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THORACOSCOPY IN HEALTHY HORSES

By

John Ferruccio Peroni, DVM

A THESIS

**Submitted to
Michigan State University
in partial fulfillment of the requirements
for the degree of**

MASTER OF SCIENCE

Department of Large Animal Clinical Sciences

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ABSTRACT
THORACOSCOPY IN HEALTHY HORSES

By

John Ferruccio Peroni, DVM

Thoracoscopy is an endoscopic surgical method used to evaluate disease present within the thoracic cavity. A review of the pertinent literature confirmed the practical usefulness of equine thoracoscopy; however, no studies have been aimed at determining the safety of the procedure. Thoracoscopy has become increasingly more applicable in equine clinical practice, justifying the need to investigate the hemodynamic and cardiopulmonary consequences of the procedure. Six healthy horses were controlled with the sedative-analgesic detomidine HCl and used to evaluate cardiovascular (heart rate, cardiac output, systemic blood pressure, and peripheral and pulmonary vascular resistances) and respiratory (respiratory rate, blood gas tension, and pulmonary arterial pressure) changes occurring during two fifteen-minute pneumothorax periods created to complete the thoracoscopic examination. Also, the study allowed the assessment of the normal thoracic structures viewed with a rigid, 58-cm-long telescope.

The procedures were safely completed, were well tolerated, and had an uncomplicated, long-term outcome in all horses. Most of the cardiopulmonary changes were associated with detomidine administration and only modest hypoxemia was caused by pneumothorax periods. Thoracoscopy was determined to be a successful and safe surgical method for the evaluation of the normal anatomical structures of the equine thorax.

To My Parents

To Dana

ACKNOWLEDGMENTS

I would not have completed this task without the guidance of my mentors: Dr. Robinson, Dr. Stick, and Dr. Derksen.

My sincere appreciation goes to Cathy Berney and Dr. Cindy Jackson for the help in the data collection process and to Victoria Hoelzer-Maddox for the technical assistance in formatting the thesis.

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INTRODUCTION

The use of telescopes to perform surgery within body cavities has been perfected over the years. Endoscopic procedures have clear-cut advantages over more traditional open methods: less postoperative pain, decreased convalescence periods and earlier return to function. State-of-the-art equipment and techniques are available for operating on articulations (arthroscopy), abdominal cavity (laparoscopy), and thorax (thoracoscopy). Thoracoscopy has been used in humans since 1910 to examine the thorax as an alternative method to thoracotomy. Access to the thoracic organs traditionally requires sternotomy, rib distraction, or resection. With the advent of thoracoscopy, procedures have been performed via small intercostal incisions sized to allow telescopes to be placed in the thorax, thus largely improving the field of vision. Compared to open procedures, thoracoscopy is considered minimally invasive, is better tolerated by the patient, and is associated with faster healing of the surgery site and minimal post-operative pain. Specific thoracoscopic techniques were developed in humans during the early 1990s and since then have been included in the board specialty of video-assisted thoracic surgery (VATS).

There are several important physiological issues associated with any type of invasive thoracic procedure. Thoracic surgery is complicated by impaired function of the heart and lungs. Relatively straightforward procedures, such as pleural biopsy, may not be technically challenging but require the invasion of the thorax. This may alter

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cardiovascular and respiratory functions and complicate anesthesia. Furthermore, the procedure is usually performed in the face of disease, thus further increasing the risks of surgically exposing the thoracic organs to the environment. Access to pleural anatomical structures is gained by penetrating the thoracic wall, therefore establishing a communication between the environment and the pleural space. Thoracic operations are conducted in the presence of a pneumothorax, which has specific cardiopulmonary consequences.

In order to maximize the view of the thorax and to maintain adequate ventilation and ensure a safe anesthesia, thoroscopic procedures in humans are generally carried out in lateral decubitus and use the one-lung ventilation technique. The use of a double lumen tube allows independent ventilation of one lung. Alternatively, bronchial blockers are used to obstruct one mainstem bronchus and cause absorptive atelectasis in the occluded lung, thus achieving alveolar collapse and exposure of the ipsilateral pleural anatomical structures.

Pneumothorax in humans has been associated with ventilation-perfusion mismatch and an increased risk for hypoxemia. Perfusion of the non-dependent, non-ventilated lung is primarily reduced because of the hypoxic pulmonary vasoconstrictor mechanism. An increase in pulmonary vascular resistance actively diverts blood flow to the dependent, ventilated lung. Increasing the fraction of inspired oxygen reduces the risks of hypoxemia and therefore ventilation with 100% O₂ is common during human thoracoscopy. Ventilatory frequency is also adapted to maintain appropriate CO₂ levels, and positive end expiratory pressure (PEEP) ventilation is used to improve oxygenation of the ventilated lung.

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The use of thoracoscopy in the horse has been reported in clinical cases for the diagnosis of pleural neoplasia. More recently, thoracoscopy was used for the diagnosis and treatment of 28 cases of equine pleuro-pulmonary infectious and neoplastic diseases. Thoracoscopy is generally performed with the horse standing, in order to avoid the possible complications associated with pneumothorax and general anesthesia. Single-lung ventilation, although common practice in small animal thoracic surgery, has not been established as a routine technique in equine anesthesia.

The cardiopulmonary consequences of thoracoscopy performed in the healthy, standing, and sedated horse were investigated in our study. The sedation protocol chosen was a continuous detomidine infusion administered to horses while performing a right hemithoracoscopy or a left hemithoracoscopy. All horses were also sedated and underwent a sham procedure. The majority of the cardio-pulmonary effects were found to be associated with detomidine administration with a modest decrease in arterial oxygen tension associated with pneumothorax. The experiments were also conducted to allow recording of the procedures and to determine the normal thoracic anatomy as viewed through the endoscope.

CHAPTER 1

LITERATURE REVIEW

Thoracoscopy in human medicine

In 1910 at the University of Stockholm Hans Christian Jacobeus presented the first work on thoracoscopy.¹ Jacobeus used a modified cystoscope without magnification to diagnose tuberculous effusions and later lyse pleural adhesions and diagnose lung cancer.² Thoracoscopy remained confined to the diagnosis and treatment of tuberculosis for several decades and was virtually abandoned, especially in the United States, in the 1950s with the advent of effective chemotherapeutic agents for treatment of tuberculosis. In the 1970s and 1980s only a few North American surgeons were using open endoscopes to diagnose pleural disease and perform small lung biopsies.^{3,4} With the improvement of lighting systems and optics, specific instrumentation was designed for endoscopic thoracic procedures.⁵ In the early 1990s the advent of video technology allowed thoracoscopy to become a procedure viewed on monitors and accessible to surgeon and assisting team. Traditional thoracoscopic techniques were abandoned in favor of video-assisted thoracic surgery (VATS).^{6,7}

There are several diagnostic and therapeutic indications in people for operations that fall under the category of VATS procedures. In the human field, commonly performed thoracic procedures include pleural and lung biopsy,^{8,9} drainage of pleural effusion and lysis of pleural adhesions, mediastinal lymph-node biopsy,¹⁰ partial

pericardiectomy,¹¹ and esophageal procedures.¹² These procedures are carried out under local, regional, or general anesthesia. Local and regional anesthesia are used for minor elective procedures such as pleural biopsies, and small-caliber endoscopes are preferred. During most diagnostic and therapeutic thoracoscopic procedures, patients are under general anesthesia, a single lung is ventilated, and the operated lung is collapsed to provide excellent exposure of the hemithoracic structures. There are two main techniques to achieve ventilatory separation of the left and right lungs: a double lumen endotracheal tube and a standard single lumen endotracheal tube coupled with a bronchial blocker.^{13,14} Collapse of the lung achieves sufficient exposure of the pleural space and does not require air insufflation into the hemithorax, however, some authors have advocated the use of CO₂ insufflation to maximize the extent of the pneumothorax.¹⁵

The development of thoracoscopy has been based on the realization that adequate and often times superior intra-thoracic observation can be obtained without a large incision and therefore the term “minimally invasive” has been associated with this procedure.¹⁶ Most thoracic procedures are completed with three ports: one for endoscopic access and two for accessory instrumentation. Portal placement may be tailored to the specific needs of the procedure; however, certain guidelines for portal placement should be followed. Portals should not be placed too close together and should not be along the same line (intercostal space). The “baseball diamond analogy” is commonly used to define the surgical approach.¹⁷ The endoscopic portal is identified with home plate, while the accessory portals are usually located in correspondence with first and third base. The area of disease should be located between the pitcher’s mound and second base. This setting allows appropriate triangulation techniques to be accomplished and achievement of tension and countertension during tissue dissection.

Once all ports have been placed, the telescope position can be varied in order to gain maximum exposure.

Thoracoscopy in veterinary medicine

In 1985 Mackey and Wheat used a 130° rigid endoscope to examine the thorax of 15 horses (10 experimental and 5 clinical cases).¹⁸ The technique was advocated by these authors as an adjunctive diagnostic procedure to provide more accurate prognosis. A report of 8 clinical cases and 6 normal horses was provided by Mansmann and Strother in 1985.¹⁹ The authors combined a description of the normal thoracic anatomy with a series of clinical cases of chronic thoracic disease. All cases were examined using a flexible, 110-cm-long fiberoptic endoscope. Thoracoscopy in the horse has since gained popularity. In 1998 a report described 28 clinical cases diagnosed and treated with thoracoscopy.²⁰ The procedures were performed in the standing horse and also under general anesthesia. Thoracoscopy was used for the diagnosis and treatment of thoracic neoplasia, pleuropneumonia (adhesion breakdown and intrathoracic drain placement) thoracic abscessation, pericarditis, and diaphragmatic hernia.

Thoracic neoplasia in the horse has been investigated by thoracoscopy. The use of this procedure provided the ante-mortem diagnosis of thoracic tumors or metastasis and allowed the collection of tissue biopsy samples and subsequent identification of the neoplastic mass. Both flexible and rigid endoscopes have been used to diagnose pleural metastatic invasion of a gastroesophageal squamous cell carcinoma,²¹ of a disseminated hemangiosarcoma,²² and of cholangiocellular carcinoma.²³

No reports were found in the veterinary literature regarding the hemodynamic consequences of thoracoscopy that are encountered when performing the procedure in the

standing or anesthetized horse. The cardiopulmonary effects of conventional ventilation and diagnostic thoracoscopy have been reported in the dog.²⁴ Conventional ventilation and non-selective single lung ventilation were performed in this study during thoracoscopy performed in lateral recumbency and with a prolonged pneumothorax period (mean time 56 ± 5 min.). The procedure was well tolerated and standard ventilation technique during sustained pneumothorax was an acceptable anesthetic method in the dog. The literature reviewed agrees on three main points with regard to the clinical importance of thoracoscopy: first, that thoracoscopy should be primarily employed in cases of chronic disease non-responsive to standard antimicrobial therapy and drainage; second, that thoracoscopy is technically difficult and horses tolerate the procedure well; third, that thoracoscopy finds an important place alongside the ancillary tools used in the diagnosis of pleuropulmonary disease.

Pneumothorax

By definition, pneumothorax indicates the presence of “free” air within the pleural space. Typically, air will be confined to the pleural space; however, free air may be contained within the adventitial tissue planes (interstitial pulmonary emphysema) or in the mediastinum (pneumomediastinum).

Classification

Primary spontaneous pneumothorax

Primary spontaneous pneumothorax occurs without trauma to the chest and in the absence of lung disease. It is primarily a disease of young tall and thin males and is six times more common in men than women.²⁵ Primary spontaneous pneumothorax results

from the rupture of small apical emphysematous blebs, which may be congenital or result from bronchiolar disease with obstruction. Tall individuals are more likely to develop the condition because of a more negative pressure present at the apex of the lung, which can cause distention of apical alveoli. Individuals may be asymptomatic, but, more commonly, clinical signs include acute chest pain and dyspnea. Cough is also associated with the disease in approximately 50% of the cases. A familial tendency for increased incidence of spontaneous pneumothorax has been found in individuals genetically predisposed to alveolar bleb formation.²⁶

Secondary spontaneous pneumothorax

Secondary spontaneous pneumothorax occurs spontaneously as a consequence of clinical lung disease. Chronic obstructive pulmonary disease is the most common underlying disease and may confuse the clinical signs, making the diagnosis of secondary pneumothorax difficult.²⁷ More severe clinical signs are associated with secondary pneumothorax. Gas exchange abnormalities may lead to hypoxemia, cyanosis, and hypotension.

