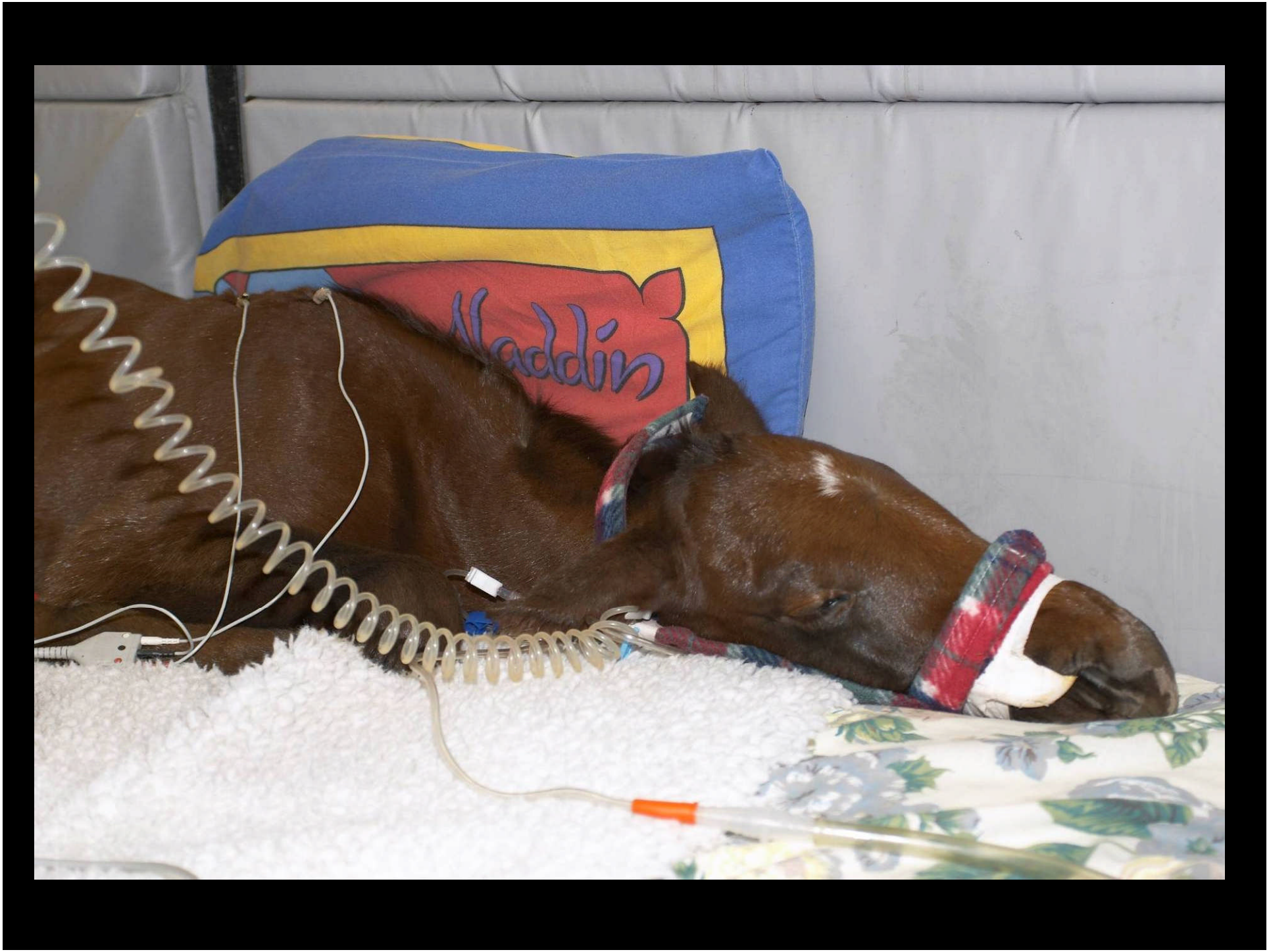
Respiratory Problems of the Neonate

Part 1

J. E. Palmer, VMD, DACVIM
Chief, Neonatal Intensive Care Service
New Bolton Center, University of Pennsylvania, USA


Aladdin

Respiratory Problems

- Neonatal cardiopulmonary physiology
- Pulmonary gas exchange
- Respiratory control
- Upper airway abnormalities
- Lower airway disease

Ventilation/Perfusion Abnormalities

- Fetal to neonatal cardiopulmonary physiology
  - Delayed transition
  - Failure transition
  - Reversion
    - Hypoxemia
    - Inflammatory mediators
    - Systemic hypotension

Ventilation/Perfusion Abnormalities

- Missmatching
  - Uneven perfusion
    - Poor cardiac output
    - Poor vascular reactivity to oxygen?
  - Uneven ventilation
    - Body position
    - Weakness
    - Fatigue
- Hypoventilation
  - Fatigue
  - Upper airway disease
- Progressive atelectasis

Progressive Atelectasis

- Unable to maintain FRC
  - Weakness/compliant chest wall
  - Stiff lungs
- Some alveoli collapse on exhalation
  - Repetitive collapse – eject surfactant
  - Some alveoli don't reopen
  - Closed alveoli pull on others
    - More alveoli close
- Decreased compliance
  - As more atelectasis
  - Causes more fatigue
- Self-perpetuating

Wave Chest Fatigue

- No longer be able to hold the chest open
- Inspiration
  - Diaphragm contracts
  - Chest wall pulled towards the lungs
  - Abdomen expands
- Expiration
  - Diaphragm relaxes
  - Chest wall moves out
  - Abdomen moves in
- Significant fatigue
  - Respiratory failure
  - Respiratory/cardiac arrest

Central Respiratory Control Neonatal Encephalopathy

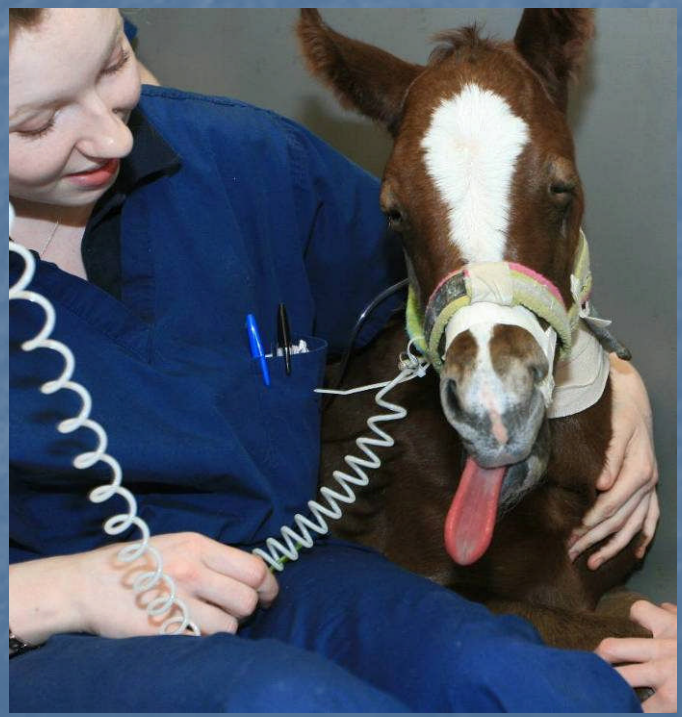
- Changes in respiratory patterns
  - Apneustic breathing
  - "Periodic breathing"
    - Cluster breathing
      - With periodic respiratory pauses
      - With apnea
    - Apnea (> 20 seconds)
      - Periods of up to 3-4 minutes
  - Ataxic breathing

Central Respiratory Control Neonatal Encephalopathy

- Inappropriate hypoventilation
  - Appropriate hypercapnia
    - Normal pH
    - Balancing a metabolic alkalosis
  - Respiratory acidosis - central
    - Inappropriate hypercapnia
  - Other reasons - mechanical
    - Upper airway obstruction
    - Weakness

Central Respiratory Control Neonatal Encephalopathy

- Inappropriate hyperventilation
  - Alkalosis
    - Central
    - Diagnosis by exclusion
  - Other reasons
    - Pain
    - Hypoxia
    - Stress
  - Hyperthermia
    - Temperatures as high as 42 C
    - Diurnal variability not present

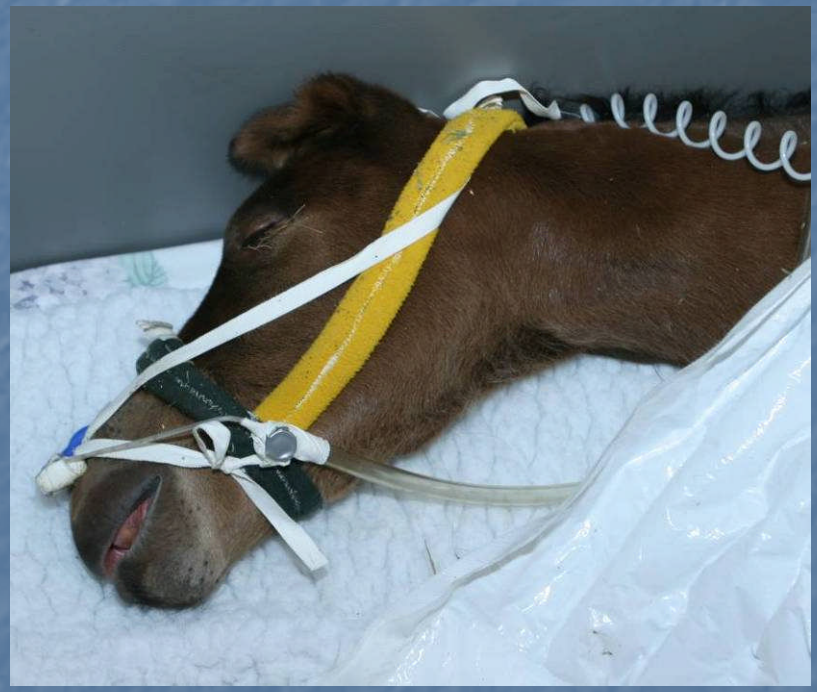


Upper Airway Abnormalities Transient Pharyngeal Paresis

- Hypotonic pharyngeal collapse
  - Etiology
    - Peripheral neuropathy?
      - Neonatal Encephalopathy?
  - Signs
    - Appear normal initially
    - Stressed
      - Respiratory rate and effort increase
      - Negative pressure in the pharynx
      - Pharyngeal collapse
      - Self perpetuating obstruction
    - Respiratory failure followed by cardiovascular failure
    - Dysphagia may present

