Non-\textit{R. equi} Pneumonia

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\section{I. INTRODUCTION}

Respiratory disease in foals is one of the major causes for morbidity and mortality in the young horse. Early detection and treatment of respiratory problems is essential for not only the animal’s well-being but also for the animal’s athleticism and future performance. Disorders of the respiratory system are second in importance only to those of the musculoskeletal system in limiting the athletic performance of the horse.

The diagnostic approach to respiratory disorders include the following:

- History
- Age and Breed
- Environment
- Physical Exam
  - Auscultation of the Chest
  - Percussion of the Chest
- Endoscopy
- Ultrasonography
- Radiography

\subsection*{Age and Breed}

Knowing the age and breed of the foal will provide insight as to the problem. Congenital problems are usually noted at birth, while acquired diseases (i.e., bacterial pneumonia) tend to occur when the foal is older (> 2 weeks of age).

The breed of the animal is important in determining the cause of the respiratory disorder. For example, Arabian horses with chronic lung infections should be evaluated for combined immunodeficiency syndrome. Selective immunoglobulin M (IgM) deficiency tends to occur more frequently in Arabians and Quarter Horses, whereas agammaglobulinemia has been documented in Thoroughbreds and Standardbreds.

\section*{Auscultation of Chest}

Auscultation of the chest includes the use of either a rebreathing bag or covering the nares to cause hyperpnea. Bronchovesicular sounds in the young animal can be easily noted because of the decreased muscle mass and less attenuation of the lung sounds compared to the adult horse. Increased airflow will also increase the bronchovesicular sounds, especially in febrile, excited or hyperpneic animals. Auscultatory findings do not always correlate with the degree of alveolar ventilation. Foals with lung consolidation may still have normal bronchovesicular sounds because of the radiation of airflow from the adjacent areas. Therefore auscultation alone cannot be used to diagnose pathologic condition of the lower airway.

Crackles may be auscultated and represent the equalization of pressure between two compartments (alveoli and the bronchioles) after the airways have opened (i.e., mucus in the airway that does not allow air to flow into the alveoli but when a deep breath is taken the air movement flows passed the mucus and into the alveoli). Wheezes are musical sounds that arise from vibrations within the airway walls by turbulent airflow noted during constriction of the airways.

\subsection*{Thoracic Ultrasound (Figs. 1 and 2)}

Ultrasonographic examinations can be performed with a multifrequency 5.0- or 7.5-MHz linear transrectal transducer. The 7.5-MHz will be able to display a depth of 4 to 12 cm, which is ideal for the thoracic examination of a foal. Isopropyl alcohol is copiously applied to the hair coat to provide surface contact between the transducer and the foal. The alcohol helps to reduce the intervening trapped air. The thorax should be thoroughly scanned in a dorsal to ventral plane from the 16th to the 3rd intercostal space.

Sound waves are completely reflected at the normal aerated lung interface allowing only the pleural surface to be evaluated. Therefore, the normal visceral pleural edge of the lung appears as a straight hyperechoic line with characteristic equidistant reverberation air artifacts indicating normal aeration of the pulmonary periphery. The pleural edge of the lung is imaged gliding dorsally and ventrally when watching the patient breathe during thoracic ultrasonography. Only when fluid or cellular accumulation in the lung occurs immediately beneath the visceral pleural surface will an acoustic window be created.
allowing visualization of pulmonary pathology. The affected area of the lung is hypoechoic and/or lacks the normal air echo at the surface. It is critical to examine the lung carefully during exhalation and inhalation because lesions can move beneath an adjacent rib or inhaled air into the alveoli and surrounding airways will cause reflection of the sound waves thereby preventing the visualization of pulmonary disease.

Pulmonary consolidation is hypoechoic and/or lacks the normal air echo at the surface. Ultrasonographic visualization of consolidated lung occurs because of the replacement of alveolar air with fluid or cells producing an acoustic window.

**Radiography (Fig. 3)**

Radiography is considered by some to be the principal imaging technique for the evaluation of the thorax. However, radiographic examination has its limits (in the field and patient size) therefore ultrasonography is a valuable alternative.