Neonatal spontaneous pneumothorax

Neonatal spontaneous pneumothorax occurs during the first few days of life. The events that lead to lung expansion shortly after birthing can lead to pneumothorax. A large gradient between intra-bronchial and intra-pleural pressures is necessary at birth to achieve lung inflation with the first inspiration act. The pressure gradient may be of the order of 40 cm H₂O while the neonate diaphragm can generate a pressure of -60 to 80 cm of water.²⁸ Complete aeration of the alveoli is achieved within the first few minutes

after birth. If groups of alveoli do not expand promptly (atelectasis), as in the premature infant, for lack of surfactant or subsequent to aspiration of mucus or meconium, abnormally high pressures can be created in certain open alveolar groups, leading to rupture of the alveoli. In addition, positive pressure ventilation used at birth for resuscitation may be implicated in the etiology of spontaneous neonatal pneumothorax.

Traumatic pneumothorax

Traumatic pneumothorax is usually secondary to penetrating chest trauma or blunt chest trauma. Wounds to the thorax may expose the thorax to the environment and create a pneumothorax. Non-penetrating blunt trauma may also cause pneumothorax by compressing and rupturing alveolar groups, causing air to leak from the lower respiratory tree into the pleural space.

Iatrogenic pneumothorax

Certain medical procedures may lead to pneumothorax as a complicating factor. This may be the case in thoracentesis and pleural or lung biopsy. A pneumothorax is a necessary feature of thoracoscopy. Lastly, barotrauma should be included in this category and can be caused as high airway pressures are induced during mechanical ventilation. Peak airway pressures should not exceed 50 to 60 cm H₂O.

Catamenial pneumothorax

Catamenial pneumothorax is associated with menstruation and can spontaneously recur in women 48 to 72 hours following menses. It is often associated with endometriosis and appears to have two possible etiologies. Pleural or diaphragmatic

endometriosis or collection of endometrial tissue within the pleural mesothelium may be one of the causes of the syndrome.²⁹ Air may leak into the pleural space from the lung where endometrial implants are present. In addition, during thoracotomy performed to treat catamenial pneumothorax, small diaphragmatic defects have been found. These defects may lead air formed in the genital tract to escape into the peritoneal cavity and then into the pleural space. Catamenial pneumothorax seems to be almost in all cases right sided and small in volume.³⁰

Tension pneumothorax

A tension pneumothorax exists when the pressure in the pleural space exceeds atmospheric pressure. It may be a complication of spontaneous or traumatic pneumothorax, but it commonly develops as a consequence of mechanical ventilation or during cardio-pulmonary resuscitation maneuvers. A ball-valve effect allows air to escape into the pleural space either from a defect in the chest wall or within the visceral pleura, but air is prevented from evacuating in the opposite direction. Over time pressure increases in the thorax, leading to severe cardio-pulmonary compromise.

Pathophysiology

During spontaneous breathing and in normal conditions, the pleural pressure (Ppl) is negative with respect to alveolar pressure and atmospheric pressure. Normal end-expiratory pleural pressure is approximately -5 cm H₂O. The Ppl becomes more negative (-7.5 cm H₂O) during inspiration when the chest wall is expanded through the effort of the inspiratory muscles (mainly diaphragm and external intercostal muscles). Alveolar pressure changes during the respiratory cycle facilitate movement of air in and out of the

lungs. When inspiration begins, alveolar pressure becomes slightly negative with respect to atmospheric pressure, allowing air to flow inward. Therefore, Ppl and alveolar pressure undergo similar directional changes during the respiratory cycle, becoming more or less negative, respectively, during inspiration and expiration. The difference between Ppl and alveolar pressure is termed transpulmonary pressure and measures the elastic forces working to collapse the lung during lung expansion. The ease with which the lung expands while transpulmonary pressure increases is called compliance (relationship between lung volume and transpulmonary pressure changes).

Maintenance of a negative pleural pressure is best understood by considering the relationship between chest wall and lung. A negative Ppl is maintained throughout the respiratory cycle because of the tendency of the lung to collapse and of the chest wall to expand. The tendency of the chest wall to expand is counteracted by the normally negative Ppl drawing the chest wall inward. A pressure-volume curve can be drawn for the lung/chest wall complex.³¹ Let us consider the lung/chest wall complex at three different lung volumes in order to illustrate this relationship. At residual volume (RV), which defines the amount of air remaining in the lung after maximum expiration, the lung is only minimally inflated so the lung/chest wall system is dominated by the high recoil force of the chest wall, which tries to expand the system. Ppl at RV is at its least negative value. Functional residual capacity (FRC) defines the volume of air remaining in the lungs at the end of a passive expiratory effort. At FRC the recoil forces of the chest wall are equal to the forces that tend to collapse the lung. Therefore the transmural pressure of the lung/chest wall system is zero. Finally, at total lung capacity (TLC), which is the volume of air contained in the lungs at the end of maximal inspiration, transmural pressure increases in opposition to the pulmonary recoil forces, which would

tend to reduce lung volume. At TLC Ppl is most negative.³² When a pneumothorax occurs, the relationship between chest wall and lung is interrupted, and the chest wall, controlled only by recoil forces, tends to expand. The lung, under the influence of its own elastic properties, collapses. When pleural and atmospheric pressures are in equilibrium, the lung reaches its minimal volume, and further increases in pleural pressure lead to ipsilateral chest wall expansion and mediastinal displacement toward the contralateral hemithorax.

Lung collapse affects pulmonary function. The presence of air in the pleural pressure uncouples the chest wall from the lung. As pleural pressure increases, transpulmonary pressure (recoil pressure) decreases. The recoil pressure across the chest wall is also changed so that the thorax expands as the lung collapses. With large pneumothoraces a mediastinal shift can be detected, on radiographic examination, toward the contralateral hemithorax. In humans, total lung capacity (amount of air contained in the lung at the end of maximal inspiration) has been shown to decrease with lung collapse and this will decrease vital capacity.³³ In addition, a decrease in arterial oxygen tension has also been found following spontaneous pneumothorax in people.³⁴ The decrease in arterial oxygen has been attributed to a low ventilation-perfusion ratio in certain areas of the lung. Airway closure has been demonstrated to occur at low lung volumes in patients with pneumothorax.³⁵ Anatomic shunts also seem to contribute to the decrease in PaO_2 , especially with large pneumothoraces.

Arterial oxygen saturation decreases immediately following lung collapse. Over time, perfusion adaptations occur and, in an effort to compensate for decreased arterial oxygen tension, blood flow is diverted from areas of low ventilation within the collapsed lung and directed toward either better-ventilated areas of the collapsed lung or to the non-

collapsed and ventilated lung. Arterial oxygen saturation has been shown to normalize in as early as 24 hours in patients with spontaneous pneumothorax treated conservatively with observation only.³³ Hypoxic pulmonary vasoconstriction (HPV) is an important mechanism by which the pulmonary circulation adapts to hypoxia. HPV is based on a direct contraction of the vascular smooth muscle to a decreased alveolar oxygen concentration. The intent of this system is to preserve arterial blood oxygenation diverting pulmonary blood flow away from poorly ventilated regions of the lung and therefore optimize ventilation and perfusion. It appears that the HPV is initiated within seconds from the hypoxic event and then progressively intensifies over time at a speed dependent upon the velocity of decline in alveolar oxygen tension. The small pulmonary arterioles are the primary site for constriction. The mechanism by which a decreased oxygen tension is detected is not entirely understood. The events leading to arteriolar contraction depend on oxygen concentration in proximity to the vascular smooth muscle cells.³⁶ In addition, HPV appears to be endothelium independent, is modulated by various peptides such as prostacycline, nitric oxide, and endothelin and is probably caused by depolarization of the smooth muscle cell leading to increased intracellular calcium and consequent contraction. With pneumothorax it is possible that alveolar groups of the collapsed lung become acutely atelectatic and the pulmonary circulation rapidly initiates this compensatory mechanism. This has been shown to occur following open chest surgical procedures that involve lung manipulation. In contrast, it has been shown that in dogs the HPV response may be attenuated following lung injury. This loss of reactivity has been shown to depend on the release of prostanoids (prostacyclin) by the endothelium. The biological response to pneumothorax appears to be complex and is unlikely to be dominated by a single event but rather involves a multitude of response

mechanisms.³⁷ Importantly alveolar hypoxia variably affects ventilation perfusion relationships in normal lungs. HPV may help improve arterial oxygenation in some cases or may have no effect. The variability is due to concurrent changes in cardiac output, pulmonary arterial pressure, and ventilation.

Cardiac output can be negatively affected by pressure changes occurring within the chest cavity. Specific sigmoid curves relating cardiac output to right atrial pressures have been established to define cardiac function. To any given right atrial pressure at input, corresponds a specific cardiac output. This relationship depends on numerous factors (exercise, age, disease) and is affected by pleural pressure. Normally an external pressure of -5 mm Hg surrounds the cardiac muscle. If the chest is opened to atmosphere, the pressure around the heart will increase to 0 mm Hg. In order for cardiac output to remain unchanged, right atrial pressure is going to increase proportionally to the increase in pleural pressure. A sustained increase in right atrial pressure, such as in pneumothorax conditions, can lead to a pressure increase in the systemic circulation and decrease venous return of blood to the heart, negatively affecting cardiac output. Severe cardiopulmonary compromise has been shown to occur with tension pneumothorax. The progressive increase in pleural pressure causes "tension" to develop on mediastinal structures. It was thought that pressure on the mediastinum decreased venous return to the heart and therefore cardiac output. However, blood gas deterioration leading to cardiopulmonary failure seems to precede cardiac output decrease.³⁸ In a study performed in goats and monkeys, tension pneumothorax initially did not drop cardiac output but increased right atrial, right ventricular, and pulmonary arterial pressures. Immediately after initiation of the tension pneumothorax, animals experienced

a rapid fall in PaO_2 (from 90 to 20 mm Hg), which was attributed to a severe ventilation to perfusion mismatch.

Treatment of pneumothorax

There are two principal goals in the treatment of pneumothorax: first, eliminate the air from the pleural space, and second, prevent recurrence. In the case of a spontaneously occurring pneumothorax the treatment options include simple observation allowing the air to be slowly evacuated from the pleural space. It has been shown that the rate of spontaneous absorption takes time.³⁹ Approximately 1.25% of the volume of one hemithorax is reabsorbed in 24 hours, which means that a 20% pneumothorax would take about 16 days to be spontaneously eliminated. The rate of pleural air absorption can be accelerated with tracheal administration of 100% supplemental O_2 .⁴⁰ This is based on the principal that gases diffuse through biological membranes at a rate depending on pressure gradients. In the case of a pneumothorax, the Fick principle dictates that the rate at which air will diffuse from the pleural space into the pulmonary capillaries depends on the partial pressure differences of each gas, the blood flow per surface available for gas exchange, and the solubility of each gas in the tissues.

If a pneumothorax occurs at sea level, the pressure of the air in the thoracic cavity is 760 mm Hg minus the -5 mm Hg of intrapleural pressure (about 755 mm Hg). In the capillary blood the sum of the partial pressures of gases is about 706 mm Hg ($\text{PH}_2\text{O} = 47$, $\text{PCO}_2 = 46$, $\text{PN}_2 = 573$, and $\text{PO}_2 = 40$ mm Hg). The gradient for gas exchange between capillary bed and pleural space is therefore 49 mm Hg (pneumothorax = 755 mm Hg - capillary blood = 706 mm Hg) favoring the slow reabsorption of the trapped air. When 100% O_2 is administered, the partial pressures in the capillary blood

favor the formation of a greater pressure gradient, thus decreasing the partial pressure of N_2 to close to zero while the partial pressures of oxygen, carbon dioxide, and water remain basically unchanged. Because of the fall in PN_2 , the net gradient increases to about 500 mmHg, which is almost 10 times greater than that achieved while breathing room air. This has been shown clinically.⁴¹

Evacuation of a pneumothorax can be achieved by several invasive methods. Simple aspiration has minimal morbidity and in veterinary medicine is reserved for small animals and in human medicine has been successfully used in children and adults presented with the first occurrence of primary spontaneous pneumothorax. Tube thoracostomy is used in cases of secondary spontaneous pneumothorax in humans and is achieved by placing a chest tube through an intercostal space. This is frequently done in horses that present with open chest trauma. The pneumothorax may be initially aspirated using a mechanical suction unit followed by the intra-thoracic insertion of a large-bore chest tube located in the proximal third of a caudal intercostal space. The chest tube is coupled with a Heimlich valve consisting of a collapsible rubber tube connected to the chest tube. On inhalation a negative pressure collapses the rubber tubing and on exhalation the tube opens and allows the air trapped in the thorax to escape. Open thoracic procedures such as thoracotomy or thoracoscopy are employed for pneumothorax conditions that do not resolve, when there are rib fractures associated with the pneumothorax causing lung laceration, or in case of a broncho-pleural fistula forming as a consequence of pleuro-pneumonia. Persistent air leaks have been treated in humans with open thoracotomy with the intent of identifying and treating the damaged lung parenchyma following either lung lobe resection, bleb resection, or trauma.⁴² Persistent communications may form between a bronchus and the pleural space (broncho-

pleural fistulas) during the healing processes of chronic pleuritis. These conditions are treated by resection of the chronic adhesion formed between the lung surface and the chest wall. This procedure has been executed in humans using thoracoscopy.