Upper Airway Abnormalities Transient Pharyngeal Paresis

- Generalized weakness
  - Critically ill
    - Generalized hypotonia
    - Fatigue
    - Recumbent
- Phenobarbital
  - Major cause hypoventilation in treated cases
- HYPP (Hyperkalemic Periodic Paralysis)



Upper Airway Abnormalities
Upper Airway Obstruction

- Bilateral or unilateral choanal atresia
- Choanal hypoplasia
- Epiglottic cysts/malformations
- Wry nose/ facial deformities
- Guttural pouch
  - Tympany
  - Empyema
- Palate malformations

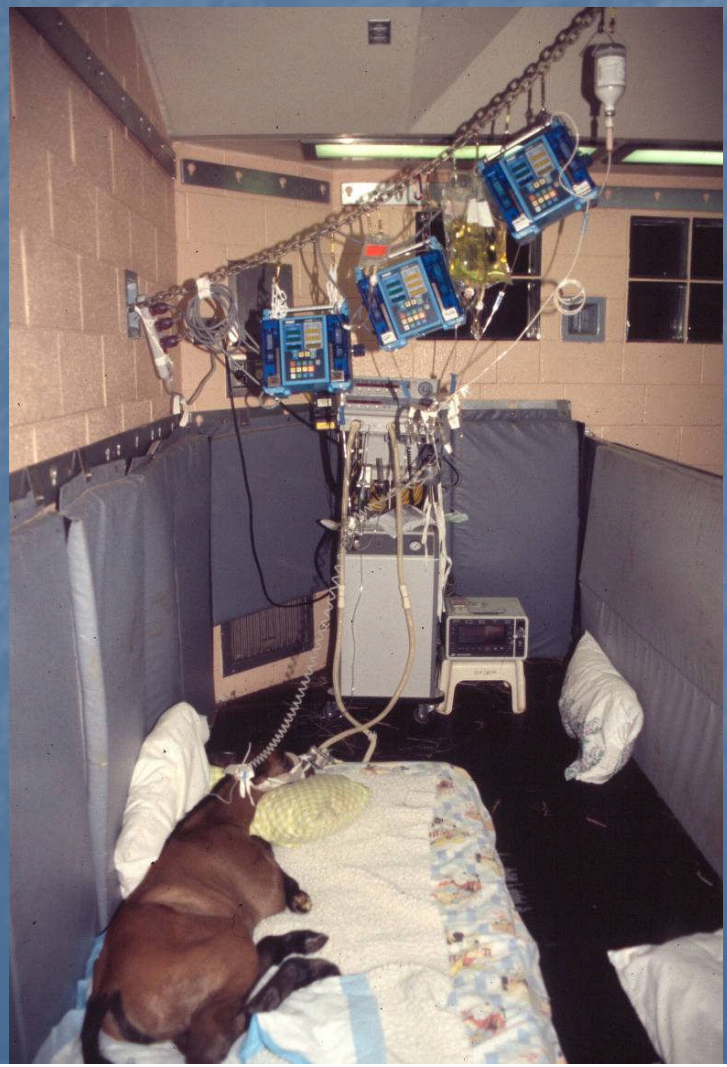
Respiratory Problems of the Neonate

Part 2

J. E. Palmer, VMD, DACVIM
Chief, Neonatal Intensive Care Service
New Bolton Center, University of Pennsylvania, USA

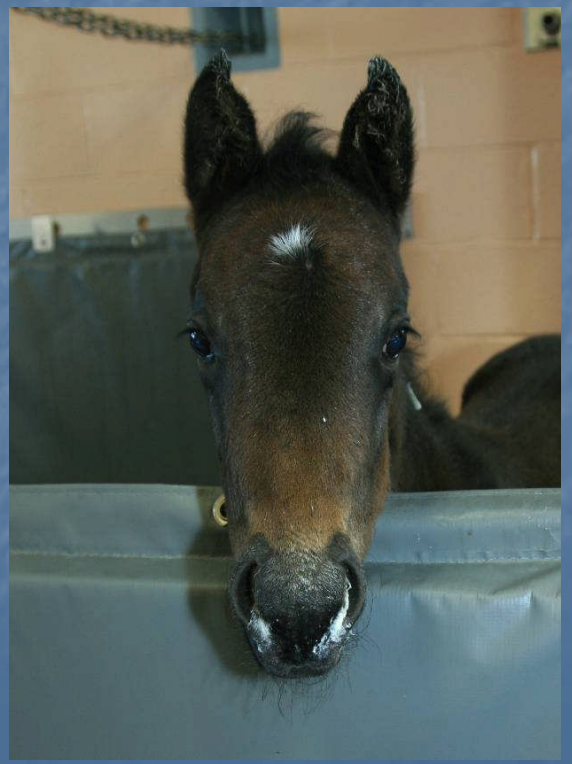
Lower Respiratory Diseases

- Aspiration pneumonia
- Traumatic chest injuries
- Infectious pneumonia
- ARDS



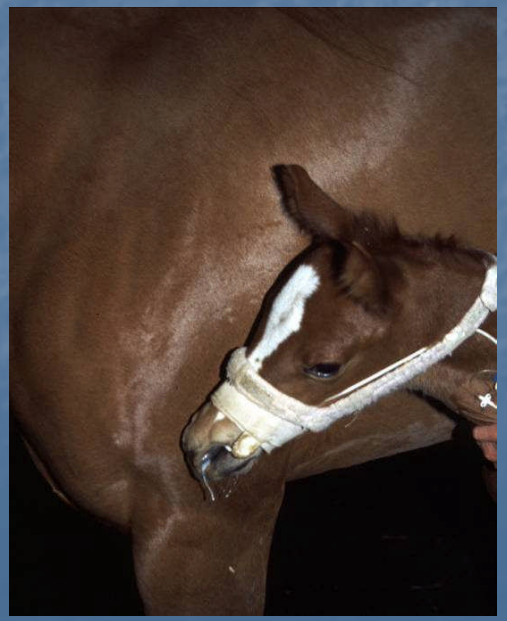
Aspiration Pneumonia Dysphagia

- Milk regurgitation from the nares
  - Cleft palate
    - Very rare cause of milk at nares
  - Dysphagia
  - Esophageal dysmotility



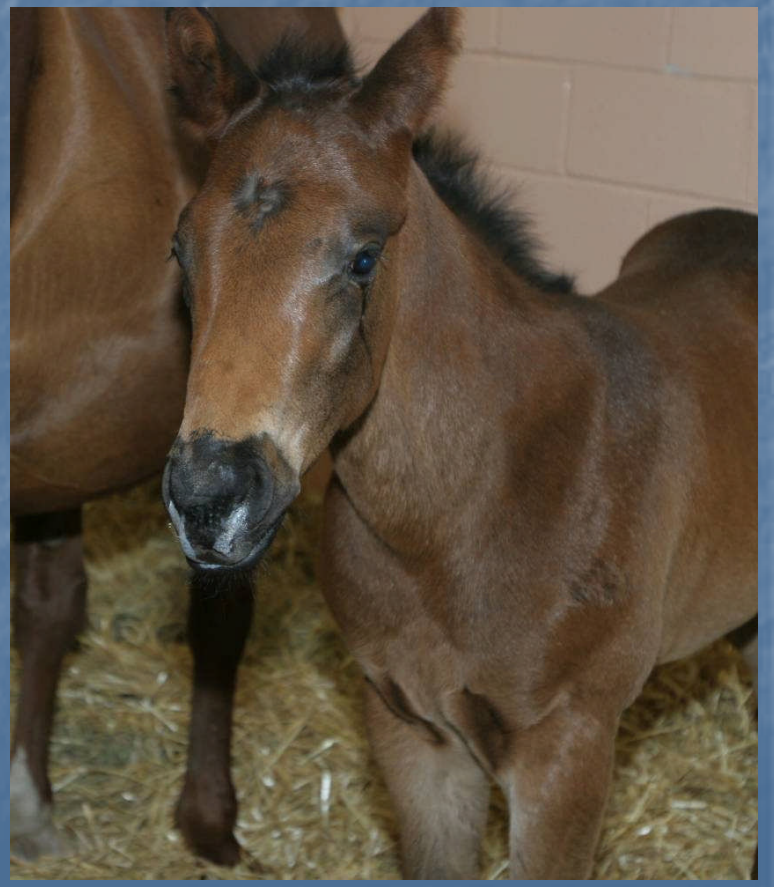
Dysphagia
No Aspiration

- Esophageal dysmotility
  - Failure to clear the cervical esophagus
  - Appear to nurse normally and effectively
  - Lower their head
    - Sneeze or shake head
    - Milk flow from one or both nostrils
    - Few drops to 60 ml
  - Delay of up to 5 minutes