In the immature foal, the cardiac silhouette will occupy a greater proportion of the thoracic cavity. Radiographs of the thorax in foals obtained within the first few hours of birth, usually have a generalized interstitial opacity due to incomplete inflation. Within 12 hours the lungs become more lucent as the foal becomes active and the lungs completely inflate. The generalized opacities noted during the first few hours of life make radiography in neonates a diagnostic challenge. Any questionable abnormalities should therefore be re-evaluated in 24-48 hours.

**Transtracheal Wash**

A transtracheal wash (TTW) is a technique used to collect fluid from the lower respiratory tract for bacterial culture and cytologic evaluation. The results of the wash can help determine a proper management of equine respiratory tract disease. Two techniques to obtain a TTW will be discussed: percutaneous and guarded endoscopic sampling.

**Blood Gas Analysis (Fig. 4)**

Arterial blood gas analysis (ABG) should be performed on any neonatal patient in respiratory compromise. Arterial samples can be obtained in neonates easily from the dorsal metatarsal, facial, brachial and femoral arteries. The site should be
Aseptically prepared and clipped to facilitate visualization and palpation of the artery. Either a topical anesthetic (lidocaine and prilocaine) or a small bleb of lidocaine placed subcutaneously may decrease motion in response to sample collection. The blood should be collected in a heparin coated syringe, evacuated of air bubbles and sealed with a rubber stopper. The sample should be analyzed within 10 minutes if at room temperature or 3 hours if placed on ice.

\( \text{PaO}_2 \) assesses the pulmonary oxygenating capability. Oxygen tension in arterial blood below 80 mmHg is associated with hypoxemia and arterial saturation of 95%. Values below 60 mmHg indicate severe hypoxemia and warrant oxygen therapy. Assessment of \( \text{PaO}_2 \) can vary from normal in the early stages of life and when collected in lateral recumbency. Clinically normal neonatal foals can have \( \text{Pao}_2 \) below 80 mm Hg in the first 12 to 24 hours of life. Foals immediately following parturition can have a \( \text{Pao}_2 \) between 40 and 50 mm Hg and if in lateral recumbency the \( \text{Pao}_2 \) can be 39.7 +/- 2.1 mm Hg.

\( \text{PaCO}_2 \) assesses the ventilatory status of the patient. Normal foals carbon dioxide tension in arterial blood ranges between 42 and 45 mmHg. \( \text{Paco}_2 \) > 60 mmHg is associated with hypoventilation and may warrant chemical stimulation of ventilation or mechanical ventilation.

**II. ACQUIRED DISEASES**

**Respiratory Distress Syndrome (Fig. 3)**

Respiratory distress syndrome is a poorly understood syndrome which results in a failure of normal gas exchange at the alveolar level. This failure of proper gas exchange results in atelectasis (no pulmonary expansion). Pulmonary immaturity and surfactant deficiency are the main causes of neonatal respiratory distress syndrome in humans.

Surfactant dysfunction is primarily associated with immaturity of the lungs and is therefore commonly associated with premature foals. Full-term foals could also be affected especially those with systemic disease, meconium aspiration as well as viral and bacterial pneumonia which can result in altered or inappropriate surfactant production.

Persistent fetal circulation and cardiac congenital anomalies are examples of non-respiratory causes of respiratory distress syndromes seen in the newborn foal.

**Clinical Signs**

Animals present with flared nostrils, tachypnea, dyspnea, marked abdominal lift with inspiration. Mucus membranes are injected and, if severe, cyanotic. Before an animal goes into respiratory failure a paradoxical respiratory pattern will be noted. Paradoxical respiratory pattern is characterized when there is a downward motion of the diaphragm and consequent outward movement of the flanks without excursion of the rib cage during inspiration. This pattern indicates complete fatigue of the respiratory muscles.

**Diagnosis**

Arterial blood gas analysis is an essential diagnostic when evaluating animals with respiratory distress syndrome. Hypoxemia (i.e. \( \text{PaO}_2 < 80 \) mmHg) is a characteristic finding with this disease. \( \text{PaCO}_2 \) will be variable because carbon dioxide diffuses more readily than oxygen.