Re-expansion pulmonary edema should be considered a potential complication of reducing a pneumothorax with suction application. Pulmonary edema may develop acutely after re-inflation and can cause hypoxemia and hypotension. Mechanically stressed pulmonary capillaries damaged by rapid and high-pressure lung inflation decrease their ability to retain protein and leak edema fluid in the interstitium. The high protein content of the fluid supports the fact that it originates from the capillary bed rather than from increased hydrostatic pressure. Typically the chance of re-expansion pulmonary edema developing is increased if the pneumothorax condition has been present for several days. High negative intra-pleural pressures should not be generated when attempting to correct a pneumothorax. A pressure of -20 mm Hg or less applied to the chest is considered safe.⁴³

Detomidine

Thoracoscopy is generally performed with horses standing in order to eliminate the risks associated with general anesthesia and because the thoracic anatomy can be effectively viewed with thoracoscopy in the awake horse. Analgesia is provided by anesthetizing the surgical sites with local anesthetics. It is, however, important to provide systemic analgesia and sedation by administering either xylazine or detomidine combined with butorphanol if necessary (neuroleptoanalgesia). Detomidine is favored over xylazine for thoracoscopy for its longer-lasting effects, for the profound sedative-

analgesic effect, and because it can be prepared and administered as an intravenous infusion for the duration of the procedure.

Detomidine is a selective α_2 -adrenergic receptor agonist⁴⁴ used to provide sedation and analgesia in the horse.⁴⁵ The behavioral, cardiovascular, and pulmonary effects of detomidine are dose-dependent and similar to those induced by xylazine, a commonly used but less selective α_2 -adrenergic receptor agonist. Compared to xylazine, detomidine has been shown to be 10- to 100-fold more potent, with a longer-lasting effect.⁴⁶

The effects of detomidine have been evaluated following bolus administration⁴⁶ and after continuous infusion.⁴⁷ α_2 -adrenoceptor activation in the brain and spinal cord is the underlying mechanism of action of detomidine. Sedation appears to result from the reduction of noradrenergic stimuli to the hippocampus, thalamus, and cerebral cortex, resulting in behavioral depression. In addition, the release of norepinephrine is prevented in the thalamus and cortex following α_2 -receptor activation, leading to profound sedation.⁴⁸

Recognized cardiovascular effects of detomidine administration are initial vasoconstriction followed by vasodilatation, arrhythmias, bradycardia, reduced cardiac output, and hypotension.⁴⁹ Bradycardia and atrioventricular conduction abnormalities (AV blockade) are attributed to a decrease in central sympathetic stimulation and a vagally mediated baroreceptor response to the initial hypertension seen following detomidine administration. Cardiac output may be affected by changes in heart rate, preload, afterload, and contractility. Detomidine-induced bradycardia plays an important role in cardiac output reduction. It has, however, been noticed that cardiac output changes do not regularly correlate with a decreased heart rate.⁴⁶ Stroke volume changes are not consistently observed with either detomidine or xylazine, whereas a detomidine-

dependent decrease in myocardial contractility may play an additional negative role on cardiac output. Transient hypertension has been reported with the use of both xylazine and detomidine.⁴⁶ This effect may be attributed to positive peripheral adrenergic effect of α -agonists and is demonstrated by a consistent increase in systemic vascular resistance. Vagal activity follows the initial hypertensive effect and causes bradycardia, promoting decreased cardiac output and hypotension.

Respiratory effects can be summarized as mild hypoxemia and bronchodilation.⁴⁹ Respiratory frequency decreases with variable periods of apnea observed shortly after administration. PaO_2 is transiently decreased and, in studies using xylazine, was correlated to an increase in PaCO_2 , indicating that hypoventilation contributes to hypoxemia.⁴⁷ Other reports did not find an increased PaCO_2 , indicating that any hypoxemia is due to ventilation/perfusion mismatching because cardiac output is reduced and/or because pulmonary vascular resistance is increased. Other reported effects of detomidine are a potent diuretic effect and decreased bowel motility.⁴⁹

Thoracoscopic surgical equipment

The endoscopic equipment used for thoracoscopy in the horse is the same as that required to perform laparoscopy. Endoscopes most commonly used in the horse are 58-cm-long, 10-mm-diameter rigid telescopes (30° Hopkins telescope) although telescopes 30 cm in length have been employed to inspect the thorax. The telescope is attached to a videocamera and light source. The videocamera is a standard endoscopic camera used for arthroscopy and laparoscopy. A 300-watt xenon light source is recommended. The telescope is inserted through the chest wall via a trocar/cannula system, which can be disposable or non-disposable and of variable length. As an endoscopic portal an 11-mm,

15-cm-long cannula with a sharp trocar is generally used. Importantly, the cannula must be equipped with a side stopcock to allow the attachment of tubing for insufflation or suction. Insufflation is not usually needed for thoracoscopy; however, a CO₂ laparoflator may be employed in foals, which have a more compliant chest wall, and also during thoracoscopy performed under general anesthesia. A suction unit is mandatory in order to re-establish the normal pleural negative pressure following the completion of thoracoscopy. Additional trocar/cannula systems of varying length and size may be used to introduce accessory instrumentation in the thorax or to change the insertion point of the telescope. Additional equipment would include 5-mm reducers to allow 11-mm cannulas to harbor 5-mm-diameter instruments (graduated probes, biopsy forceps) and non-conductive anchoring devices that, placed around the cannula, provide stabilization of the system during instrument removal from the thoracic cavity.

Objectives

The literature reviewed in this chapter identifies the principles, the applications, and the advantages of thoracoscopy. This procedure has been applied in veterinary medicine in the equine surgical field and the clinical usefulness of thoracoscopy in horses has been proven. We felt that thoracoscopy in the horse needed to be further supported by investigating the pathophysiological consequences of the procedure. Questions pertaining to the safety of the procedure both intra-operatively and in the long term were posed in the design of this study. Although the surgical principals behind thoracoscopy are readily understood, the surgeon is faced with the challenging intra-operative presence of pneumothorax. In order to investigate the physiological adaptations of horses to the

pneumothorax condition, healthy horses were selected in this study, thus avoiding the confounding influence of disease.

Surgical techniques involving the thorax will affect the cardiovascular and respiratory systems. During equine thoracoscopy, these systems will be affected by factors such as pharmacological restraint, surgical stimulation, and pneumothorax. These factors need to be addressed in order to determine the safety of the procedure and are here analyzed by evaluating cardiopulmonary parameters.

The overall goal of the research described in this thesis was to evaluate the physiological responses of normal healthy horses to thoracoscopy. This intent generated four main objectives: 1) to define the changes in cardiopulmonary function of healthy, awake, and pharmacologically restrained horses during and after exploratory thoracoscopy; 2) to determine the presence of post-operative complications 48 hours after thoracoscopy; 3) to report the normal anatomy viewed during thoracoscopy performed in healthy standing horses; and 4) to describe the surgical technique used.

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CHAPTER 2

PLEUROPULMONARY AND CARDIOVASCULAR CONSEQUENCES OF THORACOSCOPY PERFORMED IN HEALTHY STANDING HORSES

Summary

Six healthy, awake, and pharmacologically restrained adult horses were studied in order to define the changes in cardiopulmonary function during and after exploratory thoracoscopy and to determine the presence of post-operative complications occurring 48 hours after thoracoscopy.

In a randomized 3x3 latin square design with three replications, 18 procedures were performed: 6 right (RTH) and 6 left thoracoscopies (LTH) and 6 sham procedures (STH). Prior to each procedure a physical exam and a bronchoalveolar lavage fluid analysis were performed. During thoracoscopy and sham protocols, horses were sedated with a continual drip of detomidine HCl and data were collected at six time intervals: T1 (baseline), T2 (10 minutes of detomidine administration), T3 (first 15 minutes of pneumothorax), T4 (5 minutes of recovery from pneumothorax), T5 (second 15 minutes of pneumothorax), and T6 (10 minutes of recovery from the second pneumothorax and detomidine). An endoscopic thoracic examination was conducted during the two pneumothorax periods. An identical protocol was followed for sham procedures without surgery or pneumothorax. Data were analyzed by ANOVA with time and surgical procedure as main factors. Physical examinations, thoracic radiography and ultrasound,

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CBC, and bronchoalveolar lavage fluid analysis were performed 48 hours after thoracoscopy.

Heart rate, respiratory rate, and cardiac output decreased following detomidine administration. There was a trend for cardiac output to be lower during thoracoscopy. Mild systemic hypertension was associated with thoracoscopy, although there was no effect on pulmonary arterial pressure. Total and pulmonary vascular resistances were increased following detomidine administration. Thoracoscopy caused a further increase in systemic and pulmonary vascular resistances especially during pneumothorax II. Arterial O₂ tension decreased following detomidine administration and was further decreased during the second pneumothorax period. PaO₂ values were lower when thoracoscopy was performed on the left rather than the right hemithorax. No significant complications were found during the 48-hour follow-up evaluation. However, in two horses, a sub-clinical post-operative pneumothorax was detected upon radiographic examination.

Thoracoscopy performed in healthy, awake, and pharmacologically restrained horses did not have detrimental cardiopulmonary effects and did not cause postoperative complications within the first 48-hour period.

Thoracoscopy is a minimally invasive endoscopic surgical procedure that allows video-assisted examination of the thoracic cavity.¹ The procedure is carried out by inserting a rigid telescope and additional operative instrumentation through the intercostal spaces. A pneumothorax is induced after perforation of the thoracic wall and the thoracic anatomical structures are observed after collapse of the ipsilateral lung.

In people, thoracoscopy was used during the 1930s and 1940s to break down intrapleural adhesions as part of the therapeutic measures for tuberculosis.² The advent

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of video-assisted technology in the early 1990s promoted the development of video-assisted thoracic surgery (VATS).³ In the standing horse, thoracic procedures have been performed effectively with the combination of sedation/analgesia, local anesthesia, and physical restraint.^{4,5} The veterinary literature has reported diagnosis of individual cases of thoracic infectious and neoplastic disease by endoscopy using both flexible⁶ and rigid telescopes.⁷ More recently, the evaluation and treatment of pleuritis, pleuropneumonia, and neoplasia by means of an 85-cm rigid telescope and video-assistance has been described in a series of horses.⁸

Pneumothorax is a necessary feature of equine thoracoscopy. By collapsing the lung, the surgeon can access the thoracic structures. It was our goal to study the cardiopulmonary consequences of pneumothorax combined with this surgical technique and pharmacological restraint. Two hypotheses were tested in this study. First, that thoracoscopy performed in healthy, awake, and pharmacologically restrained horses does not significantly affect cardiopulmonary function; second, that thoracoscopy does not have adverse consequences to the lung and pleural space within a 48-hour post-operative period.

Materials and methods

Horses and treatments

Six horses (three geldings and three mares ranging from three to ten years of age and weighing between 440 and 560 kg) were used to study the cardiopulmonary effects of thoracoscopy. The study was approved by the All-University Animal Care and Use Committee at Michigan State University. Eighteen procedures were performed: six left (LTH) and six right (RTH) hemithoracic examinations and six sham procedures (STH).

Using a latin square design with three replications, each horse underwent the three treatments in a randomized sequence. A minimum of thirty days was allowed between each thoracoscopy.

Pharmacological restraint

Detomidine HCl^a was used to provide sedation and analgesia during all procedures. Detomidine was administered as an intravenous bolus (6 $\mu\text{g}/\text{kg}$) followed by a continuous intravenous drip (0.8 $\mu\text{g}/\text{kg}/\text{min}$).

Measurements of cardiopulmonary function

Mean systemic arterial pressure, pulmonary arterial pressure, and blood temperature were recorded by means of a physiologic data collection system^b connected to a computer that allowed the on-screen monitoring and storage of data for subsequent recall. Pressure transducers were calibrated against a mercury manometer and placed at the level of the point of the elbow. In preparation for the experiments, horses were restrained in stocks, and catheters placed with sterile technique. A 14-gauge, 5.25-in. catheter^c was placed in the right jugular vein and used for the administration of detomidine for sedation and analgesia.