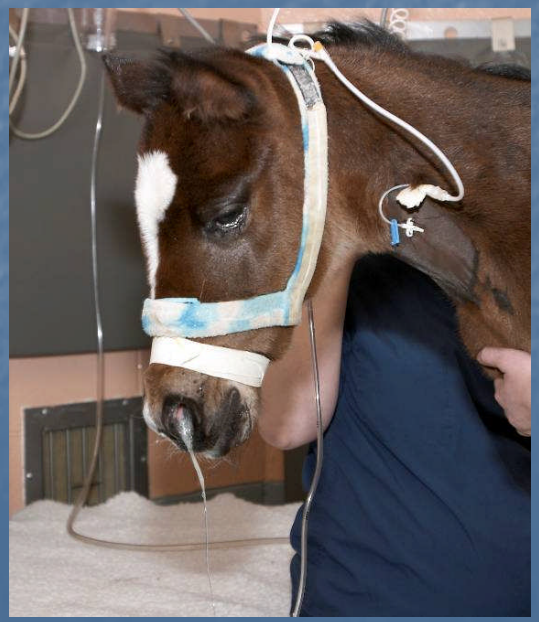
Dysphagia
No Aspiration

- Esophageal dysmotility
  - Transient problem
    - Once to several days
  - Etiology?
    - Neonatal Encephalopathy
    - Esophageal dysmotility
  - Aspiration rare
    - Swallowing normal
    - Guard airway
  - Most common reason
    - For milk coming from the nostril



Aspiration Pneumonia Dysphagia

- Congenital esophageal stricture
  - Megaesophagus
  - More danger of aspiration
- Dysphagia secondary to pharyngeal paresis
  - Degree of dysfunction variable
    - Upper airway obstruction
      - With or without dysphagia
    - Mild dysphagia – milk out nose
    - Severe dysphagia – milk aspirated
    - Most severe - aspirate saliva
  - Duration variable days to months
  - Therapy – feeding management

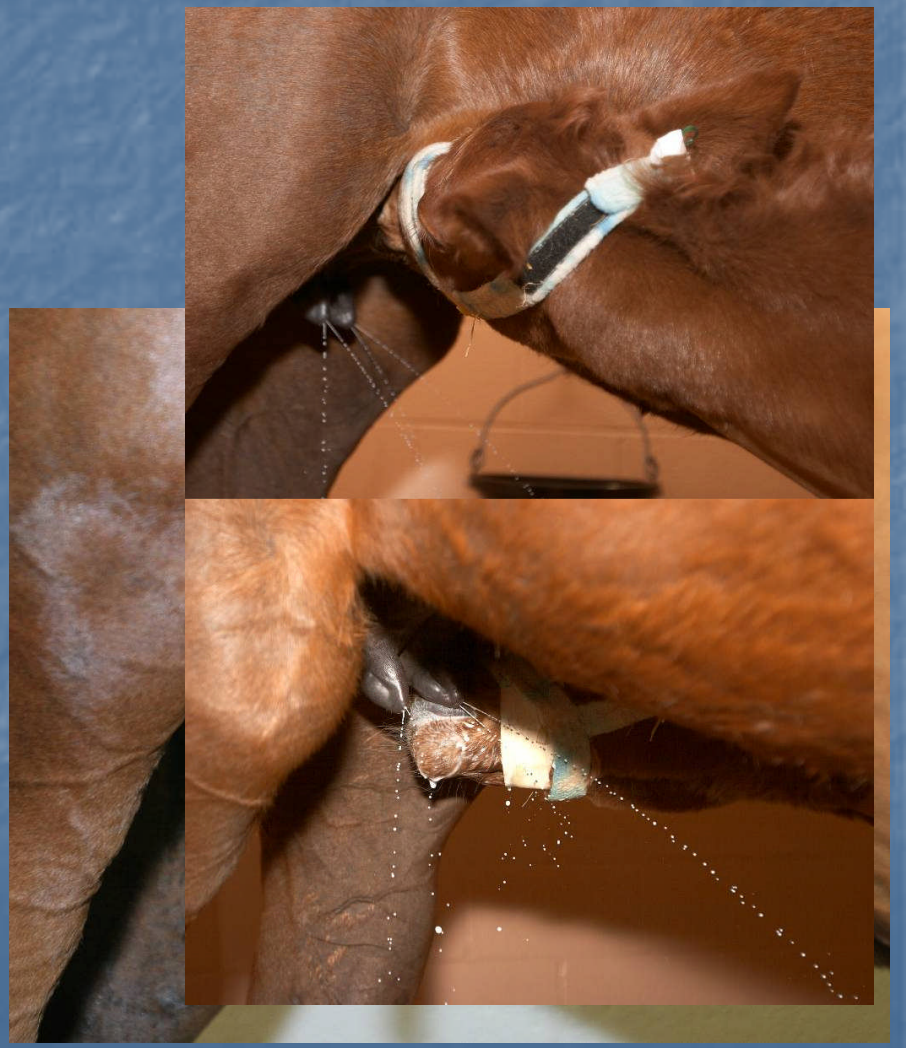


Aspiration Pneumonia Dysphagia

- Other reasons - older foal
  - Botulism
  - Choke
  - Primary oral candidiasis
  - Strangles

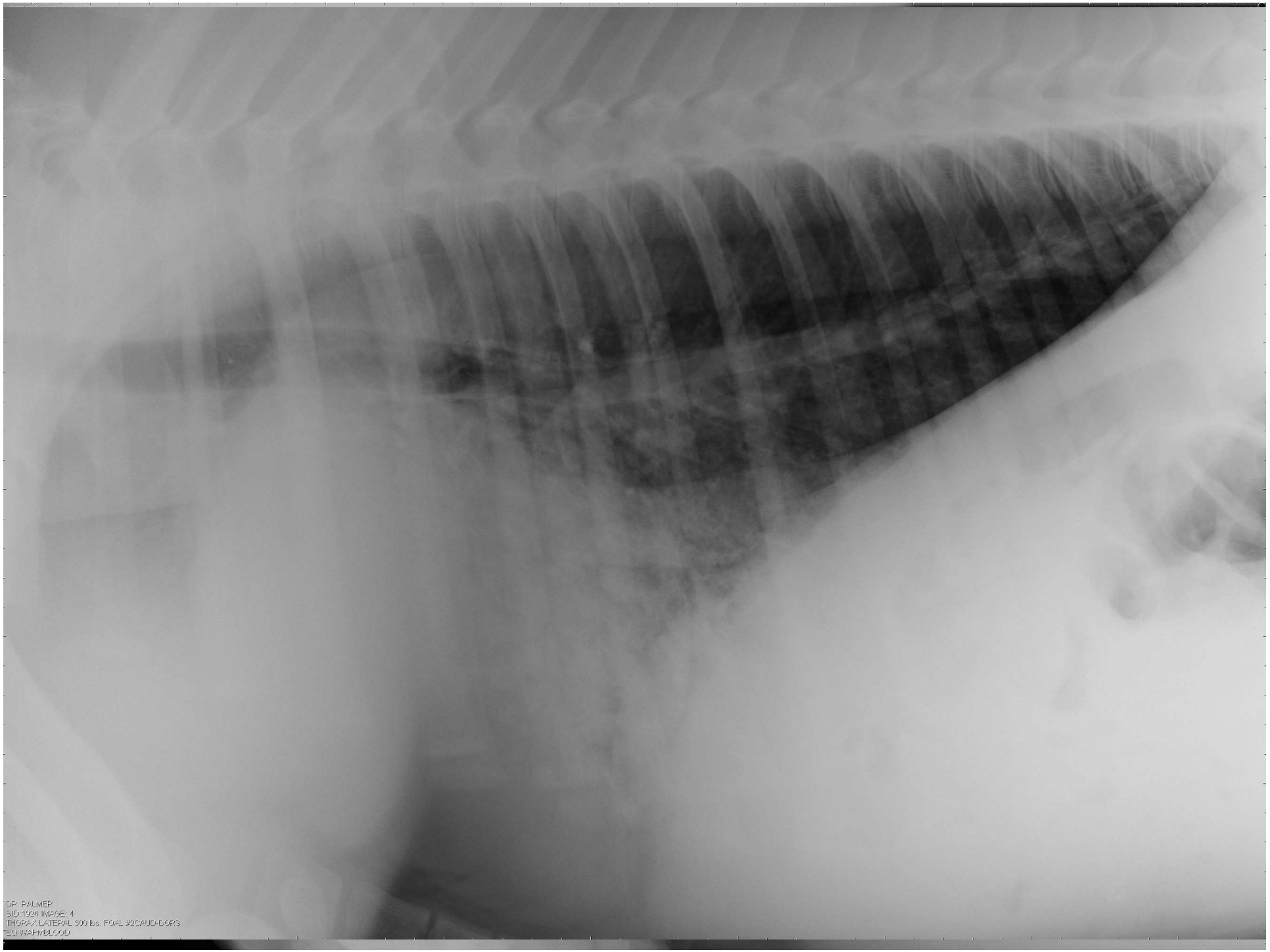
Aspiration Pneumonia
Normal Pharyngeal Function