Thoracic radiography is also important in the evaluation of these foals. Thoracic evaluation reveals a hazy, granular appearance associated with interstitial or alveolar patterns. Prominent air bronchograms can also be noted.

**Treatment**

Therapy is aimed at preventing atelectasis, correcting hypoxemia and preventing hypercapnia. The foal should be placed into sternal recumbency as this will allow better chest-wall expansion and respiration. It can help increase partial pressure of oxygen (\( \text{PaO}_2 \)) by 10 to 20 mm Hg. Nasal oxygenation at a rate of 6 to 15 L/min can help increase the fraction of inspired air. If oxygenation insufflation alone is unlikely to elevate the \( \text{PaO}_2 \) then assisted ventilation will be necessary. Vasorelaxants and bronchodilators may also be

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Fig. 3. Blood gas collection from the metatarsal artery in a neonate. A small diaper dampened with water and placed in a microwave for 20 seconds was placed over the artery for 30 seconds before collection. The warm compress allowed for dilation and easy visualization of the artery.
administered in conjunction with oxygen therapy to improve pulmonary function.

- Sildenafil 0.5 to 2.5 mg/kg PO SID
- Aminophylline 5-10 mg/kg IV QID (diluted in fluids)
- Corticosteroids soludeltacortef 1 mg/kg IV SID/BID or dexamethasone 0.1 mg/kg IV SID to BID
- Surfactant replacement therapy: should be given within 30 minutes of delivery for optimal success. The total dose is divided into four equal amounts and administered via the endotracheal tube with a long catheter. Each fourth should be administered with the foal placed in different positions (i.e. left lateral, dorsal, right lateral and ventral recumbency). The foal should be manually ventilated during and after surfactant administration. Typical dosage is 100 mg/kg.

Meconium Aspiration

In utero passage of meconium could be normal or occur during hypoxia. In a hypoxic-ischemic event fetal reflex redistribution of cardiac output away from less vital organs such as the bowel results in intestinal ischemia followed by transient hyperperistalsis, anal sphincter relaxation, and meconium passage. During periods of stress the fetus may start gasping for breath and inhale the contaminated amniotic fluid resulting in severe pneumonia. Meconium is very irritating to the lower airway initiating bronchoconstriction and pulmonary edema resulting in ventilation-perfusion mismatch.

Clinical Signs

Foals will be meconium stained with red/brown ocular and nasal secretions. Crackles and wheezes or tubular lung sounds may be ausculted in all airways. Marked abdominal lift may be noted with every expiration and inspiration of air.

Diagnosis

Obvious clinical signs. Thoracic radiography will reveal cranioventral consolidation. Blood gas analysis will be characterized by a profound hypoxemia.

Treatment

The free fatty acids in meconium displace surfactant, which results in additional atelectasis and decreased lung compliance. Meconium also induces chemical pneumonitis accompanied by alveolar collapse and edema. Type II alveolar cells produce less surfactant, causing an increase in alveolar surface tension and decrease in compliance. Sloughed epithelium, protein, edema and hyaline membrane formation further contribute to respiratory distress. Initially, because of the severe inflammation that may occur with meconium aspiration antibiotic therapy would be imperative. Corticosteroids (Nebulized or intravenous) may also be warranted to help prevent an acute respiratory distress crisis. Nasal oxygen supplementation (6-8 l/min) is usually initiated. Mechanical ventilation is usually unrewarding. The use of nebulized N-acetylcysteine alters meconium’s physical properties by liquefaction. The liquefaction will help reduce the “mechanical” obstruction that could occur.

Equine Herpes Virus Type 1 and Type 4

Equine herpes virus 1/4 are α-herpes viruses that are enzootic in most horse populations. Most foals experience EHV-1 and/or EHV-4 infections early in life. Once the animal is infected via direct horse to horse contact or inhalation of aerosolized virus, the virus may establish latency. Latency sites for EHV-1 have been identified in neuronal cells of the trigeminal ganglia, peripheral blood lymphocytes, and lymphoid tissues. EHV 4 latency has been noted in lymphoid tissue, trigeminal ganglia and pulmonary epithelial cells. Newborn foals are most commonly exposed at birth; exposure prior to birth usually causes the mare to abort or give birth to a weak foal.

