Thoracic trauma, although rare, can be a diagnostic and therapeutic challenge and clinicians should be prepared to institute emergency medical treatment particularly when injuries create an open thorax or in cases of severe blunt trauma. In the latter case, the symptomatology can be deceiving because, contusions, collapse or laceration of the lung can occur without apparent external lesions. Furthermore, the presence of cardio-respiratory symptoms with thoracic trauma requires critical care management and consequently necessitates referral to a hospital.

Pathophysiology

When evaluating horses with chest injuries, veterinarians may be challenged with the diagnosis and management of a pneumothorax. Additionally open chest injuries present challenges relating to pleural contamination and infection, presence of foreign material and rib fractures. Traumatic pneumothorax, in particular, is usually secondary to a penetrating chest injury, however, non-penetrating blunt trauma may also cause pneumothorax by compressing and rupturing alveolar clusters causing air to leak from the lower respiratory tree into the pleural space. It is our experience that horses with acute chest trauma are those most susceptible to severe complications and should therefore be carefully selected and managed as surgical candidates. Pneumothorax indicates the presence of “free” air or gas within the pleural space. In most instances, 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). Pneumothorax may occur spontaneously, may be a complication of pleuritis or pneumonia, a result of thoracic trauma and can be iatrogenically induced.

During spontaneous breathing and in normal conditions 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 cmH₂O) during inspiration when the chest wall is expanded through the effort of the inspiratory muscles (mainly diaphragm and external intercostal muscles). 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. 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 further 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. In addition, a decrease in arterial oxygen tension has also been found following spontaneous pneumothorax in people. 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.

Cardiac output can be negatively affected by pressure changes occurring within the chest cavity. 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.

**Traumatic pneumothorax.**

Blunt or sharp trauma to the chest wall and iatrogenic procedures (placement of chest tubes, drains or thoracic procedures) are leading causes of pneumothorax in horses. Most thoracic injuries are unilateral and may involve the axillary region or the lateral chest wall and associated ribs. Although possible, injuries to the pectoral region rarely involve intra-thoracic structures because of the powerful musculature covering this area, the narrow thoracic opening between the first ribs and the parabolic shape of the anterior thorax. At the time of injury, horses exhibit clinical signs resulting from the combination of traumatic shock and the onset of pneumothorax. Restlessness and apprehension, tachycardia, tachypnea, dyspnea and cyanotic mucous membranes are common physical findings. The severity of the signs associated with pneumothorax depends on the speed at which the lung collapses following injury, the presence of bilateral pneumothorax and whether the injury is open or closed. Two important complications of pneumothorax produce severe and characteristic clinical signs: tension pneumothorax and pneumomediastinum. Tension pneumothorax is a cause of severe cardiopulmonary compromise following chest trauma. A tension pneumothorax occurs when a one way valve develops either in the chest wall or the lacerated lung parenchyma. During inspiration air enters the pleural space and accumulates because the one way valve does not allow the inspired air to by released during expiration. Pleural pressure increases above atmospheric causing blood gas deterioration with severe ventilation to perfusion mismatch leading to cardiopulmonary failure. A pneumothorax may lead to a pneumomediastinum when excessive intra-alveolar pressures leads to rupture of perivascular alveoli. Air escapes into the perivascular connective tissue with subsequent dissection into the mediastinum. Air may then dissect proximally into the visceral, retropharyngeal, and subcutaneous spaces of the neck (subcutaneous emphysema) Mediastinal air can also pass into the retroperitoneum and other extraperitoneal compartments.

**Emergency intervention following chest trauma.**

There are two principal goals in the treatment of pneumothorax: first eliminate the air from the pleural space and second prevent recurrence. In case of 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. 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 O2. This is based on the principal that gases diffuse through biological membranes at a rate depending on pressure gradients. In 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 are available for gas exchange and the solubility of each gas in the tissues. Evacuation of a pneumothorax can be achieved by several invasive methods. Simple aspiration has minimal morbidity and is reserved for small animals but has been successfully used in adult horses presented with the first occurrence of primary spontaneous pneumothorax. Tube thoracostomy is used frequently in horses that present with open chest trauma. Air 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 conditions that do not resolve, when there are rib fractures causing lung laceration, in the presence of foreign material, or in case of a broncho-pleural fistula. Thoracoscopy has been increasingly used in the acute phase of thoracic trauma. Another indication for its use, which is rarely described in the literature, is the removal of retained intrathoracic foreign bodies. In the human field residual hemothorax is the second most common complication of chest trauma after rib fracture. These are common occurrences in horses with acute open or closed chest injuries. Pulmonary or mediastinal injures can be successfully evaluated in the horse assisting the clinician in making the most appropriate treatment decisions.