Systemic arterial pressure and arterial blood sampling

A 16-gauge, 2-in. catheter^c was introduced into a surgically exteriorized left carotid artery in a distal to proximal direction. This allowed continuous measurement of arterial pressure and collection of samples for arterial blood gas analysis. At each measurement period, mean arterial pressure was recorded and 3 mls of arterial blood

were drawn and stored in heparinized syringes. Arterial samples were used for blood gas analysis. pH, PaO₂, and PaCO₂ were measured using a Stat Profile Plus 9 blood gas and electrolyte analyzer,^d which underwent regular auto-calibration and was also calibrated prior to sample testing.

Pulmonary arterial pressure

A balloon-tipped 7 French, 110-cm Swan-Ganz catheter with a thermistor located near the tip was connected to a pressure transducer and floated into the pulmonary artery for continuous measurement of pulmonary arterial pressure.

Cardiac output and stroke volume

Cardiac output was measured with a thermodilution technique using a Cardiomax II (model 85) cardiac output computer.^e The computer displayed cardiac output and stroke volume and allowed verification of injectate temperature and core body temperature. Two sterile catheters were placed in the left jugular vein. A polyethylene 240 catheter^f was used to deliver the injectate to the right atrium and the thermistor/balloon-tipped 7 French 110 cm Swan-Ganz catheter placed in the pulmonary artery was used to record blood temperature. Both catheters were placed via 10-gauge venous introducers and their position was verified by observing pressure traces. Cardiac output was determined by thermodilution. Rapid manual injection of 30 mls of ice cold 5% dextrose was made into the right atrium. The dilution curves for each cardiac output measurement were displayed on the computer monitor. At each measurement period, cardiac output was determined as the mean of three consecutively collected values. Other measured variables were: respiratory rate, heart rate, and systemic and pulmonary

vascular resistances, which were calculated by dividing mean arterial pressure and pulmonary arterial pressure, respectively, by cardiac output.

Thoracoscopy

In preparation for surgery, horses were weighed and treated with flunixin meglumine^g (1 mg/kg BID) and procaine penicillin^h (20,000 iu/kg BID). Antibiotic and anti-inflammatory treatments were discontinued 48 hours postoperatively. Horses were restrained in stocks and instrumented for data collection. The hair of the operated hemithorax was clipped over an area extending from the caudal aspect of the shoulder region to the last rib and from the dorsal thoracic region to the ventral thorax at the level of the elbow joint. The area was aseptically prepared for surgery. Local anesthetic was infused subcutaneously into the site for placement of the endoscopic portal, which was located either at the 8th, 10th, or 12th intercostal space just ventral to the serratus dorsalis muscle. A pneumothorax was created by making a 2-cm skin and subcutaneous tissue incision and inserting a stainless steel, blunt cannula into the pleural space. Following removal of the cannula, a sharp 10-mm diameter trocar/cannula systemⁱ was inserted through the skin incision into the thoracic cavity and served as the endoscopic portal. A 30-degree 85-cm long and 10-mm diameter rigid telescopeⁱ connected to a videocamera and light sourceⁱ was used to explore the hemithorax through the cannula. The examination was carried out for 15 minutes. Proceeding from the caudo-dorsal to the most cranial region of the thorax, the exam was completed by inspecting the pulmonary surface along the ventro-lateral thoracic wall. After the 15-minute examination, the pneumothorax was interrupted by applying suction to the pleural space via sterile tubing connected to the endoscopic portal system. This re-established the

negative pleural pressure. While suction was applied to the pleural space the endoscope was left in the thorax to observe complete lung inflation and was then removed. Complete re-inflation occurred in approximately 15–20 seconds. The horse was allowed to rest for 5 minutes and then a second pneumothorax period was started by opening the trocar/cannula system. In addition, an accessory portal was created either at the 8th or 11th intercostal space about 10 cm distal to the endoscopic portal. Under direct endoscopic observation a second 10 mm sharp trocar/cannula system coupled with a 5-mm reducerⁱ was inserted into the thorax after incising skin and subcutaneous tissues. Through this secondary portal, a 50-cm long, 5-mm diameter blunt probeⁱ was passed into the thorax. The probe allowed assessment of the distance of the thoracic organs from the chest wall, areas of potential surgical access, and tissue palpation. The second pneumothorax also lasted 15 minutes and was then reversed with suction until complete lung inflation was observed. The procedure ended by removing the cannulas of the accessory portal first and then the endoscopic portal. Skin incisions were apposed with a simple interrupted pattern using 0 prolene^k suture material for both endoscopic and accessory portals.

Sham procedures

The same data collection protocol used for right and left thoracoscopy was followed during the sham procedures except that no surgical interventions were carried out.

Experimental design

Phase one—On day one, a pre-operative evaluation was performed two hours before all treatments. Physical examination included the assessment of mucous membrane color and capillary refill time, heart and respiratory rates, and rectal temperature. Bronchoalveolar lavage fluid (BALF) was collected using the following technique. With the horse restrained in stocks, a 3-meter video-endoscope was passed via the nose and wedged in a peripheral bronchus. Six 50-ml aliquots of phosphate-buffered saline (PBS) were infused into the tube and recovered by suction. The sampling site depended on which hemithorax was to be operated on. When LTH or RTH were scheduled, the endoscope was advanced in the opposite airway and BALF collected. Before performing STH, either the left or the right distal airway was sampled following a random order. Total cell counts in BALF were performed manually using a hemacytometer. Cell smears were made with a cytocentrifuge at 350 G for 10 min and stained with Diff-Quick. Differential cell counts for neutrophils, macrophages, lymphocytes, eosinophils, and mast cells were done by counting 200 cells per sample.

Phase two—Phase one was completed in one hour and then horses were prepared for the experimental trials. Administration of detomidine, initiation of pneumothorax and recovery periods, and collection of data were done at specific times (Figure 1). Over a total experimental time of 55 minutes, data were collected at six time intervals. At baseline (T1) all variables were measured before both detomidine administration and surgical intervention. Immediately after collecting T1 data, detomidine was administered as a bolus and the intravenous infusion was started. The second set of values was obtained 10 minutes later (T2). These values demonstrated the acute effects of detomidine administration. Following T2 data collection, the horses underwent the first

15-minute thoracoscopy (RTH and LTH) or sham (STH) procedure. At the end of 15 minutes and before applying suction to the pleural space, data were recorded (pneumothorax I/sham, T3). At this point negative pleural pressure was re-established and horses were left to recover for 5 minutes. At the end of the 5-minute recovery period data were recorded (recovery I, T4). Following T4, horses underwent a second 15-minute thoracoscopy. When 15 minutes of surgery time had elapsed and before applying suction to the pleural space, data were recorded (pneumothorax II/sham, T5). Negative pleural pressure was re-established and detomidine administration was discontinued. The final set of data were collected after 10 minutes of recovery (recovery II, T6).

Phase three—Horses were rested in a stall in the Large Animal Clinic for two days after the procedures. On day 3 (48 hours after LTH, RTH, or STH) horses were evaluated for post-operative complications. When thoracoscopy had been performed, a venous blood sample was collected to evaluate complete blood cell count and fibrinogen concentration. Thoracic radiographs were taken to determine the presence of residual air within the pleural space. Digital radiography (640 ma, 90 KVP) was used with a phototimer to perform the radiographic study.¹ A thoracic ultrasound exam was performed bilaterally using a 3.5 mHz curvilinear probe^m to detect pleural effusion. Bronchoalveolar lavage fluid was collected from the distal airway corresponding to the operated hemithorax. After the sham procedure, BALF was collected from the airway opposite that used before the sham.

Statistical methods

A factorial analysis was performed to determine the main effects of time and procedures. When significant ($p < 0.05$) main effects were observed, differences between means were evaluated by use of a one-way ANOVA, and post-hoc comparisons were made using Tukey's highly significant difference test. The analysis was performed first with right and left thoracoscopic procedures maintained as separate treatments. Except for PaO_2 , no differences were found between RTH and LTH and the analysis was then performed considering RTH and LTH combined. A factorial analysis was done to investigate the relationship between preoperative and postoperative CBC and fibrinogen values and BALF analysis results. Statistical analysis was performed using a computer software program.⁸ All values in figures and text are expressed as mean $\pm$ standard deviation.

Measurement of pleural pressure

One of the questions that arose during the design of the experimental methods concerned the issue of pleural pressure. Most anatomical descriptions of the equine thoracic cavity usually include a statement regarding the lack of continuity of the mediastinum.⁹ The cranial mediastinum is thought to be perforated in its most ventral fold distal to the cranial vena cava.

This raised questions about the pneumothorax induced during thoracoscopy. Was the pneumothorax going to remain confined to the operated hemithorax? Was the opposite lung going to collapse and if so to what degree? Were horses going to be able to tolerate a possible bilateral pneumothorax? These issues led to the measurement of pleural pressure in the non-operated hemithorax.

Methods

A 14-GA 5.25-inch catheter was used to measure pleural pressure. The catheter was inserted into the pleural cavity at the middle third of the 8th intercostal space. The catheter was connected to a pressure transducer, and inspiratory and expiratory efforts were recorded. Change in pleural pressure during tidal breathing ($\Delta P_{pl_{max}}$) was calculated as the difference between maximal inspiratory and expiratory pressures. When LTH or RTH were performed, pleural pressure was measured in the hemithorax opposite to the operated side. During STH, the site of measurement of pleural pressure (right or left hemithorax) was randomized among horses.

Findings

Pleural pressure was increased by detomidine administration. Intra-operative bilateral pneumothorax did not occur. Changes in $\Delta P_{pl_{max}}$ were not different whether horses underwent thoracoscopy or sham procedures. In addition, during all thoracoscopic procedures the dorsal mediastinum was clearly visible once the lung was collapsed. The contralateral lung could always be seen in complete inflation through the mediastinum for the duration of both pneumothorax periods. The increased respiratory efforts could have been due to upper respiratory obstruction due to muscle relaxation caused by detomidine within the nasal passages. This could be partially responsible for the increased respiratory efforts along with the fact that, while sedated, horses maintained a five-point stance with the head in a lowered position, thus increasing the chance for formation of nasal edema.

Results

Thoracoscopy was successfully and safely completed in all horses. Occasional signs of discomfort depended on the position of the endoscopic portal. Directing the rigid telescope cranially elicited signs of discomfort when the more anterior approach to the thorax was used (8th intercostal space). Despite sedation and analgesia, horses became more alert and more reactive when this approach was used. Horses rapidly became comfortable and appeared sedated when cranial telescope movements were interrupted. A more posterior approach (12th intercostal space) was not associated with signs of pain even with marked cranial advancement of the telescope. The thoracic anatomy could be thoroughly inspected following lung collapse. The contralateral lung was visible through the dorsal mediastinal membrane and remained inflated throughout the procedure. Bilateral pneumothorax never occurred intra-operatively. Significant hemodynamic changes occurred over time and in association with the second pneumothorax period (T5). With the exception of PaO_2 there were no differences between RTH and LTH, therefore the data generated from all the other variables have been combined and analyzed as sham versus thoracoscopy.

Cardiovascular function

When compared to baseline (T1), heart rate (T2 through T5) and cardiac output (T2 through T6) decreased over time, whereas stroke volume did not change (Figure 2). An irregular heart rate was observed for several minutes following T2. With respect to baseline, mean systemic and pulmonary arterial pressures did not change over time, whereas systemic and pulmonary vascular resistances increased at T2, T3, and T5 (Figure 3). Significant differences between sham procedures and thoracoscopy were



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found primarily during the second pneumothorax (T5): cardiac output was lower, whereas mean arterial pressure and systemic and pulmonary vascular resistances were higher at the end of the second pneumothorax period (Figures 2–3). Additional differences between sham and surgical procedures were found at other time intervals. In horses undergoing thoracoscopy, cardiac output was lower at T2 and systemic vascular resistance was higher at T4 and T6.

Respiratory function

PaCO₂ and pH did not change over time as a result of the procedures (Figure 4). Respiratory rate had a tendency to decrease following T2: mean respiratory rate ($\pm$ SD) at T1 combined for all procedures was 28 ($\pm$ 4) breaths/minute and was 14 ($\pm$ 5) following T2. Arterial O₂ tension changed over time and was significantly lower than baseline at T3. Differences between sham and thoracoscopy were associated with the second pneumothorax period (T5). At T5 left thoracoscopy had a worse effect on PaO₂ than right thoracoscopy (Figure 4).