Clinical Signs

Newborn foals may be stillborn or very weak with death frequently occurring in 3 days. Should the foal live past 3 days, they are usually weak with pneumonia, depressed or convulsing.

Older foals will have initial signs including fevers, depression, lethargy, anorexia, lymphadenopathy, serous nasal discharge and coughing. The incubation period is between 3 and 10 days with clinical signs persisting for 2 to 7 days.

Diagnosis

Neonates

Antemortem diagnosis is difficult. Buffy coat could be assessed for EHV 1/4 polymerase chain reaction (PCR) and virus isolation. Liver and lung biopsy could help identify the infectious agent. Profound lymphopenia is characteristic of foals with EHV 1 infection.

Older Foals

Preliminary diagnosis can be made based on the rapid spread of the disease and the presence of compatible signs. Hematological findings are inconclusive. Virus isolation and PCR assessment of pharyngeal swabs anduffy coat provide good evidence for active infection. Acute and convalescent titers (3 weeks after the acute febrile stage) can be assessed.

Treatment

There is no specific therapy for the respiratory form of EHV 1/4. Palliative therapy is usually initiated which consists of NSAIDS to reduce fevers and the reduction of dust in the environment (i.e., soak hay and bedding, turnout when cleaning the stalls).

Neonatal forms of EHV1 require aggressive therapeutic support including fluids, total parenteral nutrition, nasogastric feedings
and antiviral medication (valacyclovir 30 mg/kg orally 2x daily for 7-10 days) may be of some benefit. However, this disease in foals is usually hopeless.

Prevention

Vaccination appears to be the best method of controlling this infection. Current vaccinations do not prevent the infection of EHV 1/4, but they can help reduce the severity of signs, viremia and attenuation of viral shedding to help protect the herd. Foals are usually started their vaccine program at 4 to 6 months of age with 2 doses 3 to 4 weeks apart and then the third booster 8 to 12 weeks later. Mares are vaccinated with a commercially approved vaccine at 5, 7 and 9 months of gestation in order to prevent fetal infection. Institution of an isolation program for incoming horses as well as a stall disinfecting program can help reduce the spread of this endemic disease.

Influenza

Equine influenza virus (EIV) is a major cause of equine respiratory disease. Equine influenza is a species-type A influenza virus from the orthomyxovirus family. The virus is transmitted by aerosol, wind, nose to nose contact, fomites such as tack, grooming equipment, machinery, feed and human contact. The virus is delicate within the environment and easily killed by heat, cold, desiccation and disinfectants.

The incubation period lasts 1 to 3 days and shedding of the virus may occur as early as 24 hours post infection. Shedding may continue for 7 to 10 days. This disease is uncommonly diagnosed in foals less than 8 months of age. Young immunologically inexperienced horses and unvaccinated horses are particularly susceptible to infection.

Clinical Signs

Clinical signs include fever, lethargy and anorexia. Fevers may range from 39.1°C to 41.7°C (102.5°F to 107°F). These signs last between 1 to 5 days. Inflammation and irritation of the airways may cause affected horses to acquire paroxysmal coughing. Increased inspiratory and expiratory bronchovesicular sounds are often heard on auscultation of the chest. Serous and/or light mucoid discharge, muscle soreness and stiffness are also frequently noted in affected animals. Some animals may develop a secondary bacterial pneumonia because of decreased bacterial clearance by the mucociliary apparatus.

Diagnosis

Influenza spreads rapidly and therefore rapid detection is necessary for control. Clinical diagnosis can be obtained with virus isolation from nasal or pharyngeal swabs, ELISA which detects viral nucleoproteins from nasopharyngeal swab samples, and/or paired serology looking for a 4x rise in titers.

Treatment

Treatment is mostly symptomatic. Nonsteroidal anti-inflammatory drugs may be administered to resolve the pyrexia and inappetence. Keeping the environment dust free with the soaking of hay, wetting down bedding and removing the patient from the barn when cleaning is imperative in preventing a secondary bacterial pneumonia. Broad spectrum systemic antibiotics may be needed for animals suffering from secondary bacterial pneumonia.