- Weak foals
  - Neonatal Encephalopathy
    - Poorly coordinated swallowing
  - Prematurity
  - Fatigue
  - Heavily producing mares
- Musculoskeletal problems
  - Contracture
  - Laxity
- Fractured ribs
- Tachypnea
- Bottle feeding
  - Weak foals
  - Inexperienced caregivers



Aspiration Pneumonia

- May or may not be symptomatic
- Lung changes caudal heart base
  - Except lateral recumbent foals
- Signs
  - Respiratory effort and rate are increased
  - Pneumonic sounds
    - Referred upper airway sounds
  - Apneustic breathing pattern
  - Radiographs or ultrasound examination
  - Hematology and blood fibrinogen
- Mixed bacterial flora expected
- Prognosis
  - Most important factor - stopping aspiration


DR. PALMER
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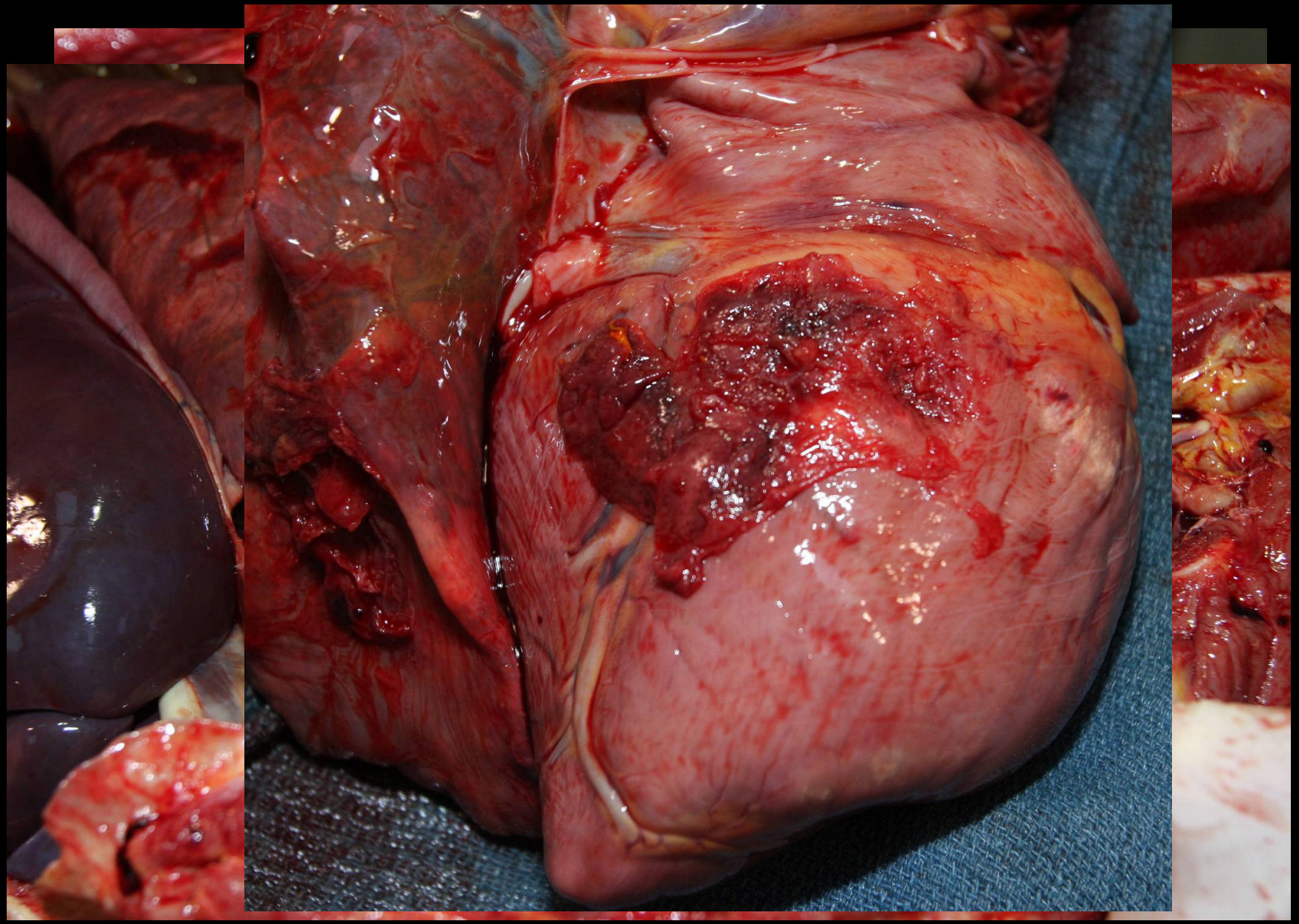
THORAX LATERAL 300 lbs. FOAL #2CAUDDORS
EQ: WARMBLOOD

Meconium aspiration

- Rare
  - Before birth
    - Associated with asphyxia
    - Fetal gasping
  - During delivery
    - Liquid meconium - upper airways
- Diagnosis
  - Stained nasal discharge
  - Radiographic changes
- Signs
  - Persistent tachypnea
  - Inflammatory hemogram
  - No bacterial infection
    - Persist up to a week or longer
    - Tachypnea and hyperfibrinogenemia
    - No radiographic changes
  - Secondary bacterial infections

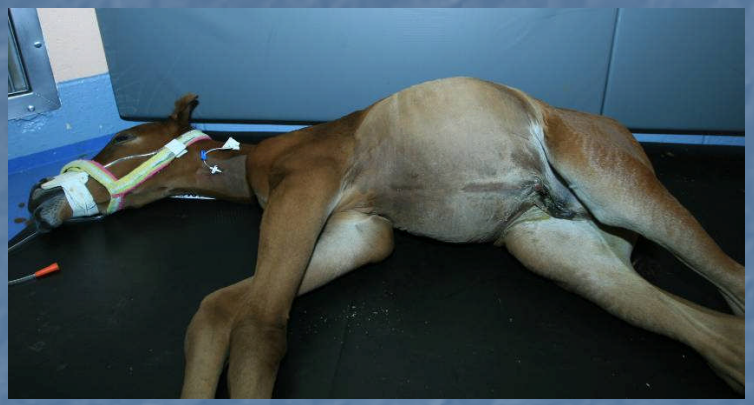
Secondary Pulmonary Disease
Traumatic Pulmonary Disease

- Fractured ribs
  - Pulmonary contusions
  - Pulmonary/Plural hemorrhage
  - Lacerations of major arteries
  - Pneumothorax
  - Traumatic diaphragmatic hernia
- Pleuritis and pleural effusion



Secondary Pulmonary Disease Abdominal Hypertension

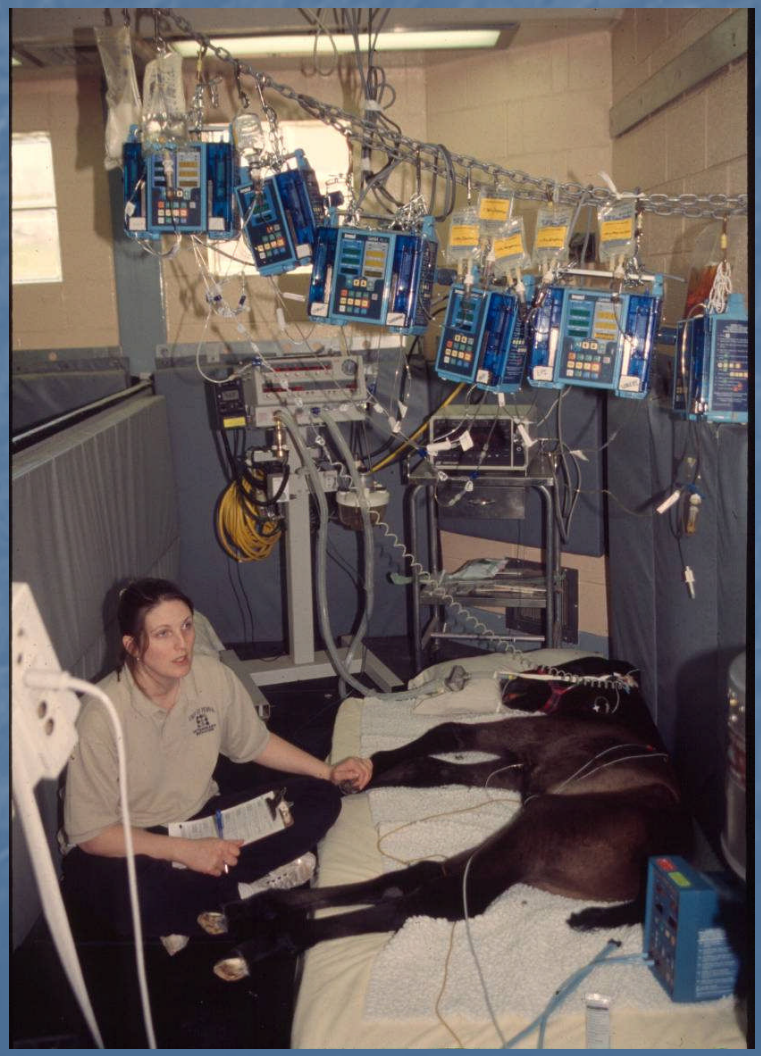
- Abdominal hypertension
  - Ruptured bladder
  - Intestinal distension
    - Acute enteritis
    - Ileus



- Decreased pulmonary blood flow
- Increased atelectasis
- Decreased compliance
- Increased mismatching/shunt fraction