48-hour follow-up evaluation

No abnormalities were found on physical exams performed following thoracoscopy. Pre- and post-thoracoscopy CBC and fibrinogen concentrations values did not differ (Table 1). There were no significant effects on total and differential cell counts in BALF. This was found both when sham procedures were compared to thoracoscopy and when pre-and post-thoracoscopy results were compared (Table 2). Residual post-operative pneumothorax was detected in two horses on follow-up thoracic radiographic evaluation. Both horses were hospitalized for further observation and

recovered from the pneumothorax without further intervention and without complications. Pleural fluid or effusions were not detected after thoracoscopy.

Discussion

The thorax of the standing, sedated horse was safely explored via thoracoscopy using an 85-cm long rigid telescope and video-assistance. The normal anatomical structures could be observed in the right and left hemithoraces when horses were restrained in stocks and controlled by administering the sedative analgesic detomidine. Although the use of flexible and rigid endoscopes has been reported in the veterinary literature for the diagnosis and treatment of thoracic disease,⁶⁻⁸ it was our intent to study the effects of thoracoscopy without the complicating factors associated with pleuropulmonary pathology. Two main factors required attention in the analysis of the cardio-pulmonary consequences of the surgical procedure: detomidine administration (causing the changes observed over time) and pneumothorax. In this context, sham procedures were an important part of the study design, allowing us to isolate the effects of the sedation/analgesia protocol from the effects of surgery. Detomidine was an efficacious method of sedation and analgesia for thoracoscopy; however, it was responsible for most of the cardiovascular and respiratory changes observed.

An irregular heart rate was recorded following administration of a detomidine bolus. Atrio-ventricular conduction abnormalities (II degree A-V blockade) have been previously reported to be associated with α_2 -agonist administration.¹⁰ Detomidine caused heart rate to decrease. A lower heart rate led to a decrease in cardiac output. The negative effect of detomidine was maintained on cardiac output beyond T5 when detomidine was discontinued. This prolonged effect occurred despite heart rate returning

to normal values following T5 (Figure 2). No changes in stroke volume were recorded. The longer-lasting depressant effects of detomidine have mainly been attributed to a decrease in central sympathetic outflow, although a direct negative chronotropic effect on the heart and a decrease in myocardial contractility have also been postulated to play a role in the cardiovascular depression caused by α_2 -agonists.¹⁰ Despite the decrease in cardiac output, systemic and pulmonary arterial pressures did not change in our horses because of a long-lasting increase in systemic and pulmonary vascular resistances (Figure 3).

Respiratory changes associated with detomidine were a decreased respiratory rate and a slight decrease in PaO₂ (Figure 4). Arterial oxygen tension decreased transiently 25 minutes after detomidine administration and then normalized toward the end of the experiments. The decreased PaO₂ could be related to an altered ventilation/perfusion relationship following the decrease in cardiac output and the increase in pulmonary vascular resistance associated with the peripheral effects of detomidine. Adequate ventilation, as demonstrated by the constant PaCO₂, was maintained throughout the experiments (Figure 4).

The cardiopulmonary changes associated with thoracoscopy occurred at the second pneumothorax period. Pneumothorax II exacerbated the changes seen with detomidine alone (sham) with regard to decreased cardiac output, increased systemic and pulmonary vascular resistances, systemic hypertension, and hypoxemia (Figures 2, 3, 4). The loss of negative pleural pressure and lung collapse likely reduced venous return to the heart during pneumothorax and decreased cardiac output more than with detomidine alone (Figure 2). During pneumothorax, the cranial slope of the diaphragm caused the collapsed lung to be located in the most dependent and anterior region of the chest.

Cardiac function could also have been hindered by the pressure exerted by the pulmonary mass on the heart base and related vascular structures.

Systemic vascular resistance increased with thoracoscopy and contributed to the higher mean arterial pressure associated with the second pneumothorax period (T5). These changes were likely a result of greater surgical stimulus present during the second period of thoracoscopy as they were not found following pneumothorax I. The insertion of an accessory portal and manipulation of the thoracic organs may have released catecholamines causing peripheral vasoconstriction and hypertension. The presence of a secondary portal could have led to a greater degree of pneumothorax during the second thoracoscopy phase. Pronounced lung collapse could explain the increase in pulmonary vascular resistance seen during pneumothorax II and not during pneumothorax I (Figure 3).

In other species, such as humans and dogs, reported consequences of pneumothorax have been decreased arterial O_2 tension and decreased vital capacity.¹¹ In people airway closure has been demonstrated at low lung volumes during pneumothorax and has been advocated as a primary cause of hypoxemia and poor ventilation.¹² Horses did not hypoventilate during thoracoscopy; $PaCO_2$ was unchanged throughout the procedures (Figure 4). For this reason, the reduction in arterial O_2 tension during the second pneumothorax was due to ventilation/perfusion mismatching as a result of lung collapse. When undergoing a prolonged thoracoscopic exploration, horses may require regular phases of recuperation between pneumothorax periods to allow re-establishment of ventilation/perfusion matching. This can be easily achieved by re-inflating the lung with suction. In addition, increasing the fraction of inspired O_2 by tracheal insufflation of 100% O_2 may be beneficial.

PaO₂ was the only variable that differed when right and left thoracoscopy were compared (Figure 4). Lower values of PaO₂ were found at the end of the second pneumothorax period during left hemithoracoscopy. Anatomical characteristics of the horse may explain these differences. Important vascular structures emerge from the heart base and are located at first within the left hemithorax: the aortic arch, which leads cranially to the brachiocephalic trunk and caudally to the thoracic aorta and the truncus arteriosus from which the left and right pulmonary arteries originate.¹³ It is therefore plausible that pulmonary perfusion problems during lung collapse may be worse when the procedure involves the left thorax. Lung size differences would not seem to sufficiently explain the lower PaO₂ found with left pneumothorax. The right lung is larger than the left in the horse and therefore ventilation/perfusion mismatching would be expected to be worse with a right-side pneumothorax.

All horses recovered without clinically obvious consequences from the procedures and had normal physical examinations and hematological values when the post-operative evaluation was performed. No pleural fluid or effusions were detected on thoracic ultrasound, indicating minimal trauma to the intercostal muscles and pleura caused by portal placement and surgical manipulation. Upon radiographic examination of the thorax, a residual post-operative pneumothorax was found in two cases. In both horses a left hemithoracoscopy had been performed. The degree of pneumothorax was negligible in one case and may have resulted from insufficient lung inflation at the end of pneumothorax II. The second post-operative pneumothorax was more radiographically obvious and was associated with perforation of the lung parenchyma, which occurred during the insertion of the sharp trocar/cannula for the endoscopic portal. The horse was observed for 7 more days following the 48-hour evaluation, developed no clinical signs,

and was then released from the Large Animal Clinic. Lung perforation can be avoided by entering the chest cavity with a small, blunt cannula and allowing enough time for the lung to collapse below the entry point of the trocar. In most cases, one minute was sufficient to achieve a safe pneumothorax for trocar placement.

Bronchoalveolar lavage fluid analysis was performed to identify signs of inflammation of the lower airway. Thoracoscopy did not produce lower airway inflammation. However, the lowest PaO₂ values were recorded from the horse that preoperatively demonstrated the most significant signs of lower airway inflammation upon BALF analysis. This finding may support the use of tracheal insufflation of oxygen when operating on a diseased thorax. The presence of pleuropulmonary disease may alter the response of the horse to the procedure. Importantly, the duration of the pneumothorax periods should be regulated depending on the clinical status of the horse, and lung function should be frequently assessed by blood gas evaluation.

Thoracoscopy was safely performed in the healthy horse and can be recommended for the exploration of the thorax. Detomidine, administered continually throughout thoracoscopy, provided good pharmacological restraint and has been recommended for prolonged procedures in the standing horse.¹⁴ When administering detomidine, careful monitoring of the hemodynamic condition of the horse is mandatory since most of the cardiopulmonary changes recorded during thoracoscopy were associated with detomidine administration. Lung collapse was well tolerated despite a slight decrease in PaO₂ that occurred during a second pneumothorax period. The degree and the duration of the pneumothorax can be readily regulated with the use of suction and therefore thoracoscopy can be easily adapted to the condition of the horse.

Manufacturers' addresses

^aDormosedan, Pfizer Animal Health, Exton, Pennsylvania, USA.

^bColburn Instruments (model L19-69), Allentown, Pennsylvania, USA.

^cAngiocath, Becton Dickinson, Sandy, Utah, USA.

^dNova Biomedical, Waltham, Massachusetts, USA.

^eColumbus Instruments, Columbus, Ohio, USA.

^fBaxter Pharmaceuticals, Valencia, California, USA.

^gBanamine, Schering Plough, Kenilworth, New Jersey, USA.

^hButler, Dublin, Ohio, USA.

ⁱStorz Veterinary Endoscopy, Goleta, California, USA.

^jStryker Endoscopy, Santa Clara, California, USA.

^kEthicon, Somerville, New Jersey, USA.

^lGE Advantex, GE Medical Systems, Milwaukee, Wisconsin, USA.

^mFlexus model SD 1100-V Aloka, Tokyo, Japan.

ⁿSPSS Graduate Pack, Version 7.0 for Windows, Chicago, Illinois, USA.

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Table 1: Total and differential whiteblood cell counts and fibrinogen concentration before (PREOP) and 48 hours after (POSTOP) thoracoscopy. Data are mean values + SD.

	RIGHT	RIGHT	LEFT	LEFT
	PREOP	POSTOP	PREOP	POSTOP
WBC (x10 ³ /ul)	7.1±1.89	6.82±0.95	6.87±1.62	8.01±1.63
SEG%	54±8.2	62±3.4	81±7.5	76±4.8
LYMPH%	42±2.4	34±1.8	32±2.8	30±0.9
MONO%	2±0.02	4±0.01	6±0.1	4±0.05
FIB (gm/dl)	0.18±0.07	0.18±0.05	0.18±0.07	0.18±0.13

Table 2: Total and differential cell counts in BALF before (PREOP) and 48 hours after (POSTOP) right and left thoracoscopy and before (PRE) and 48 hours after (POST) sham procedures. Data are mean values + SD.

	RIGHT		LEFT		SHAM	
	PREOP	POSTOP	PREOP	POSTOP	PRE	POST
TOTAL CELL						
COUNT (x10 ³ /ml)	14.5±12	9.2±8	8.4±3.6	12±5.6	4.5±0.8	10.1±5.6
PAM%	47.8±8.4	53.3±16.5	69.6±13.2	51±26.9	55.4±3.7	46.8±17.4
PMN%	1.9±2.11	1.8±2.64	1.8±1.72	6.8±12.88	7.7±7.76	10.8±13.12
LYMPH%	46.2±6.25	42.7±13	26.3±9.34	40.8±23	35.1±9.01	38.1±11.39

Figure 1: Data collection protocol indicating times and sequence of events.