Pneumocystis Carinii

Formerly considered a protozoan organism which has recently been reclassified as a fungus. The infective stage or source of P. carinii is unknown but recent investigations suggest that it may be transmitted in water or an airborne route. P. carinii pneumonia is thought to occur primarily in immune-compromised foals as a complication of some other serious illness such as infectious pneumonia or severe combined immunodeficiency (SCID).

Clinical Signs

Dyspnea secondary to the plasmacytic lymphocytic interstitial pneumonia and the flooding of the alveoli with foamy exudate. This is usually an acutely fatal disease.

Diagnosis

It is very difficult to obtain a diagnosis antemortem. The use of thoracic radiographs may show a reticulonodular pattern (miliary). The use of either a bronchoalveolar lavage or a transtracheal wash has been suggested for identifying these organisms intracellularly in macrophages but results may be variable. Confirmation is by identification of the organisms by either silver staining or immunostaining.

Treatment

Prognosis is considered guarded but successful treatment has been achieved with the use of either potentiated sulfonamides and/or dapsone (3 mg/kg [1.4 mg/lb], PO, q 24 h; dose extrapolated from human data. Treatment is usually prolonged (45-50 days). Dapsone may be a useful adjunct to traditional treatment for P. carinii pneumonia in horses or as a sole medication for horses that cannot tolerate other treatments.

Pneumothorax (Figs. 5A and 5B)

Pneumothorax results when air enters the pleural space and reduces the negative pressure that keeps the lungs expanded and allows the horse to breathe. Pneumothorax is characterized as either open or closed. An open pneumothorax results in an injury to the thoracic wall leading to an influx of free air into the pleural space and lung collapse. A closed pneumothorax is the leakage of air into the pleural space from a pulmonary source such as a bronchopleural fistula. A tension pneumothorax occurs when a section of traumatized lung acts like a “valve” that allows the air to enter but not leave the pleural space.
The treatment of choice is prompt removal of free air via a thoracocentesis and suction. This procedure rapidly re-expands the lung and relieves the respiratory distress. If an open pneumothorax is diagnosed then surgical closure is warranted.

**Bacterial Pneumonia (Figs. 6-10)**

Pneumonia is the leading cause of morbidity and mortality in foals. Pneumonia in neonates is often associated with septicemia but may also occur secondary to meconium or milk aspiration. The etiology of foal pneumonia is complex because of the large number of factors that foals are exposed to which can set the animal up for a bacterial pneumonia. The majority of older foals develop bacterial pneumonia following a viral infection. Viral agents damage epithelial cells in the respiratory tree resulting in desquamation and focal erosion of the respiratory epithelium, interruption of the protective mucociliary blanket, and impairment of clearance mechanisms. Parasites can also predispose to bacterial pneumonia by causing immunosuppression secondary to malabsorption of nutrients or mechanical damage due to the migration of ascarids through the lung.

**Clinical Signs**

Foals usually present with exaggerated abdominal lift, elevated respiratory rate and flared nostrils. Bilateral pneumothorax will result in cyanosis and severe dyspnea.

**Diagnosis**

Thoracic radiography reveals a horizontal shadow beneath the thoracic transverse processes, which is consistent with a “line” representing the collapsed lung(s).

Thoracic ultrasonography will reveal horizontal air artifacts in the midthoracic or dorsal regions thereby not allowing the examiner to identify the sliding motion of the visceral pleural against the parietal pleura.

If radiological diagnostics are not available then the clinician can use suction via a thoracocentesis as a diagnosis.

**Treatment**
The trocar has been removed from the chest drain and drainage of the effusion should occur immediately. Fluid can be collected for cytological and culture evaluation. A non-lubricated condom is placed at the end of the tube to allow unidirectional drainage. The drain should be monitored closely to ensure proper function so that aspiration of air and pneumothorax does not occur.

Clinical Signs

Clinical signs vary considerably, with some foals having high respiratory rates while others may have an occasional cough. Coughing, either intermittent or paroxysmal, is an important early indicator for respiratory disease. Respiratory rates greater than 30 BPM warrant further diagnostic evaluation which should include auscultation of the chest to assess if inspiratory or expiratory crackles or wheezes are noted. Most severely affected foals will have flared nostrils, an abdominal component to breathing with minimal thoracic excursion. Bilateral discharge from profuse to scant and fevers are common findings in foals.