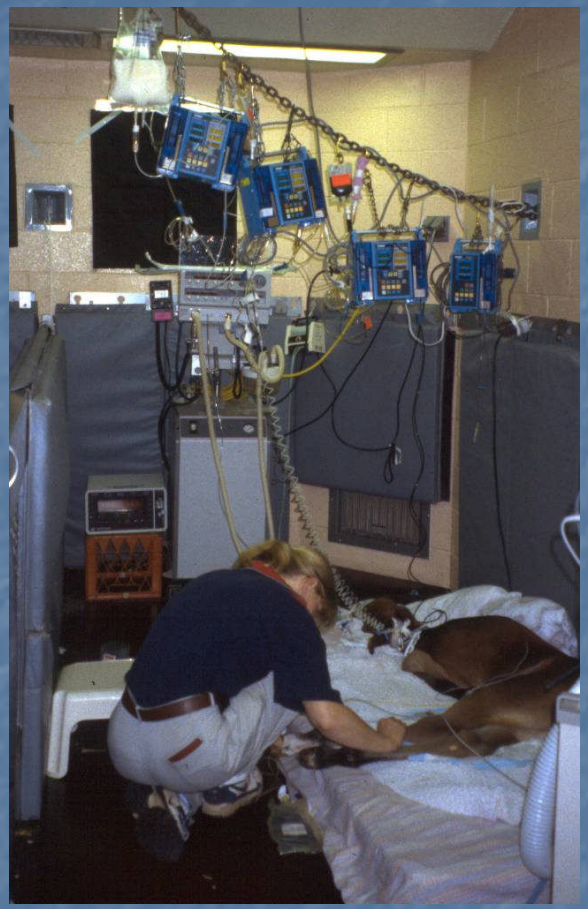
Sepsis

- Septicemia
  - Systemic localize in lungs
  - Primary pulmonary infection
- Bacterial
  - Hematogenous colonization
  - Aspiration
- Viral pathogens
  - Herpes Virus
  - Equine Viral Arteritis virus
  - Equine Influenza virus



ARDS

- Absence of pathogens
- Inflammatory response
  - Profound disruption of the lungs
  - Inflammatory mediators
    - Inducers of pulmonary hypertension
    - Significant right-to-left shunting
    - Face of systemic hypotension
- Acute lung injury (ALI)
- Acute Respiratory Distress Syndrome (ARDS)



Supportive Respiratory Therapy

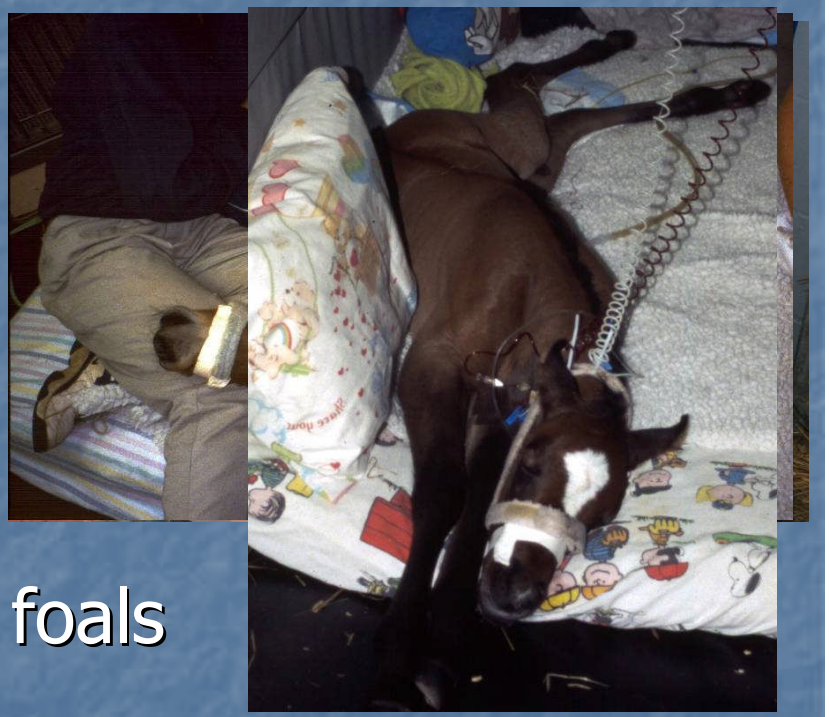
- Respiratory failure
  - Supportive therapies
  - Helpful
  - Harmful

Supportive Respiratory Therapy

- Supportive Therapy
  - Positional therapy
  - Intranasal oxygen insufflation
  - Increasing cardiac output
  - Inhaled nitric oxide (NO)
  - Supportive care
  - Stenting the airway
  - Respiratory stimulants
  - Positive pressure ventilation

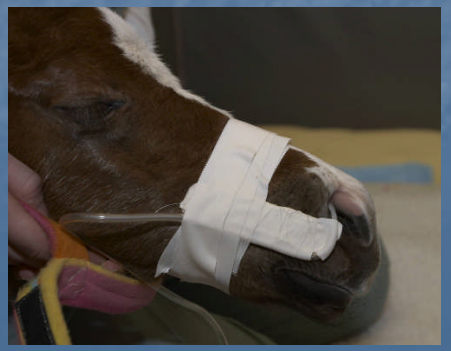
Hypoxemia Positional Therapy

- Help with V/Q matching
  - Aid oxygen loading
- Helpful if
  - Weak
  - Poor inspiratory excursions
  - Marginal perfusion
- Difference is not seen in all foals
  - Fighting the position
  - Arterial blood gas samples "worst case scenario"



Hypoxemia
Intranasal Oxygen Insufflation

- Oxygen is
  - Most useful/ Most dangerous drug
- INO_2 will correct mismatching
- Not be universally applied
  - Based on careful monitoring
  - Stall side blood gas analyzers
  - More realistic goal
- Complications
  - Oxygen toxicity
  - Nasal irritation
  - Rhinitis
  - Airway drying
    - Tracheal and nasal discharge
    - Increased upper airway resistance

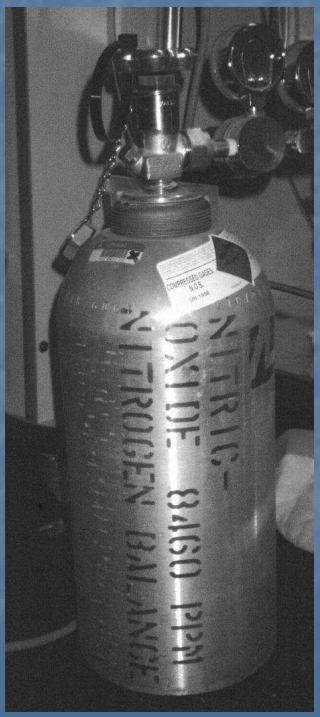


Hypoxemia Increasing Cardiac Output

- Remain hypoxemic despite INO_2
- Alveolar dead space ventilation
- Shunt fraction
  - Pulmonary hypertension
  - Increasing CO - decrease shunt Fraction?
- Dobutamine
  - Euovolemic hypoxemic
  - Dramatic improvement in oxygenation

Hypoxemia
Inhaled Nitric Oxide (NO)

- Pulmonary hypertension
  - 5-10 ppm NO
- Uneven ventilation and perfusion
  - Vasodilatation to ventilated alveoli
- Clinical improvement
  - Septic shock
  - ARDS
  - Transient – reverse early pathology

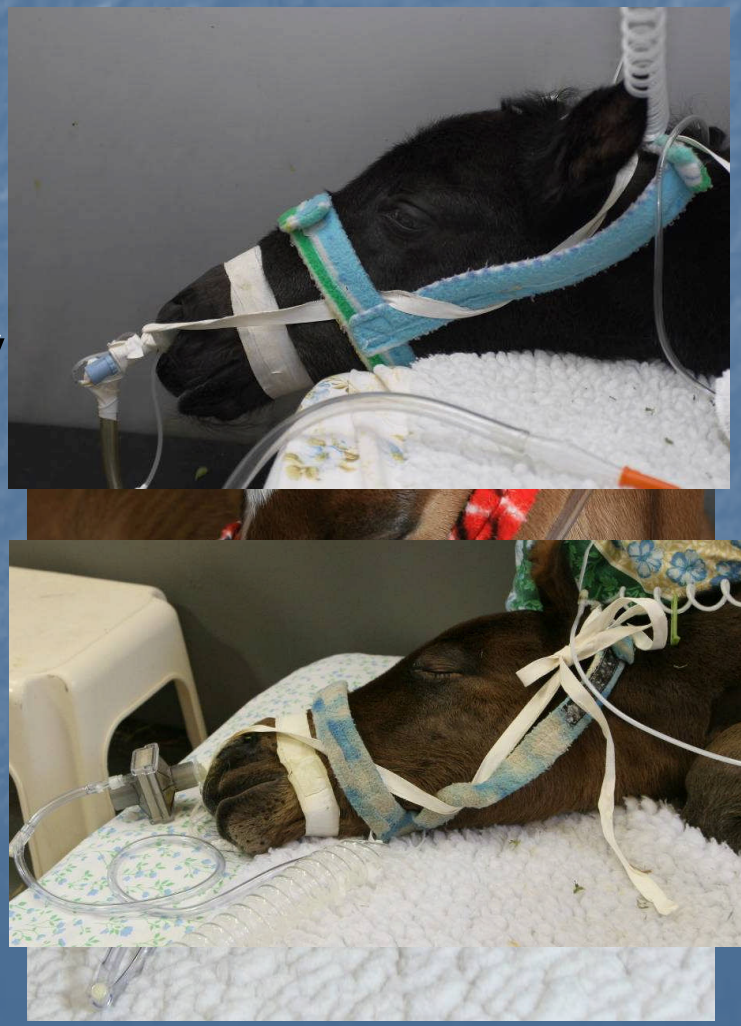


Hypoventilation

- Achieve a normal blood pH
  - Not "normal Paco2"
  - Appropriate hypoventilation
- Permissive hypercapnia
- Therapeutic hypercapnia
- Therapeutic hypoxemia