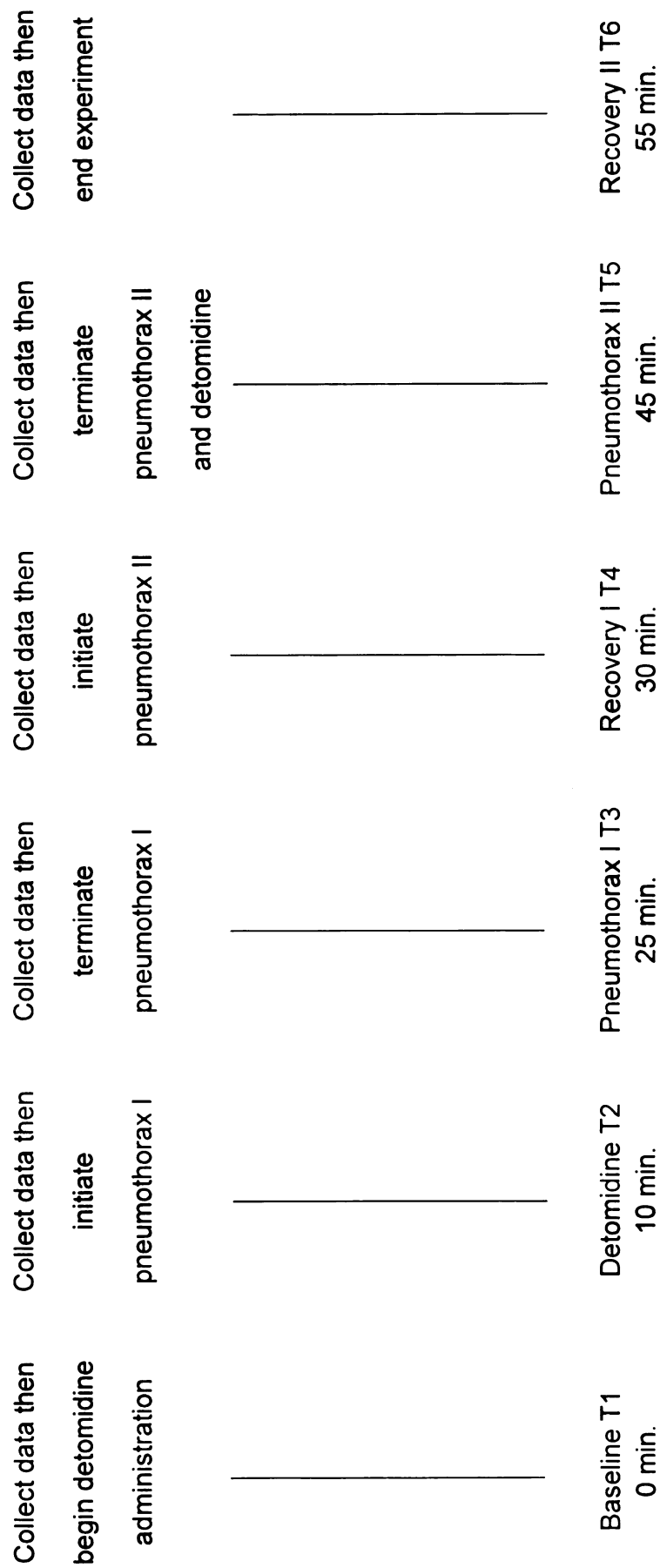


Figure 2: Effects of detomidine and thoracoscopy on heart rate, cardiac output and stroke volume. Data are expressed as mean ($\pm$ SD); * = significantly different ($p < 0.05$) from baseline; ‡ = effect of thoracoscopy significantly different ($p < 0.05$) from **sham** at the indicated time period; solid bars = sham procedures; gray bars = thoracoscopy).

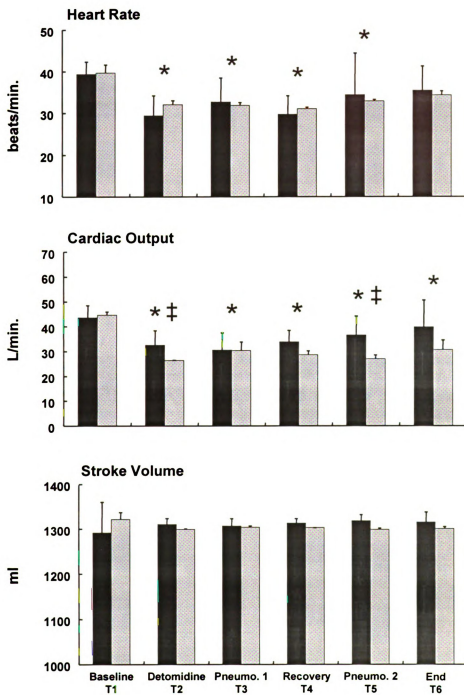


Figure 2



Figure 3: Effects of detomidine and thoracoscopy on mean arterial pressure, systemic vascular resistance, pulmonary arterial pressure and pulmonary vascular resistance. Data are expressed as mean ($\pm$ SD); * = significantly different ($p < 0.05$) from baseline; ‡ = effect of thoracoscopy significantly different ($p < 0.05$) from sham at the indicated time period; solid bars = sham procedures; gray bars = thoracoscopy).

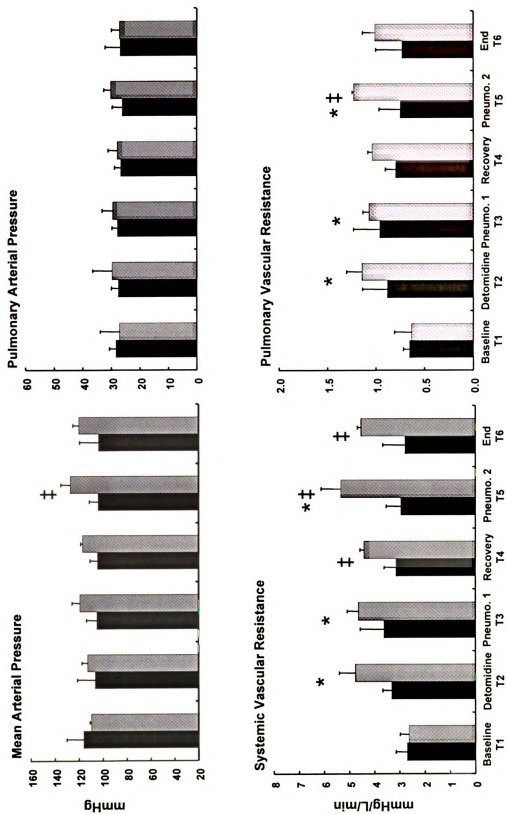


Figure 3

Figure 4: Effects of detomidine and thoracoscopy on pH, PaCO₂ and PaO₂. Data are expressed as mean ($\pm$ SD); * = significantly different ($p < 0.05$) from baseline; ‡ = effect of thoracoscopy significantly different ($p < 0.05$) from sham at the specific time period; † = LTH significantly different from RTH at the specific time period; solid bars = sham procedures; gray bars = thoracoscopy; clear bars = left thoracoscopy; diagonal cross hatch bars = right thoracoscopy).

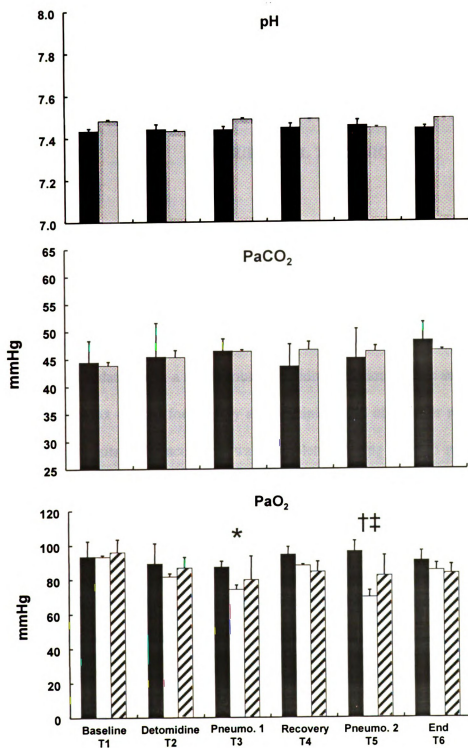


Figure 4

CHAPTER 3

EQUINE THORACOSCOPY: NORMAL ANATOMY AND SURGICAL TECHNIQUE

Summary

Six normal, healthy horses ranging in age from 3 to 10 years and weighing from 440 to 560 kg underwent left and right thoracoscopic examination using a rigid telescope. A minimum of thirty days was allowed between procedures. Horses were restrained in stocks and sedated with a continuous detomidine infusion. After surgical preparation of the hemithorax elected for surgery and administration of local or regional anesthesia of the surgery sites, thoracoscopy was completed during two 15-minute pneumothorax periods. During the procedures the thoracic structures were viewed using a 58-cm, 10-mm diameter, rigid telescope, and digital video-recording was done for subsequent recall. The exploration of each hemithorax started from the dorsal-caudal quadrant, continued toward the cranial thorax, and was completed by observing the diaphragmatic and caudal pulmonary region. The telescope was inserted in the thoracic cavity via three different intercostal spaces. The 8th, 10th, and 12th intercostal spaces were randomly selected and used among horses.

Collapsed lung, aorta, esophagus, and diaphragm were readily viewed in either hemithorax. On exploration of the right hemithorax, the azygos vein, the thoracic duct, and pulmonary veins were also identified. Horses tolerated thoracoscopy well. Signs

of discomfort such as increased respiratory rate, coughing, and decreased level of sedation, were associated with acute lung collapse in one horse, with pneumothorax in two occasions, and when the thorax was approached through the 8th intercostal space. Surgery performed via the 8th intercostal space was hindered by the rigidity of the 8th and 9th rib, which did not allow easy cranial and caudal movements of the telescope. Suction applied to the thorax allowed prompt regulation of the degree of pneumothorax and a more gradual collapse of the lung.

This paper was part of a larger study, performed in healthy standing horses, aimed to verify the safety of thoracoscopy by evaluating the cardiopulmonary changes associated with the procedure.

We and others have demonstrated that the equine thorax can be safely and successfully examined with rigid and flexible endoscopes.^{1,2,3} This examination technique, known as thoracoscopy, has been used to diagnose and treat pleuropulmonary disease in the horse.⁴ In people, thoracoscopy carries the advantages of a shortened hospital stay, earlier return to function, and less intra- and post-operative pain when compared to thoracotomy.⁵ Small skin incisions coupled with specially designed endoscopic equipment allow observation of intra-thoracic structures in a “minimally invasive” fashion. Direct examination offers the advantage of the close inspection of normal and diseased structures. In certain conditions, thoracoscopy complements traditional diagnostic methods such as clinical evaluation, ultrasonography, radiography, and endoscopy.⁶ The purpose of this report is to illustrate the normal anatomy viewed during thoracoscopy in healthy, standing horses. A detailed description of the surgical technique used is also provided.

Materials and methods

Horses

The study was approved by the All-University Committee on Anima Use and Care at Michigan State University and was part of a larger study aimed at defining the cardiopulmonary changes associated with thoracoscopy performed in healthy, standing horses. Six horses (three mares and three geldings) between the ages of three and ten years and weighing between 440 and 560 kg were used in a study aimed to determine the safety of the procedure.³ In these same animals we also defined the structures visible with the thoracoscope and described the surgical technique used. Each horse underwent a left and right hemithoracoscopy with a minimum 30-day interval between procedures.

Patient preparation

Before surgery, each horse was treated intravenously with flunixin meglumine^a (1 mg/kg twice a day) and intramuscularly with procaine penicillin G (20,000 IU/kg twice a day). Blood was drawn to evaluate complete white blood count and fibrinogen concentration. Horses were groomed and restrained in stocks, and the tail was wrapped with an elastic bandage to avoid contamination of the surgical field. A catheter^b was aseptically placed in the right jugular vein for the administration of the sedative-analgesic detomidine^c HCl. A single intravenous bolus (6 μ g/kg) was administered and followed by a continuous intravenous infusion (0.8 μ g/kg/min). A square area extending from just distal to the dorsal midline to the point of the elbow and from the caudal scapular margin to the 15th rib was clipped and aseptically prepared for surgery. A large disposable drape was used to cover the horse and a window was cut in the drape. Draping was secured

by means of an adhesive plastic sheet,^d which was attached to the drape and to the surgical site.

The endoscopic portal was placed just ventral to the serratus dorsalis muscle (epaxial muscle group). Either the 8th, 10th, or 12th intercostal space was randomly selected among horses. The accessory portal was located within the 8th or 11th intercostal space, approximately 10–15 cm below the endoscopic portal. Upon selection of the surgical sites, regional or local anesthesia was provided to the area. Four out of twelve procedures were performed under regional anesthesia. Local anesthetic was placed over the intercostal nerve that provides sensation to the intercostal space designated for surgery. In addition, sensation was abolished from one intercostal space cranial and one caudal to the surgical site. Local anesthetic^e (2% carbocaine) was placed subcutaneously in the proximal third of the selected rib. An 18 1½" needle was then directed toward the caudal border of each rib and used to deposit 8–12 ml of local anesthetic. An 18 g 3½" spinal needle was necessary to provide anesthesia for the 8th intercostal nerve. In the remainder of the cases (8/12), anesthesia of the surgery sites was achieved by infiltrating 8 ml of anesthetic locally into the subcutaneous, muscular, and pleural tissues.

Surgical technique

Following digital palpation of the intercostal space designated for the endoscopic portal, a #10 scalpel blade was used to create a 2–2.5 cm linear incision through the skin and subcutaneous tissues. Deeper blunt dissection of adipose tissue and external fascia was necessary when the endoscopic portal was placed through the 8th intercostal space. Once the external intercostal muscle was found, a blunt, stainless steel, teat cannula was passed through the intercostal muscles and parietal pleura to create a pneumothorax. Air

was heard passing through the cannula into the thoracic cavity during each respiratory cycle. The teat cannula was removed after three to four respiratory cycles. A 15-cm-long, 11-mm-diameter, non-disposable, unguarded cannula coupled with a sharp trocar^f was placed through the skin incision and directed caudally to avoid the intercostal neurovascular structures present at the posterior border of each rib. The instrument was advanced through the musculature and pleura using a gentle corkscrew motion. Following complete advancement of the cannula into the thoracic cavity, the trocar was removed. Suction tubing was attached via a coupling device to the side stop-cock present on the cannula. A 30-degree, 58-cm-long, 10-mm-diameter rigid telescope^f, attached to a video camera and a xenon light source cable, was passed through the cannula and used for the exploration. Thoracoscopy was performed for 15 minutes, during which time the thoracic structures were examined and digitally recorded on videotape for subsequent viewing. At the end of the examination, negative pleural pressure was re-established by applying suction to the pleural space. This was achieved by attaching a portable suction unit with sterile tubing to the insufflation stopcock present on the endoscopic cannula. During suction, complete re-inflation of the lung was observed by retracting the telescope within the distal end of the endoscopic cannula. The telescope was then removed.