Diagnosis

Auscultation of the chest with and without a rebreathing bag is needed for assessment of bronchovesicular (BV) sounds relating to alveolar ventilation. Adventitious lung sounds are considered abnormal and are described as either crackles or wheezes. Crackles are produced upon immediate equalization of pressure in two compartments after airways have reopened. Crackles have been characterized as sounds like cellophane being crumpled and are associated with obstructive disorders such as bronchopneumonia. Wheezes are associated with vibrations of airway walls and can be auscultated during inspiration or expiration. Wheezes are characterized as either monomorphic or polymorphic depending on the extent of the pneumonia (multiple sites). Pleural friction rubs are generated by the movement of visceral and parietal pleura which cross each other.

Percussion of the thorax consists of tapping the intercostal spaces of the thorax using a large spoon and a neurological hammer while evaluating the sound produced. Aerated tissues produce a resonant sound while consolidated lung, lung abscesses and bowel will produce a dull sound. Once the entire lung field has been percussed it is compared to that of a normal foal’s lung field.

Ultrasonography and radiography can help identify and characterize pathologic lesions. Culture and cytological evaluation via a transtracheal wash can be performed to help determine the etiological bacteria involved.
**Treatment**

The selection of an antimicrobial regimen is based on the following:

- Identifying the causative agents
  - Common gram + agents include *Streptococcus zooepidemicus*
  - Common gram – agents include *Actinobacillus equuli and Pasteurella*
  - Uncommon: Fungal. Foals with diarrhea are more at risk
- Determining the susceptibility patterns
- Host factors
- Tissue distribution of the drug

Foals with pneumonia secondary to severe septicemia require broad-spectrum therapy. Older foals with uncomplicated pneumonia can be initially treated with a gram positive regime with the understanding that if a poor response is noted then the institution of broad-spectrum therapy will be necessary.

Anti-inflammatories are commonly used in foal pneumonia to help control fever and potentially improve inflammation of the lower respiratory tract. Foal pneumonia can also benefit from treatment with bronchodilators (see Hyperactive Airways). It is very important to keep dust levels to a minimum in the foal’s environment. This includes soaking all hay given to the mare and foal for 14 days and removing the foal from the stall when it is being cleaned.

Pleural drainage may be warranted when the removal of exudate and debris from the pleural space allows for the re-expansion of the lung. Decisions regarding pleural drainage are based on classification of the pleural fluid as a complicated or uncomplicated effusion. Uncomplicated effusions are those that do not have sufficient volume to cause respiratory distress and are classified as a transudate which contain less than 10,000 nucleated per µl and less than 2.5 g/dL of protein. Nondegenerative neutrophils are the primary cells seen, mononuclear cells (macrophages, lymphocytes) are the second most common. Complicated effusions are characterized as having a sufficient volume to cause respiratory distress, increased numbers of degenerative neutrophils, putrid odor, cytologically visible bacteria or positive culture results. Most complicated effusions require pleural drainage.

**Juvenile Hyperreactive Airway Disease**

This disease tends to affect foals 4-7 months of age and is poorly understood. It appears that juvenile hyperreactive airway disease is a syndrome of small airway disease that has minimal response to antibiotics alone. The majority of the cases in the United States present between June and September.

**Clinical Signs**

Clinical signs are consistent. Foals present with a history of tachypnea with an exaggerated abdominal component. A tracheal rattle and flared nostrils with minimal air flow is a common clinical finding. Auscultation of the chest reveals polyphonic crackles and wheezes associated with the marked bronchoconstriction and excessive mucus production that accompanies this disease.

**Diagnosis**

Clinical signs are in excess of the diagnostic results. Minimal growth is noted on the transtracheal wash. The complete white blood cell count is typically normal with a normal fibrinogen level. Signs tend to persist despite broad-spectrum antimicrobial treatment.

Thoracic ultrasonography is usually unrewarding revealing minimal pleural irregularities (comet tails). Thoracic radiographs reveal thickened bronchioles and a mild interstitial pattern.