Respiratory Acidosis

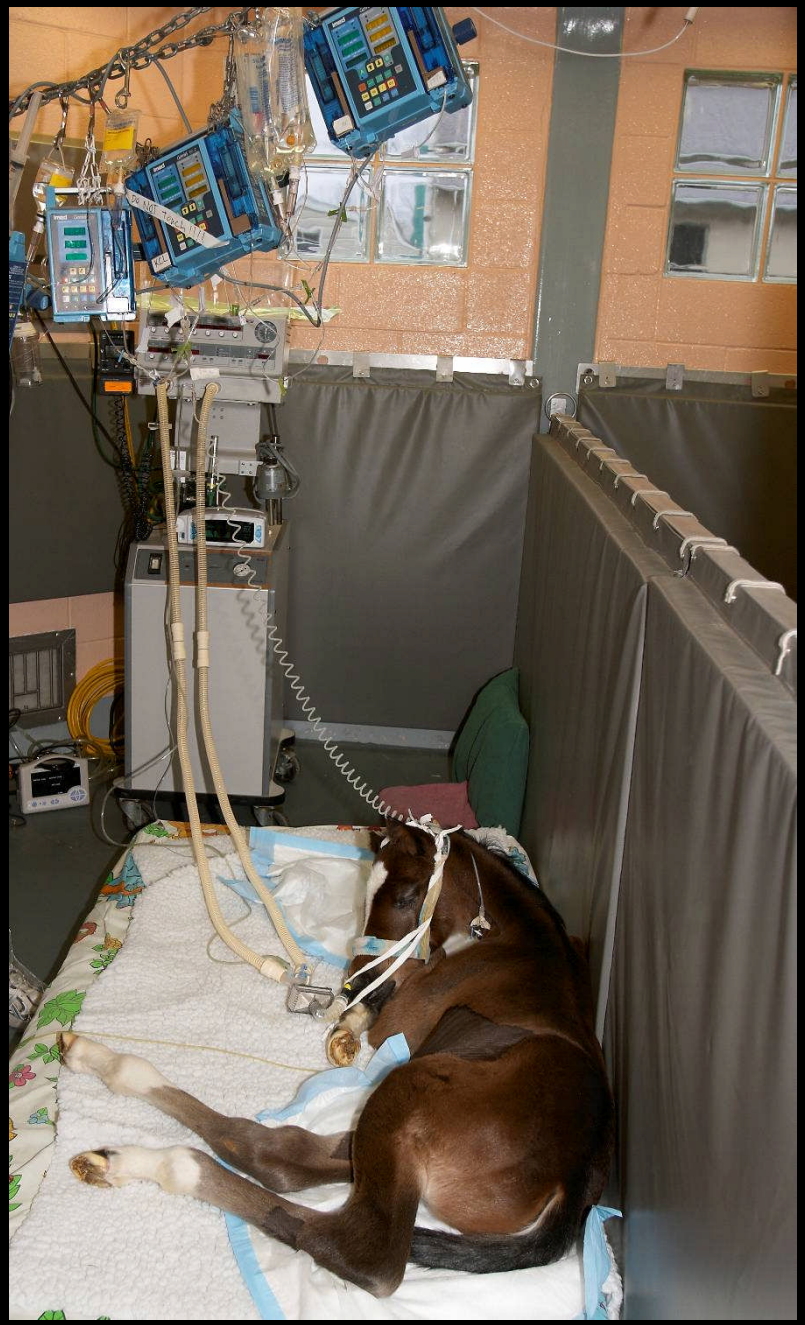
- Upper airway collapse
  - Endotracheal tube stent
- Neonatal Encephalopathy
  - Blunted central sensitivity
  - Chemical stimulants
    - Caffeine
    - Doxapram

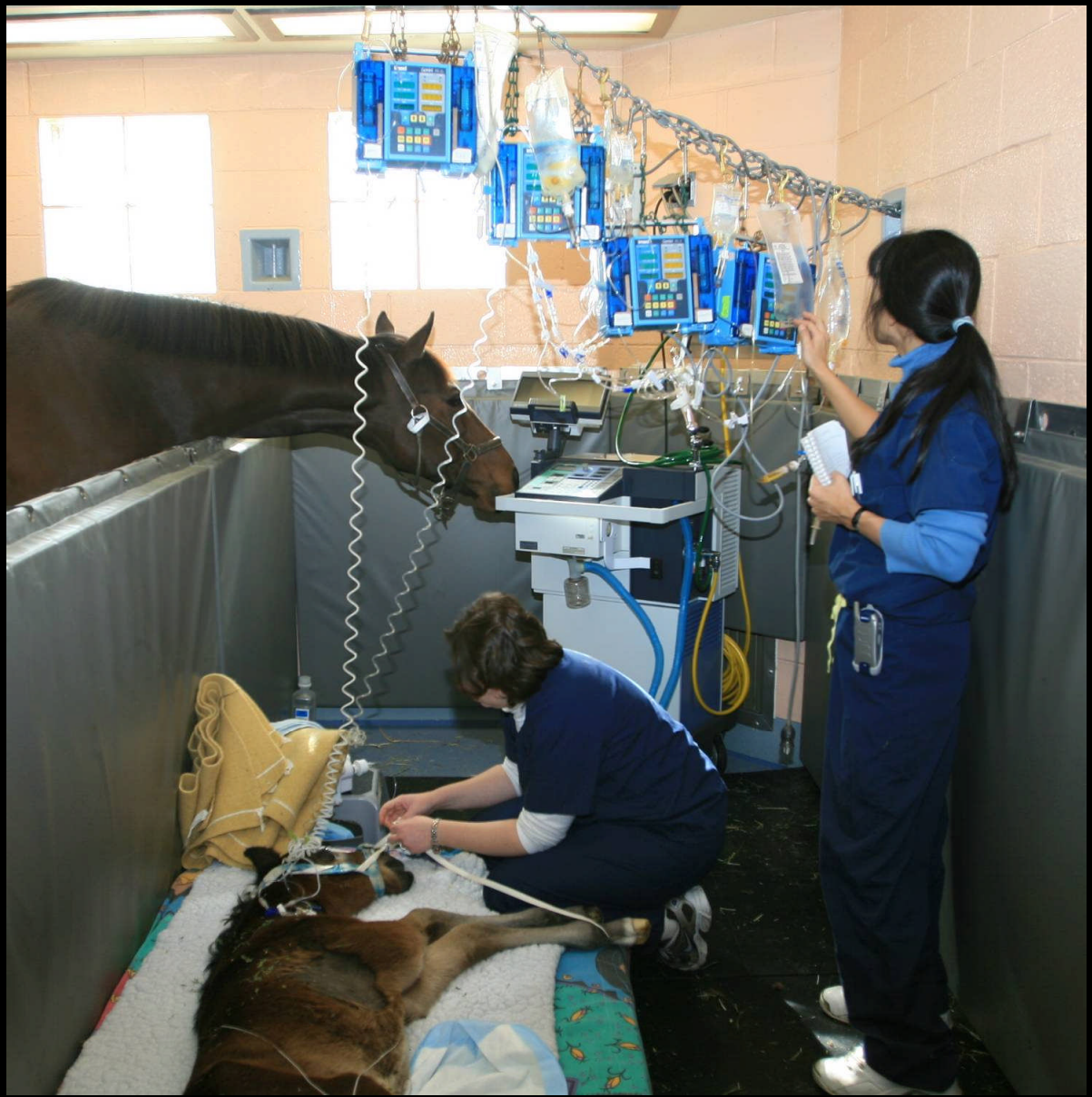


Positive Pressure Ventilation

- Manipulation of pulmonary gas exchange
- Increase lung volume returning normal FRC
- Decrease the work of breathing
  - Relieve fatigue
  - Decrease respiratory oxygen and energy utilization
  - Redirect perfusion away from respiratory muscles
- Modern ventilators
  - Normal lungs – easily ventilated
  - Severe pulmonary damage – possible to be successful
    - Septic pneumonia
    - ARDS


LASB





Respiratory Problems of the Neonate
Part 1

J. E. Palmer, VMD, DACVIM
Chief, Neonatal Intensive Care Service
New Bolton Center, University of Pennsylvania
Kennett Square, Pennsylvania, USA.

During the neonatal period foals may have an array of respiratory problems including problems adapting to neonatal cardiopulmonary physiology, problems with pulmonary gas exchange, problems with respiratory control, upper airway abnormalities and lower airway disease. This will be a brief review of the clinical aspects of some of these problems.