The skin incision for the accessory portal was made during a five-minute recovery period from the first thoracoscopy. Before inserting the accessory portal, a pneumothorax was re-created by opening the trumpet valve within the endoscopic cannula. This was achieved by placing a narrow surgical instrument (forceps or hemostatic clamps) through the cannula. The telescope was introduced into the thorax and directed toward the expected intra-thoracic location of the accessory portal. A blunt stainless steel teat cannula was used to puncture the pleura at the sight of the accessory portal while under

endoscopic observation. Using a technique identical to that for the endoscopic portal, an 11-mm-diameter 15-cm-long trocar/cannula system, coupled with a 5-mm reducer,^f was advanced into the thorax. A 50-cm-long, 5-mm-diameter, blunt, graduated probe was passed through the secondary portal. A second 15-minute thoracoscopic examination was performed as structures were probed and moved. Upon completion of the examination, the accessory portal was removed as a unit. The lung was re-inflated by suction. The endoscopic portal was removed after observing complete lung inflation. The skin and subcutaneous tissues were closed using 0 prolene^g in a simple interrupted pattern.

Post-operative patient evaluation

Horses were returned to a stall in the Large Animal Clinic after recovering from detomidine and were monitored for 48 hours. Horses continued to receive flunixin meglumine (1 mg/kg twice a day) and procaine penicillin G (20,000 IU/kg twice a day) throughout this period.

Results

Thoracoscopy was successfully completed in all horses. The technique used allowed the exploration of each hemithorax, and horses tolerated the procedures well. The thoracic structures seen with thoracoscopy were best evaluated when the 10th and 12th intercostal spaces were used for endoscopic portal placement. The ribs associated with these spaces have less soft tissue and muscle coverage and are not as firmly attached to the sternum as the more cranial ribs are. The ribs associated with the caudal surgical approaches could, therefore, be distracted without damaging the surrounding anatomical

structures, without harming the horses, and without damaging the equipment. Free cranial and caudal movements of the rigid telescope were tolerated well by the horses when the more caudal approaches were used. The telescope could be advanced without complications into the cranial aspects of both hemithoraces (Figure 5a and b). By rotating the 30-degree lens in a cranial direction a panoramic view of the cranial thorax was obtained. Similar telescope movements could not be achieved when the 8th intercostal space was selected for the approach. When the 8th intercostal space was used, the telescope could be moved proximally and distally, but cranial and caudal motions were limited by the solid construct of the cranial thorax. Lateral movements of the telescope were physically hindered by the rigidity of the ribs associated with the 8th intercostal space.

Physical restraint and the sedation protocol were effective in maintaining the horses stable during the procedures while providing adequate sedation and analgesia. Horses showed signs of discomfort when the 8th intercostal space approach was used. Particularly when trying to angle the telescope to explore the cranial thorax, the force necessary to distract the ribs was a source of pain for the horses, despite the use of regional and local anesthesia. Horses demonstrated agitation, restlessness, and a decreased level of sedation. These signs were rapidly abolished when lateral telescope motions were interrupted.

Signs of distress (agitation, awakening from sedation) were demonstrated by one horse and were associated with lung collapse. The rapid induction of pneumothorax caused the horse to cough and become restless. The situation was corrected by applying suction to the hemithorax and allowing a more gradual pneumothorax to occur. Extreme lung collapse was the probable cause of agitation in two other horses. During the second

pneumothorax period, in these two cases, it was necessary to apply suction to reduce, but not eliminate, the pneumothorax and allow the horses to regain comfort and adequate sedation levels.

Bilateral pneumothorax never occurred during or after the procedure. Inadequate lung collapse led to pulmonary parenchymal perforation in one horse. All horses recovered uneventfully from thoracoscopy. Post-operative complications included mild subcutaneous emphysema associated with the surgical site (3/12 procedures), and subclinical pneumothorax (2/12 procedures).

The collapsed lung was readily visible when either left or right hemithoracoscopies were performed (Figures 5a and b). Depending on the degree of lung collapse, additional structures could be seen, such as the aorta, the esophagus, and the azygos vein in the right hemithorax. When a caudal approach was used (10th or 12th intercostal spaces), the collapsed lung was observed to be attached dorsally to the mediastinum by the pulmonary ligament (Figure 6a). The pulmonary ligament appeared as a thin, triangular-shaped fold of pleural tissue containing a tortuous branch of the broncho-esophageal artery, which coursed along the caudal margin of the lung. Above the pulmonary ligament the dorsal mediastinal membrane was seen, through which the opposite lung could be observed moving during the respiratory cycles. In all cases, the contralateral lung was observed in complete inflation through the thin dorsal mediastinum, indicating the absence of a bilateral pneumothorax.

Advancing the telescope cranially within the left hemithorax, over the collapsed lung and toward the mediastinum, the aorta, thoracic esophagus, and collapsed lung with the aortic impression were observed (Figure 6b). When the same region of the right hemithorax was in view, the same structures were found, with the addition of the azygos

vein, thoracic duct, and pulmonary veins (Figure 7a). The azygous vein appeared as a black vessel dorsal to the aorta. Intercostal veins branched into the azygous vein from the caudal border of each rib. The proximal portion of the azygous vein descended ventrally along the mediastinum and disappeared medial to the dorsal border of the lung. Just caudal to the descending azygous, the right pulmonary veins were seen partially covered by the esophagus. The pulmonary vessels were large, triangularly shaped and emerged from the cranial-dorsal border of the lung (Figure 7b). The pulmonary veins could not be seen within the left hemithorax. The esophagus was observed ventral to the aorta as a dark red, flaccid, tubular structure. Occasionally, peristalsis was seen in the esophagus. Close inspection of the esophagus showed the dorsally located esophageal artery and vein and the vagus nerve (Figure 7b).

The aorta was seen as a large pulsating vessel located dorsally next to the costo-vertebral arch in both hemithoraces (Figure 8a). Vessels stemmed from the dorsal aspect of the aorta and formed the dorsal intercostal arteries coursing along the caudal border of each rib. The aortic vasa-vasorum were seen upon close inspection. Above the aorta, the sympathetic trunk and its major branch (splanchnic nerve) were seen in both hemithoraces as a flat band of nerve fibers coursing along the thoracic vault, lateral to the costo-vertebral articulations and dorsal to the aorta (Figure 8a). The aorta could be followed cranially to the aortic arch and caudally to the aortic hiatus of the diaphragm. A large mediastinal lymph node could be found in both hemithoraces within the mediastinum below the aorta and above the esophagus (Figure 8b). The vagus nerve was closely associated with the mediastinal lymph node and, in this location, divided into dorsal and ventral branches. This could be found in both hemithoraces but was best seen when performing a left hemithoracoscopy.

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Approximately two-thirds of the costal surface of each lung was easily observed. The lung margins could be followed starting from the pulmonary ligament proceeding along the caudal-most aspect and on to the ventral (costal) margin (Figure 9a). Associated with the dorsal border of right and left lungs were the esophageal groove medially and the aortic groove laterally. The diaphragmatic surface of the lung was inspected after guiding the telescope beneath the pulmonary ligament. The telescope then could be advanced cranially to reach what appeared to be pericardial adipose tissue.

When moving the telescope ventrally, the ribs and associated internal intercostal muscles and vasculature could be followed, and the caudal edge of the collapsed lung was seen gliding on the pleural surface of the diaphragm (Figure 9a). Directing the telescope caudally, the costal attachment of the diaphragm to the rib cage could be found (Figure 9b). The diaphragmatic hiatus was limited on each side by the combined psoas minor and major (Figures 10a and b). The diaphragm was partially covered by collapsed lung and, in most horses, the hiatal region was surrounded by adipose tissue, thus preventing a distinct view of the esophagus, the aorta, and the thoracic duct coursing into the abdominal cavity. In the left hemithorax, the hiatal region was clearer and the aorta could be found easily with the esophagus below (Figure 10b).

The trocar/cannula used for the accessory portal was inserted while the pleural aspect of the selected intercostal space was in view of the telescope. This avoided accidental lung perforation and allowed accurate portal placement. With the blunt tissue palpation probe distance from the costal surface and accessibility of the thoracic anatomical structures could be assessed. The accessory instrument could be placed on the lung (costal, caudal and dorsal surfaces), aorta, esophagus, vagus and sympathetic nerves, mediastinal lymph nodes, and diaphragm (costal muscular attachments and hiatal

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region). The majority of the diaphragm was covered by the collapsed lung. However, the probing instrument could be directed beneath the lung surface to elevate the lung parenchyma and inspect a greater portion of the tendinous diaphragm. The probe also aided in inspecting the medial (diaphragmatic) surface of the lung. This was achieved by elevating the pulmonary ligament with the probe and advancing the telescope in the space formed between the lung and the diaphragm.

Discussion

The endoscopic examination of the normal equine thorax was successfully completed using an 58-cm-long rigid telescope and video assistance. Restraining the horses in stocks was effective in limiting movement, and with the sedation and analgesia provided by detomidine administration, the procedures were carried out safely. Pain control was aided by the use of regional and local anesthesia. In four out of twelve cases, regional anesthesia was provided via intercostal nerve blocks. Three intercostal spaces were anesthetized in order to maximize the area of desensitization and include both the endoscopic and accessory portal surgical sites. The 7th, 8th, and 9th intercostal nerves were most difficult to anesthetize due to large amounts of subcutaneous adipose tissue and heavier muscular coverage in the cranial thoracic regions. A 3½-inch 18 g spinal needle helped deliver the local anesthetic appropriately to these nerves. Regional anesthesia provided optimal desensitization when successfully completed. However, in one instance, additional intra-operative local anesthesia was required to place the accessory portal.

Approximately two-thirds of the thoracic cavity could be explored with observation of major portions of the lungs, thoracic aorta, esophagus, and diaphragm.

The cranial and ventral one-third of the thoracic cavity were difficult to observe even when an approach as cranial as the 8th intercostal space was used. When entering the thorax via the 8th intercostal space, the rigid telescope could not be easily advanced cranially and caudally without causing the horses to show signs of discomfort such as agitation, groaning, and decreased level of sedation. These signs of discomfort were likely associated with the distraction of the more rigid cranial thoracic ribs by the telescope. This response was not dependent upon the method chosen to provide local anesthesia and was not observed when more caudal approaches were used. Entering the thorax via the 10th and 12th intercostal spaces allowed a comfortable exploration of the thorax, including the cranial-most thoracic regions. The telescope could be easily advanced cranially and caudally for its entire length, without eliciting a painful response.

A blunt, stainless steel teat cannula inserted through the parietal pleura provided the necessary degree of pneumothorax to introduce the trocar/cannula system. Pneumothorax, however, was inadequate in one horse and parenchymal perforation occurred during insertion of the trocar. The perforated lung was inspected once the telescope was placed within the pleural space. The laceration was approximately 6-7-mm long and involved the visceral pleura and a portion of the lung parenchyma. Minimal hemorrhage was associated with the injury site and the exploration of the thorax proceeded uneventfully. Postoperatively, thoracic radiographs indicated the presence of a closed pneumothorax, which was not associated with clinical signs such as respiratory distress or tachycardia. The horse remained asymptomatic, was observed for 7 days postoperatively, and was released from the Large Animal Clinic. Guarded, disposable cannulas coupled with blunt trocars could be an additional safety measure employed to

prevent pleural damage, although adequate lung collapse alone should ensure safe trocar placement.

Sudden lung collapse and degree of pneumothorax may be an additional source of discomfort for horses undergoing thoracoscopy. Acute pneumothorax caused coughing and distress in one case and may have been associated with the sudden pressure exerted by the lung parenchyma on the mediastinal vascular and nervous structures. The lung can be gradually collapsed by intermittently opening and closing the teat cannula used to create the pneumothorax. In addition, the degree of lung collapse can be varied during the procedures by applying suction to the pleural space. It is important to regulate the degree of pneumothorax depending upon the anatomical region to be inspected and the overall cardio-pulmonary status of the horse. In order to ensure appropriate control of the pneumothorax, it is critical to use endoscopic cannulas manufactured with a side stopcock to which the suction tubing can be attached. This will allow rapid control of intrapleural pressure and relief of the pneumothorax if needed.