**Treatment**

Treatment is aimed at resolving cyanosis with intranasal oxygen, bronchodilators and reducing the inflammation with in the lung. Foals should be placed on 7-10 liters of oxygen via a nasal cannula. Bronchodilators are used to relieve the obstruction of the small airways caused by airway smooth muscle contraction. The administration of bronchodilators should always be combined with strict environmental dust control (soaking hay and removing from stall/barn when cleaning) and corticosteroid therapy to help reduce the inflammation of the lower airways.

Various bronchodilators used include:

- **Clenbuterol** (0.8 to 3.2 µg/kg PO BID) is an excellent bronchodilator with mucokinetic properties to help clear mucus from the airways.
- **Aminophylline** (5 to 10 mg/kg diluted in fluids can be given BID to QID IV) is a xanthine derivative that is a bronchodilator, enhances mucociliary clearance, contractility of the diaphragm and delays fatigue of the muscles responsible for respiration.
- **Glycopyrrolate** is used as a “rescue” drug when immediate bronchodilation is needed. Glycopyrrolate is a synthetic antimuscarinic agent that blocks the M3-muscarinic receptors and causes bronchodilation (0.002 to 0.007 mg/kg IV once).
- **Inhalation bronchodilators** (during severe airway obstruction these drugs have poor pulmonary distribution).
  - Albuterol is a beta2 agonist which serves as a rescue therapy. This medication can improve pulmonary function by 70% within 5 minutes of administration. Unfortunately the effect only lasts 1-3 hours.
    - **Give 3-6 activations (450 mcg) 2x daily**
Nebulize 2 mg to 5 mg ranging from every 15 minutes for 1 hour to every 6-8 hours
  - Ipratropium bromide
    - 3 activations 2x daily
    - Nebulize 1 vial (500 mcg) 3-4 x daily

Systemic dexamethasone has been demonstrated to improve lung function within hours of administration with a maximal response obtained by day 7. Steroids not only reduce inflammation within the chest but over time can up regulate the number of beta2 receptors within the airways. Dexamethasone is administered intravenously (0.1 mg/kg) 1x daily and then for another 4-5 days (0.05 mg/kg) after clinical signs resolve. Inhaled steroid therapy can also be used in conjunction of systemic steroids.

Inhaled steroids may continue to administer for several weeks after the systemic steroid therapy has been discontinued.

Examples of inhaled steroids in foals include the following:
  - Fluticasone (220 mcg/activation) Give 4-8 activations once a day for 2 weeks and then every other day for another 2 weeks
  - Beclomethasone dipropionate (80 mcg/activation) give 5-8 activations once a day for 2 weeks and then every other day for another 2 weeks

### Table 1. Dosages for Nebulization Therapy

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<thead>
<tr>
<th>Gentamicin</th>
<th>Aminoglycosides</th>
<th>2.2 mg/kg (dilute with sterile 0.45% saline solution to 50 mg/ml (Ref 5)</th>
<th>q 12-24 h</th>
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<tr>
<td>Ceftiofur Sodium</td>
<td>Cephalosporin</td>
<td>1-3 mg/kg (Ref 5 and modified by Author)</td>
<td>Q 12-24 h</td>
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**Bronchodilators**

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<tr>
<th>Ipratropium</th>
<th>Anticholinergic Agent</th>
<th>0.5 mg (Ref. Author)</th>
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<tr>
<td>Albuterol</td>
<td>B2-adrenergic receptor agonist</td>
<td>0.01-0.025 mg/kg (Ref Author)</td>
<td>q 4 h -12h</td>
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**Corticosteroids**

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<tr>
<th>Dexamethasone</th>
<th>Corticosteroid</th>
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<tr>
<td>Fluticasone</td>
<td>Corticosteroid</td>
<td>1 mg (Ref Author)</td>
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**Mucolytics**

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<th>N-Acetylcysteine 20%</th>
<th>Mucolytic</th>
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<tr>
<td>0.9% Saline Solution</td>
<td>Mucolytic</td>
<td>10-30 mL</td>
<td>As often as required</td>
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</table>

### REFERENCES AND FOOTNOTES


4. Tripathi S, Saili A. The effect of steroids on the clinical course and outcome of neonates with...


b. Flexineb®, Haygain, Union City, TN.