously in a well-controlled manner, preferably by using an infusion pump, which makes delivery in a farm situation a challenge. Glucose boluses are counterproductive since they will cause significant hyperglycemia and may result in glucose diuresis resulting in loss of the infused glucose, fluids and electrolytes in urine, followed by rebound hypoglycemia. Glucose boluses are often more harmful than continued hypoglycemia.

Respiratory Support: Compromised neonates are almost always hypoxemic with spontaneous ventilation of room air. Often ventilation perfusion mismatching caused by weakness (secondary to hypoglycemia, neurologic disease), a compliant chest wall, prolonged lateral recumbency and poor perfusion is the underlying cause. Foals suffering from perinatal diseases often have recurrent bouts of hypoxemia with or without hypercapnia. Hypoxemia should be treated to avoid ongoing damage. Usually intranasal oxygen administration is sufficient. Oxygen therapy is indicated in any neonate with a Pa_{O2} < 60 torr (< 8 pKa) or a Sa_{O2} < 90%. The goal of oxygen therapy is to maintain the Pa_{O2} between 80 and 110 torr (10.7-14.7 pKa), and a Sa_{O2} > 92%. The usual technique for delivering oxygen insufflation involves the use of a nasal cannula with the tip at the level of the medial cantus of the eye and several auxiliary openings closer to the external nares because nasal discharge often occludes the openings. The nasal line can be secured to the nose using tape and a tongue depressor to act as a brace and to direct the line so that the foal has difficulty displacing it. The oxygen should be delivered at a flow rate of 2 to 15 liters per minute (6-10 lpm most commonly needed) and preconditioned by using a water filled humidifier (do not use saline!) controlled by a flowmeter. The Fi_{O2} resulting from intranasal oxygen cannot be predicted easily because it will vary with placement of the nasal cannula, patency of the cannula openings, tidal volume, minute volume and size of the foals nares.

Blood pH should also be monitored and maintained in a normal range. Frequently a metabolic alkalosis develops resulting in an appropriate physiologic hypercapnia, which corrects the pH. It is contraindicated to correct the hypercapnia. Rather this problem should be approached by correcting the cause of the metabolic alkalosis. Neonatal encephalopathy may result in central respiratory depression. Some of these foals will

respond adequately to a central respiratory stimulants such as caffeine (10 mg/kg PO or PR) while others require positive pressure ventilation.

Fluid Therapy: Hypoperfusion in the critical neonate is usually secondary to hypovolemia due to poor vascular tone. Sick neonates are almost never dehydrated during the first 48 hours of life unless there is significant diarrhea, reflux or GI tract pooling of fluids, in rare cases of polyuria or when there are high insensible losses as in extremely hot weather when tachypnea is present. In fact, neonates which have been subjected to intrauterine stress are usually born overhydrated because of intrauterine fluid shifts. Critical neonates presenting during the first 48 hours of life are often hyperhydrated but hypovolemic. So when approaching fluid therapy, it is essential to administer high enough volumes to correct the hypovolemia but to use care not to give excessive fluid volumes which will further exacerbate the hyperhydration. In the critically hypovolemic patient, delivering 20 ml/kg blouses of fluids over 10 to 20 minutes allows deliver the fluids in a timely manner but also imposes a set time for reassessment (after completion of each bolus) so as to achieve rapid return of perfusion while avoiding excessive fluid delivery. Thus, in a typical 50 kg foal, a 1 liter bolus is given rapidly (usually over 10 minutes with the aid of a pressurized cuff) and once delivered, a rapid assessment of return of perfusion (peripheral body temperature, pulse quality, return of signs of organ function) is used to decided if the bolus should be repeated. One or more liters of plasma are also given for its colloid properties and for the bioactive proteins it contains.

Once volemia is achieved, fluid administration should be slowed to a maintenance rate. There is no one maintenance fluid rate that fits all individuals. On the contrary, the amount of fluids needed depends on a number of factors including metabolic rate, insensible water losses (influenced by humidity and ventilatory rate), ambient temperature, gastrointestinal losses, and even the osmotic load introduced by medications. The clinician should not be afraid to adjust the fluid rate to the individual. The fluid volume formula that I use has a build an allowance for mass to surface area changes with body weight and calculates a "dry" fluid rate for large neonates. Fluid overload is potentially more of a problem than dehydration in most cases, justifying using a dry formula. I give 100 ml/kg/day for the 1st 10 kg body weight plus 50 ml/kg/day for the 2nd 10 kg of body weight plus 25 mg/kg/day for each kg above 20 kg (example: 50 kg foal = 1000 ml for 1-10 kg + 500 ml for 11-20 kg + 750 ml for next 30 kg = 2250 ml/day = 94 ml/hr).