Ventilation/Perfusion Abnormalities

At birth the transition from fetal to neonatal cardiopulmonary physiology depends on a relative decrease in pulmonary vascular resistance and concurrent increase in systemic pressures. Although this transition often occurs rapidly within minutes of birth, it may be delayed for hours. The neonate may cope with this delay and show few signs beyond tachypnea until the transition occurs (usually by 12 hours). However if the transition is delayed and there are added stresses on the neonate or if there is a reversion to fetal

physiology after the initial transition the ability to cope may not be sufficient. The full decrease in pulmonary resistance takes several weeks and the ductus arteriosus and foramen ovale remain capable of being patent during this period. The balance of the right to left pressures is such that even small increases in pulmonary resistance or decreases in systemic pressure may increase the right to left shunt fraction resulting in significant hypoxemia. Circulating inflammatory mediators or hypoxia itself can result in an increase in pulmonary vascular resistance and there are many situations where systemic blood pressure may fall although most will affect both pulmonary and systemic circulation simultaneously. The resulting right to left shunt will result in hypoxemia despite oxygen therapy and if the shunt fraction is extremely large, hypercapnia. It can be difficult clinically (without careful ultrasound examination) to distinguish persistent pulmonary hypertension of the neonate from anatomic cardiac defects which result in right to left shunts. However persistent pulmonary hypertension can reverse spontaneously and may respond to oxygen therapy, nitric oxide or sildenafil.

There are other more common reasons for poor gas exchange in weak, critically ill neonates including mismatching, hypoventilation and progressive atelectasis. Mismatching caused by

uneven ventilation and perfusion is one of the most common gas exchange abnormalities in the ill neonatal foal. Uneven perfusion may be caused by poor cardiac output in combination with what appears to be poor vascular reactivity to oxygen causing misdirection of perfusion to under ventilated alveoli. Uneven ventilation may be caused by positional issues, weakness or onset of fatigue. Mismatching is easily corrected by intranasal oxygen insufflation which by adding oxygen to those alveoli that are over perfused will correct oxygen loading of the blood.

Weak foals will often suffer from hypoventilation as they fatigue. These foals may not be able to maintain the normal FRC and some alveoli may collapse during expiration and must be opened on each breath to receive ventilation. Alveoli that repeatedly close in this manner will tend to loose surfactant. As the amount of surfactant decreases it becomes more difficult to open these alveoli on inspiration and eventually they can no longer be opened and atelectasis results. This further decreases the compliance of the lungs and further tends to cause collapse of more alveoli. The sum affect of this is progressive atelectasis. Even in those alveoli which are being ventilated, the ventilation is less evenly distributed because alveoli not already open will not open until midway through inspiration. Other alveoli that are

already opened will accept gas throughout inspiration. This results in maldistribution of ventilation and perfusion.

The added work of opening alveoli during progressive atelectasis and the decreased compliance may result in fatigue of the respiratory muscles in the foal. Eventually the intercostal muscles will become so fatigued that they will no longer be able to hold the chest open during inspiration. As the diaphragm contracts producing a negative pressure in the thorax, the chest wall will tend to be pulled towards the lungs resulting in very inefficient ventilation. When these foals are observed, the chest wall will be seen to drop during inspiration as the abdomen expands secondary to the contraction of the diaphragm. This results in "wave chest" in which the thorax moves inward as the abdomen moves outward during inspiration. The abdomen moves inward as the chest moves outward during expiration. The development of "wave chest" heralds the onset of significant fatigue and respiratory failure which may lead to respiratory and cardiac arrest if not corrected.

Problems with Central Respiratory Control

There are a number of respiratory consequences of neonatal encephalopathy. Some of the most interesting involve changes in respiratory patterns. The most common patterns include apneustic breathing, cluster breathing with periodic respiratory pauses or apnea, apnea alone and ataxic breathing. Another common problem associated with Neonatal Encephalopathy is abnormal central control resulting in inappropriate hypoventilation or hyperventilation. Care needs to be made in making a diagnosis of central hypoventilation as neonates often have appropriate hypercapnia. Normally neonatal respiratory centers lack sensitivity allowing $PaCO_2$ to increase above adult levels. This is generally balanced by a concurrent metabolic alkalosis which also commonly occurs in neonates. The result is a normal pH despite a $PaCO_2$ in the 6.7 – 8.7 kPa (50 – 65 mmHg) range. This is appropriate hypercapnia which I don't believe has any physiologic significance except maintaining a normal blood pH. True central hypoventilation will result in a respiratory acidosis. All other reasons for hypoventilation such as an upper airway obstruction or weakness must be ruled out before a diagnosis on central hypoventilation can be made. Central hyperventilation can also occur during the excitatory phase of Neonatal Encephalopathy. Again there will be an inappropriate hypocapnia with an

alkalosis (often mixed metabolic and respiratory alkalosis). All other possible causes of hyperventilation need be ruled out such as pain or hypoxia. Central hyperventilation is often accompanied by hyperthermia with body temperatures as high as 42 C. Hyperthermia can sometimes be distinguished from fever as the later often has a diurnal variability whereas the former tends to be more consistent without the variability.

Upper Airway Abnormalities

Neonatal foals can have significant upper airway problems. By far the most common is an upper airway obstruction caused by hypotonic pharyngeal collapse secondary to transient pharyngeal paresis. The etiology of this problem is unknown but appears to be a peripheral neuropathy associated with Neonatal Encephalopathy. These foals often have dysphagia as well as pharyngeal collapse. It is not unusual for these foals to appear normal for the first few days of life. But when stressed and their respiratory rate and effort increase resulting in negative pressure in the pharynx, this in turn causes pharyngeal collapse and obstruction. The obstruction is self perpetuating since its presence will result in increased respiratory effort resulting in greater negative pressure in the pharynx causing more collapse. The obstruction

can cause respiratory failure followed by cardiovascular failure. The foal may die or as the foal looses consciousness, and the obstruction is relieved, the foal may recover. We frequently see critically ill foals with pharyngeal paresis. These foals may have generalized hypotonia and fatigue and are frequently recumbent. Phenobarbital may exacerbate pharyngeal collapse. In fact, much of the hypoventilation we see associated with phenobarbital therapy is secondary to pharyngeal collapse. Many of these foals benefit from placement of a nasotracheal tube as an airway stent. Also some foals with severe HYPP may have pharyngeal paresis.

Other less common causes of upper airway obstruction in neonatal foals include bilateral or unilateral choanal atresia (usually membranous), choanal hypoplasia, epiglottic cysts/malformations and distortions caused by wry nose and other facial deformities. These rare problems are easily diagnosed but difficult to correct.

Respiratory Problems of the Neonate
Part 2

J. E. Palmer, VMD, DACVIM
Chief, Neonatal Intensive Care Service
New Bolton Center, University of Pennsylvania
Kennett Square, Pennsylvania, USA.

During the neonatal period foals may have an array of respiratory problems but the most serious are lower respiratory diseases. Aspiration pneumonia, traumatic chest injuries, infectious pneumonia and ARDS all commonly occur in neonatal foals and may require intensive care. There are a variety of supportive care techniques which are commonly utilized when treated critically ill neonatal foals with respiratory problems. This will be a brief review of the clinical aspects of some of these problems and techniques used for therapeutic intervention.

Lower Respiratory Disease

Pulmonary disease in neonatal foals is more serious than upper respiratory problems. One of the most common pulmonary diseases of the neonate is aspiration pneumonia secondary to dysphagia. Even though many veterinarians think of cleft palate when they see foals with

milk coming of their nose, cleft palate is rare and accounts for less that 1% of the problems in such foals. The most common reason for milk regurgitation from the nares is not dysphagia at all but the failure to clear the cervical esophagus of milk after drinking. Foal's with this problem will appear to nurse normally and effectively but after they stop, and as they lower their head from the typical nursing posture they may sneeze or shake their head and if they lower their head a little more an amount of milk ranging from a few drops to 60 ml or more will flow from one or both nostrils. Sometimes there is a delay of up to 5 minutes between nursing and milk dripping from the nose. In these cases the milk is swallowed normally but is retained in the cervical esophagus and will run out the nose. This may be a very transient problem occurring only once or twice during initial nursing episodes the first day of life or may persist for several days. This problem is associated with Neonatal Encephalopathy or is a form of esophageal dysmotility. Although large quantities of milk may run freely out of the nose, aspiration from this syndrome is rare as swallowing and the ability to guard the airway are normal. The milk simply follows gravity running back out of the esophagus, through the nasopharynx and out the nasal passages. The rare congenital condition of esophageal stricture with secondary megaesophagus can cause similar signs, but with more danger of aspiration because of the larger quantity of milk involved. But the most common reason for milk

coming from the nostril after nursing resulting in aspiration is pharyngeal paresis. The degree of dysfunction is variable and can cause upper airway obstruction without dysphagia as noted above or may cause variable degrees of dysphagia. Severely affected cases will not only be unable to prevent milk from entering the nasopharynx while swallowing, they will also be unable to guard their tracheal and milk will freely be aspirated. The most severely affected foals will even aspirate their own saliva. In the older foal (1 - 8 weeks) botulism, primary choke, primary oral candidiasis or strangles may cause similar signs and aspiration.

Aspiration pneumonia can also occur in the face of normal pharyngeal function. Weak foals, foals with poorly coordinated swallowing secondary to Neonatal Encephalopathy, foals tachypneic and having a hard time standing because of contracture, laxity, fractured ribs or other musculoskeletal problems or foal tachypneic for other reasons are at high risk of aspiration. Also, weak foals being bottle fed by inexperienced caregivers or nursing heavily producing mares are also at great risk.