Mediastinal integrity is an important issue when performing thoracoscopy. The caudal mediastinum is thought to be occasionally incomplete in the adult horse,⁷ allowing the two pleural cavities to be in direct communication. In fact, bilateral pneumothorax was never observed during thoracoscopy. Throughout both left and right hemithoracoscopic procedures, the fully inflated contralateral lung could be seen through the dorsal mediastinum.

Endoscopic portal placement requires additional considerations. Portal site incisions should only involve skin and subcutaneous tissues. Large or deep incisions may not provide an adequate seal around the cannula. This may make it difficult to maintain a negative pleural pressure following the use of suction and may cause peri-incisional

postoperative subcutaneous emphysema. Following selection of the intercostal space, the trocar should be directed toward the cranial aspect of the rib to avoid the intercostal neurovascular bundle located on the caudal aspect of each rib. The trocar should be gently rotated while advanced until a sudden decrease in resistance indicates entry into the pleural cavity.

Thoracoscopy was well tolerated and no clinically important complications were observed during and after the procedures. Thoracoscopy allows direct observation of the intrathoracic structures. Thoracoscopy can be successfully used to explore the thorax and may be beneficial in disease as a diagnostic and therapeutic technique in combination with traditional methods such as radiography and ultrasound.

Manufacturers' addresses

^aBanamine, Schering Plough, Kenilworth, New Jersey, USA.

^bAngiocath, Becton Dickinson, Sandy, Utah, USA.

^cDormosedan, Pfizer Animal Health, Exton, Pennsylvania, USA.

^d3M Ioban II, 3M Health Care, St. Paul, Minnesota, USA

^eCarbocaine, Sanofi-Winthrop, New York, New York, USA

^fStorz Veterinary Endoscopy, Goleta, California, USA.

^gEthicon, Somerville, New Jersey, USA.

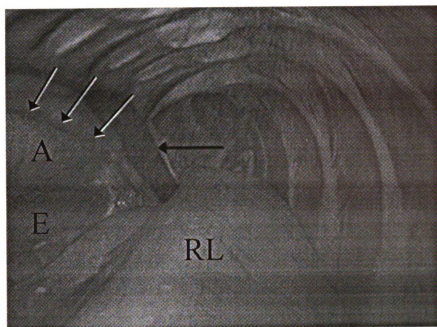
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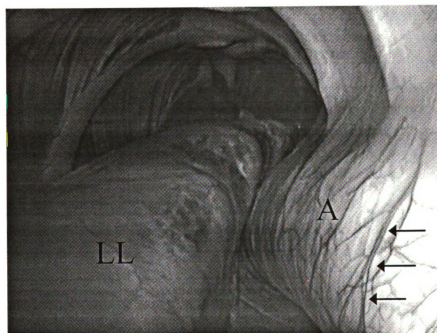
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Figure 5a: Thoracoscopic view of the cranial region of a right hemithorax showing, in the foreground, the collapsed right lung (RL). Mediastinal structures such as esophagus (E), aorta (A), thoracic duct (black arrows) and azygos vein (white arrow) are also visible.

Figure 5b: Thoracoscopic view of the cranial region of a left hemithorax showing the collapsed left lung (LL) medial to which the aorta (A) with its vasa vasorum (black arrows) is seen.



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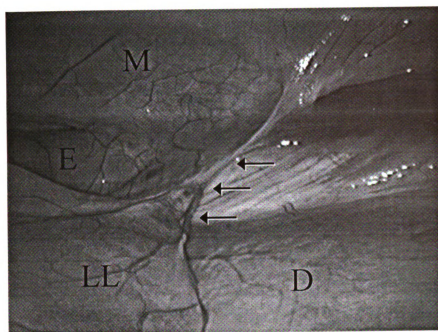


b)

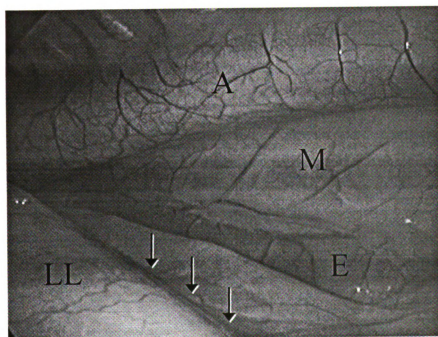
Figure 5

Figure 6a: Thoracoscopic view of the caudal-dorsal region of a left hemithorax. The pulmonary ligament can be identified (black arrows) leading to the collapsed left lung (LL). The caudal edge of the lung lies on the diaphragm (D). The dorsal mediastinum (M), through which the inflated right lung can be seen, is located dorsally. Below the mediastinum the esophagus (E) is found.

Figure 6b: Thoracoscopic view of the mid-dorsal region of a left hemithorax. With respect to figure 2a, the telescope has been directed cranially and dorsally. In the foreground is the dorsal border of the collapsed left lung (LL) with the aortic impression (black arrows). Medial to the lung the esophagus (E) is seen and the dorsal mediastinum (M), through which the inflated right lung can be seen and the aorta (A) are identified dorsally.



a)



b)

Figure 6

Figure 7a: Thoracoscopic view of the cranial region of a right hemithorax. The dorsal border of the right lung (RL) is seen. The esophagus (E) with its vasculature and the vagus nerve (black arrows) partially covers the right pulmonary veins (white arrow). The aorta (A) and azygos vein (AZ) are observed dorsally.

Figure 7b: Thoracoscopic view of the cranial region of a right hemithorax. Close up view of the pulmonary veins (white arrows) seen in figure 3a. The collapsed lung (RL) with the aortic impression, the esophagus (E) and the vagus nerve (black arrows) are also seen.

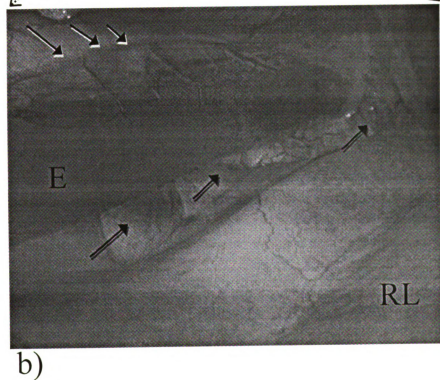
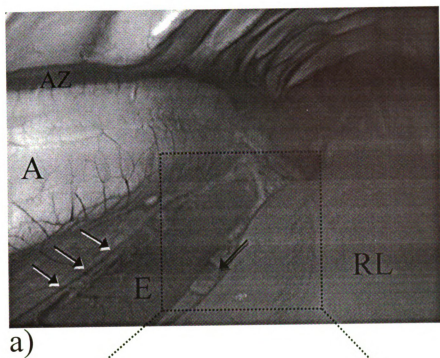
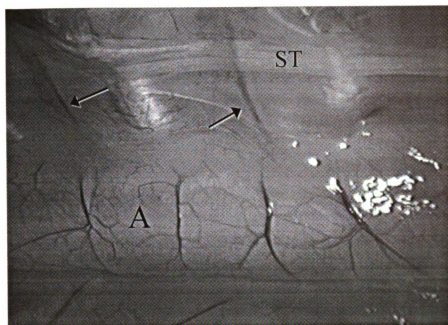


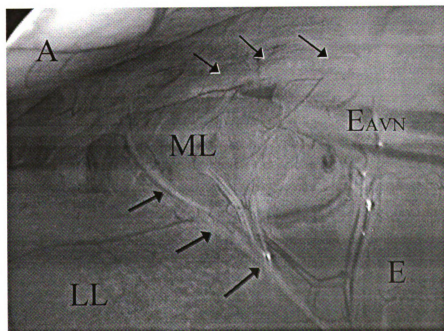
Figure 7

Figure 8a: Thoracoscopic view of the dorsal-medial region of a left hemithorax. Detail view of the sympathetic trunk (ST) coursing dorsal to the aorta (A) and over the intercostal vasculature (black arrows).

Figure 8b: Thoracoscopic view of the cranial-medial region of a left hemithorax. Located between the aorta (A) and the collapsed medial lung lobe (LL) is a mediastinal lymph node (ML). The white arrows and the black arrows respectively identify the ventral and dorsal branches of the vagus nerve. The esophagus (E) and the esophageal artery, vein and nerve (EAVN) are also seen.



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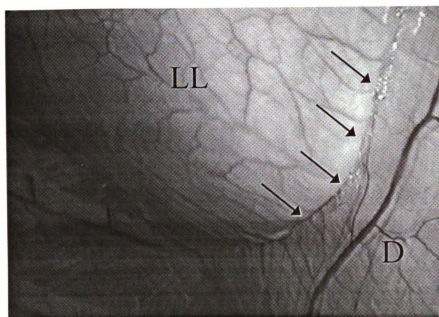


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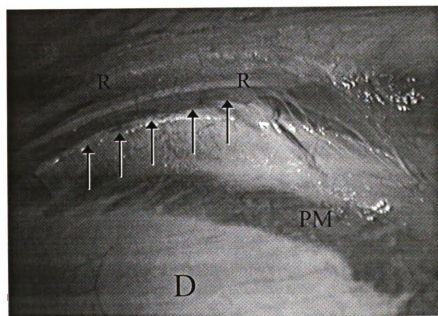
Figure 8

Figure 9a: Thoracoscopic view of the caudal-ventral region of a left hemithorax. The telescope is directed between the thoracic wall and the diaphragm (D). The collapsed lung is seen (LL) and the black arrows identify the caudal (diaphragmatic) lung edge.

Figure 9b: Thoracoscopic view of the caudal-dorsal region of a right hemithorax. The dorsal most aspect of the diaphragm (D) is seen where it attaches to the rib cage (black arrows). Ribs 14 and 15 can be identified (R).



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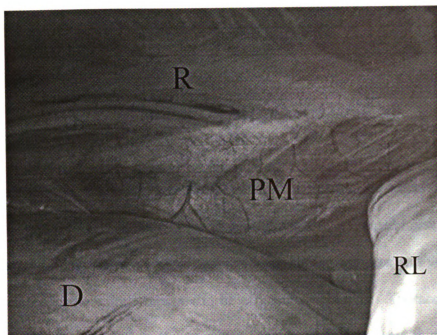


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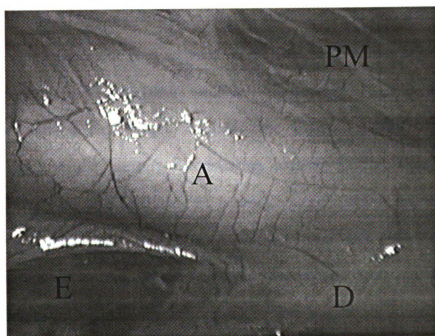
Figure 9

Figure 10a: Thoracoscopic view of the region correspondent to the diaphragmatic hiatus of a right hemithorax. The psoas major muscle (PM) defines the hiatal area of the diaphragm (D) as it connects to the rib cage in the area of the 14th rib (R). The partially collapsed right lung (RL) can also be seen.

Figure 10b: Thoracoscopic view of the region correspondent to the diaphragmatic hiatus of a left hemithorax. The aorta (A) is located ventrally to the psoas major muscle (PM) as it passes into the abdominal cavity through the diaphragm (D). The esophagus (E) is seen below the aorta.



a)



b)

Figure 10

SUMMARY AND CONCLUSIONS

With this study we have been able to demonstrate the safety of thoracoscopy performed in the healthy, awake, and pharmacologically restrained horse. Thoracoscopy allowed us to examine the normal thoracic anatomical structures and verify that with a rigid telescope the equine thorax can be efficiently explored. Thoracoscopy has been proven to be useful clinically in the diagnosis and treatment of pleuropulmonary pathology and with this research we support the use of this technique having verified its advantages and described the cardiopulmonary consequences of the procedure.

Thoracoscopy is meant to be utilized in clinical cases, therefore, the assessment of the consequences of pneumothorax in the face of thoracic disease should receive further attention. Multiple factors would merit further attention during thoracoscopy: the use of intra-tracheal oxygen administration, different sedation modalities from detomidine, pleural pressure determination intra-operatively, operations conducted consecutively on both hemithoraces, improved surgical techniques, and thoracoscopy performed under general anesthesia. Thoracoscopy has been shown to be an effective and safe method for thoracic examination and its use in the horse should be encouraged.