Tissue Perfusion: Tissue perfusion and oxygen delivery to tissues must be maintained to avoid further damage. Adequate perfusion can be assessed by monitoring urine output, assessing peripheral perfusion (leg warmth, pulse quality), monitoring mental awareness as an indication of brain perfusion (of course Neonatal Encephalopathy may negate these observations), assess perfusion of bowel as indicated by normal GI function, etc. Often compromised foals who continue to have poor perfusion despite fluid resuscitation will benefit from inotrope and pressor therapy (e.g. dopamine, dobutamine, epinephrine, norepinephrine) or vasopressin therapy. To safely deliver these drugs constant monitoring and use of accurate continuous delivery infusion pumps are required. Usual

dose range include: dopamine 2-20 µg/kg/min. with a starting point in shock of 10 µg/kg/min.; dobutamine 2-40 µg/kg/min. with a starting point in shock of 10 µg/kg/min.; epinephrine 0.1-2.0 µg /kg/min. with a few difficult cases requiring 3 to 4 µg/kg/min.; norepinephrine 0.1-3.0 µg /kg/min. with a few difficult cases requiring 4 to 5 µg/kg/min.; vasopressin 0.25-1.0 mU/kg/min. Full description of inotrope/pressor therapy is beyond the scope of this article.

Thermogenesis: Thermoregulation is often deficient in a compromised neonate and thermogenesis does not keep up with heat loss. The best approach to the situation is to help the foal conserve whatever heat it is producing. This can be achieved by placing the foal in a thermoneutral to slightly warm environment with blankets, etc. Active external warming should be avoided in any neonate who is hypotensive or marginally so since it will induce vasodilation of skin and exacerbate the cardiovascular failure. Once perfusion becomes adequate, thermogenesis will increase raising core temperature without active warming, however some foals with continued poor thermogenesis will benefit from active warming. The most effect active warming is by using forced hot air as with a hot air blanket. This effect can be mimicked by using a variable output hair drier under a sheet, but care needs to be used to avoid burns.

Seizure Control: Cerebral oxygen use increases almost fivefold during seizures. Diazepam is helpful for emergency control of seizures, but causes respiratory depression. Phenobarbital is the standard of therapy, however, to avoid problems its side effects should be anticipated. Phenobarbital will cause a drop in core body temperature, a decrease in respiratory drive inducing hypercapnia and it may potentiate hypotension resulting in deterioration of perfusion. All of these side effects can be minimized by early intervention. Phenobarbital can be given repeatedly until seizures are controlled, infused over 15-20 minutes with a peak activity at 45 minutes. Once the seizures are controlled, in rare cases it may be necessary to repeat the dose in 6 to 12 hours. The half-life of phenobarbital in some foals may be >200 hours (others may have faster clearance) making maintenance unnecessary and even contraindicated. The degree of sedation achieved may be prolonged. If phenobarbital fails, phenytoin may be tried. At all costs ketamine and xylazine should be avoided, as they will cause cerebral hypertension. During seizures it is also important to protect the foal from injury.

Cerebral Support: Maintaining cerebral perfusion, achieved by careful fluid replacement and by maintaining adequate blood pressure is the most important therapy. The lesion from hypoxic ischemic insults is cellular edema and not cerebral edema. I occasionally treat with thiamine or $MgSO_4$, however their efficacy is unproven and in some cases $MgSO_4$ may be contraindicated. Mannitol or DMSO have been used by others. Over the past decade I have not used DMSO or mannitol and I have seen no difference in outcome, with uncontrolled clinical observations suggesting improved outcome without these therapies.

Metabolic Abnormalities: Compromised foals often have a variety of metabolic problems such as hypoglycemia/hyperglycemia, hypocalcemia/hypercalcemia, hypokalemia/hyperkalemia, hypochloremia/hyperchloremia, and various degrees of

metabolic acidosis/alkalosis. These problems should be addressed. Their therapy is beyond the scope of this article.