Aspiration pneumonia may or may not be symptomatic depending on how widespread the lesion and the chronicity. The lesion is always centered in the lung overlying the caudal heart

base except in the cases where aspiration occurs when the foal is in lateral recumbency. Usually the resting respiratory effort and rate are increased and there are pneumonic sounds ausculted over the heart base although the pneumonic sounds may be more widespread. Moist sounds are often heard over the trachea and at the nares and a moist cough is frequently present. Referred upper airway sounds are frequently ausculted over the lung fields making accurate assessment of the lungs difficult. Some cases with mild disease will not have an increased respiratory rate or effort but instead will show an apneustic breathing pattern. The extent of pneumonia can be assessed with either chest radiographs or ultrasound examination. Progress of the inflammatory reaction can be followed by hematology and blood fibrinogen levels. There is usually a mixed flora of pathogens involved in the pneumonia so wide spectrum antimicrobials are required. The most important factor in recovery is stopping the ongoing aspiration.

Meconium aspiration is a special case of aspiration pneumonia. Meconium aspiration is an unusual problem which occurs most often associated with fetal diarrhea and passage of liquid meconium before birth. The aspiration may occur before birth associated with asphyxia and fetal gasping or after delivery with liquid meconium in the upper airways. Diagnosis is made

by finding meconium stained nasal discharge and radiographic changes. Foals may continue to drain meconium-stained nasal fluid for several days. They usually have tachypnea, an inflammatory hemogram and may have secondary bacterial infections. Tachypnea and hyperfibrinogenemia may persist up to a week or longer despite absence of radiographic changes or signs of bacterial infection.

Traumatic pulmonary disease is not rare in neonatal foals because of the frequency of occurrence of fractured ribs. Fractured ribs can cause pulmonary contusions, pulmonary/plural hemorrhage, lacerations of major arteries, pneumothorax and traumatic diaphragmatic hernia. The most common cause of pleuritis and pleural effusion in neonatal foals is traumatic injury secondary to fractured ribs. Another cause of secondary pulmonary disease in the foal is abdominal hypertension secondary to such problems as ruptured bladder or intestinal distension with fluid or gas in acute enteritis. In such cases, the increased abdominal size results in decreased pulmonary blood flow, increased atelectasis, decreased compliance and increased mismatching and shunt fraction compromising pulmonary function.

The most serious pulmonary diseases of the neonatal foals are those associated with sepsis. Neonatal foals with septicemia may have localization of the infection in the lungs. Bacterial septicemia may result in hematogenous colonization of the lungs progressing to diffuse bacterial pneumonia leading to respiratory dysfunction and finally respiratory failure. Several viral pathogens including herpes virus, equine viral arteritis virus and equine influenza virus target the fetal and neonatal lungs leading to pulmonary failure. Even in the absence of pathogens, the inflammatory response may result in profound disruption of the lungs. Inflammatory mediators are strong inducers of pulmonary hypertension. Even modest increases of pulmonary resistance may result in significant right-to-left shunting though the foramen ovale and ductus arteriosus in the face of systemic hypotension. The inflammatory response in the lungs will lead to acute lung injury (ALI) which may progress to Acute Respiratory Distress Syndrome (ARDS).

Supportive Respiratory Therapy

When respiratory problems in neonates lead to respiratory failure, there are a number of supportive therapies which when employed can be very helpful. However all of these

therapeutic manipulations have negative as well as positive aspects so the clinician needs to choose therapeutic options with care.

Positional therapy may help with V/Q matching and thus aid oxygen loading. Recumbent foals maintained in a sternal position may have a Pa_{O2} 1.3 kPa (10 mmHg) and sometimes as much as 2.7 pKa (20 mmHg) higher than the Pao_2 of the same foal in lateral recumbency. But this difference is not seen in all foals, only those who are weak, have poor inspiratory excursions or marginal perfusion. Other foals will not benefit and in fact the effort the foal expends fighting the position may make forcing this position contraindicated. I usually draw arterial blood gas samples for the "worst case scenario" with the foal in lateral recumbency. If oxygenation is adequate, I place the foal in whatever position the foal seems most comfortable. I think the over zealous creed of some clinicians to keep all foals sternal at all times may be as often harmful as helpful.

Intranasal oxygen insufflation is the most commonly used treatment modality in hypoxemic foals. As mismatching is the most common reason for hypoxemia in neonatal foals it is often effective. The most useful drug in neonatal intensive care may be oxygen. The most

dangerous drug in neonatal intensive care may be oxygen. Because of this duality, oxygen therapy should not be universally applied, but based on careful monitoring. The advent of stall side blood gas analyzers has made this a more realistic goal. Complications of intranasal oxygen insufflation include oxygen toxicity, nasal irritation and rhinitis and airway drying resulting in excessive tracheal and nasal discharge causing increased upper airway resistance.

Foals who remain hypoxemic despite intranasal oxygen insufflation and positioning especially those with a high percent of alveolar dead space ventilation indicating lung hypoperfusion will benefit from increasing cardiac output. Ensuring adequate cardiac output is not only important in oxygen delivery to the tissues but also in providing pulmonary perfusion allowing for adequate ventilation perfusion (V/Q) matching necessary for effective gas transport in the lungs. In addition, in foals with right-to-left shunts secondary to pulmonary hypertension, increasing cardiac output appears to decrease the shunt fraction. In euovolemic hypoxemic foals, dobutamine therapy will often result in a dramatic improvement in oxygenation.

The advent of inhaled nitric oxide (NO) therapy has revolutionized the treatment of neonatal pulmonary hypertension, and appears to be effective in foals. Inhaled nitric oxide at rates as low as 5 to 10 ppm NO can dramatic reversal of pulmonary hypertension. NO is also very helpful in cases with uneven ventilation and perfusion as it will cause vasodilatation to those alveoli being ventilated. Foals with pulmonary failure secondary to septic shock and those with ARDS may, at least temporally, have a dramatic improvement in gas exchange when placed on NO. One problem with NO is that it can cause significant pulmonary toxicity through the production of free radicals in the presents of high oxygen concentrations, but at low concentrations this appears to be a rare complication.

There are several approaches which may help correct respiratory acidosis secondary to hypoventilation. The goal of treating hypoventilation is to achieve a normal blood pH (7.35 – 7.40) and not a "normal $Paco_2$." Many critically ill neonatal foals, especially those who are catabolic, have a significant metabolic alkalosis. In such cases hypoventilation which corrects the blood pH is appropriate and should not be treated. On the other hand, in foals with a metabolic acidosis, hyperventilation is appropriate. Only hypoventilation contributing to an acidosis should be treated.

Some fatigued, weak foals will have mild hypoventilation contributing to acidosis which will resolve with simple supportive care such as continuous rate infusion glucose, cardiovascular support, etc. Hypoventilation resulting in acidosis can occur secondary to upper airway collapse. This is often easily corrected by stenting the airway with an endotracheal tube. Hypoventilation contributing to acidosis can be secondary to blunted central CO_2 receptor sensitivity as often occurs with neonatal encephalopathy. This can be treated with the use of respiratory stimulants such as caffeine or doxapram. I prefer caffeine as it is the safest and most effective methylxanthine for use in foals with central respiratory center depression. Also it does not seem to correct hypoventilation which is appropriately correcting the pH unless high levels are used. Others prefer to use doxapram but it has been associated with metabolic acidosis.

The most definitive treatment of hypoventilation and failure of oxygenation is the use of mechanical ventilation. Positive pressure ventilation will support and allow manipulation of pulmonary gas exchange, increase lung volume returning normal FRC, and decrease the work of breathing allowing ventilatory muscles to rest when fatigued and decrease the oxygen and

energy utilization and perfusion that would be required to support the work of breathing. Foals tolerate mechanical ventilation well, not requiring sedation or other special restraint. Modern ventilators designed for human medicine work quite well in neonatal foals. In fact, these ventilators with their advanced modes make ventilating foals that have normal lungs quite simple and in addition make successful treatment of some foals with severe pulmonary damage possible. Some cases of septic pneumonia and ARDS can be treated successfully with the aid of these advanced medical devices.

Short summaries:

Respiratory Problems of the Neonate Part 1

During the neonatal period foals may have an array of respiratory problems including problems adapting to neonatal cardiopulmonary physiology, other problems in pulmonary gas exchange, problems with respiratory control, upper airway abnormalities and lower airway disease. Ventilation/perfusion abnormalities are common in this age range and include persistent fetal circulation or reversion to fetal circulation caused by development of pulmonary hypertension, mismatching, hypoventilation, increased alveolar dead space ventilation, hypoperfusion and progressive atelectasis. Neonatal encephalopathy, a common problem of neonatal foals is associated with a number of changes in central respiratory control and also a group of functional upper airway abnormalities. This will be a brief review of the clinical aspects of some of these problems.

Respiratory Problems of the Neonate Part 2

During the neonatal period foals may have an array of respiratory problems but the most serious are lower respiratory diseases. Aspiration pneumonia, traumatic injuries, infectious pneumonia and ARDS all commonly in neonatal foals and may require intensive care. There are a variety of respiratory supportive care techniques which are commonly utilized when treated critically ill neonatal foals. Oxygenation can be enhanced with positional therapy, intranasal oxygen insufflation, increasing cardiac output and inhaled nitric oxide therapy. Hypoventilation may be addressed by stenting the airway with an endotracheal tube in the case of upper airway obstruction, respiratory stimulants in the case of problems with central control and ultimately by positive pressure mechanical ventilation. This will be a brief review of the clinical aspects of some of these lower respiratory diseases and techniques used for therapeutic intervention.