Renal Function: Many neonatal diseases target the kidney but even the normal neonatal kidney has trouble handling fluid and sodium loads. Neonatal challenges may result in altered renal blood flow patterns or acute tubular damage, despite common hyperhydration. Because of this, it is important to minimize renal work by carefully regulating fluid and sodium balance. Early signs of fluid and sodium overload include inappropriate weight gains and development of edema. There is little role of dopamine or furosemide in protecting the kidney or reversing kidney damage, but these drugs will enhance urine output and can be useful in fluid overload situations. Flunixin meglumine causes a number of adverse effects in neonates and is a drug to avoid. Compromised neonates have high resting arginine vasopressin levels. This hormone is important in regulating blood pressure and blood flow in the fetus and neonate. Its effect at the renal tubular cell is blocked in the neonate by normal renal parenchymal prostaglandin levels so the urine is not inappropriately concentrated. However, when NSAIDs are used, vasopressin's antidiuretic effect is uncovered. Neonatal foals given flunixin meglumine often stop urinating and produced very concentrated urine probably because of this mechanism. This will exacerbate fluid overload situations. Also the decrease in renal parenchymal prostaglandins will alter perfusion patterns and may lead to interstitial or tubular damage.

Oral Nutrition: Large volumes of colostrum should not be fed to a severely compromised neonatal foal. Often the original insult along with ongoing damage due to persistent hypoxemia, hypoperfusion, hypoglycemia, hypothermia, etc. has resulted in GI damage. In this environment, especially in the face of hypothermia, if the enterocytes and intestinal smooth muscle are asked to perform metabolic work (e.g. digestion, motility) without the substrate support for that work, more damage will result. In addition, it is unlikely that there will be significant absorption because of the damage. I generally withhold feeding until the Pao_2 and blood glucose are normal, systemic perfusion is adequate, the core temperature is > 100 F, borborygmi are present and meconium is being passed. Even then, if a significant GI insult was likely, I will only initially feed trophic amounts (1-2 % of body wt/day), which I hope will stimulate normal mucosa development through trophic hormones that are plentiful in colostrum. My general philosophy is to let the GI tract tell me when it is ready to handle feeding. Once the foal shows it can tolerate small feedings, I gradually increase feedings with the goal of intake at the level of 15-25% of their body weight. Having the neonate on partial parenteral nutrition allows for a slow introduction to larger oral feeding. Ideally the initial milk fed should be fresh colostrum. If fresh colostrum is not available, fresh mare’s milk is the best substitute. Milk replacer should be avoided if possible since it is hypertonic making it hard on the compromised intestinal mucosa. Delivery of milk to the foal with abnormal nursing behavior is best achieved using an indwelling enteral feeding tube designed for humans. This tube is small enough to cause minimal nasal/pharyngeal/esophageal irritation and can be secured in a manner similar to intranasal oxygen lines.

General Supportive Care: General nursing care is very important to prevent secondary problems and to speed recovery. A clean, dry, thermally regulated environment is important. Frequent position changes and physical therapy is also important. Close attention to details such as fecal production, urine production and changes in attitude can be very important. If at all possible, daily weights should be recorded. After discounting changes caused by fluid shifts, weight gain is the most accurate method of assessing the overall well-being of the patient.

Ulcer Prophylaxis: For at least the past 15 years, I have not routinely used ulcer prophylaxis in compromised neonates. There are at least six reasons that have convinced me not to use acid suppression therapy in compromised neonatal foals.

1. Sick neonates produced little acid.
2. H_2 blockers have a decreased efficacy in sick neonates.
3. Acid production plays a minor role in gastric ulcer pathogenesis in neonates.
4. Acid is protective against colonization of potential pathogens preventing nosocomial infections, so if acid is present it should not be suppressed or neutralized.
5. Ulcer prophylaxis does not affect the incidence of ulcers in sick neonatal foals. We performed a retrospective study supporting this observation.
6. The occurrence of gastric ulcers may be decreasing, as we are more effective in treating neonates. This is also reflected in a retrospective study we completed.

I believe, as in other species, that gastric ulcers found in our critical care cases are secondary to poor perfusion, hypoxic damage, proinflammatory damage and pathogenic flora which take advantage of these problems. As we have learned to better treat these cases, the incidence of ulcer disease has decreased. In my experience treating approximately 2000 critical care neonates, the incidence of fatal outcomes from secondary gastric ulcers (as distinct from the primary gastroduodenal ulcer syndrome) is very low. Although I'm sure others have seen more, I only recall one such case. This case had clear histological evidence of forming secondary to sepsis with invading pathogens that normally are suppressed by gastric acid.

In summary, when dealing with a compromised neonate: Treat for sepsis, maintain blood glucose homeostasis, maintain fluid balance, keep the patient warm, give respiratory support, maintain tissue perfusion, control seizures and support cerebral perfusion, correct metabolic abnormalities, maintain renal function with conservative fluid and sodium therapy, conservatively approach oral nutrition and above all deliver general supportive nursing care.

Things I avoid: Excessive fluid and sodium therapy, aggressive warming, large volumes of colostrum or other oral feedings until the GI tract is ready, NSAIDs (flunixin meglumine), DMSO, and gastric acid blocking therapy.