Thoracic Trauma
The Deadly Dozen

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Deadly Dozen ... Lethal Six ... Hidden Six ... Major thoracic injuries are known as the Deadly Dozen. The Lethal Six (airway obstruction, tension pneumothorax, cardiac tamponade, open pneumothorax, massive hemothorax, and flail chest) are immediate, life-threatening injuries that require evaluation and treatment during primary survey. The Hidden Six (thoracic aortic disruption, tracheobronchial disruption, myocardial contusion, traumatic diaphragmatic tear, esophageal disruption, and pulmonary contusion) are potentially life-threatening injuries that should be detected during secondary survey. Each of these may present as immediately life-threatening or potentially life-threatening events. This article provides an overview of these 12 injuries and appropriate management for each. Case studies are included. Key words: airway obstruction, aortic disruption, cardiac tamponade, flail chest, esophageal disruption, massive hemothorax, myocardial contusion, open pneumothorax, pulmonary contusion, tension pneumothorax, tracheobronchial disruption, traumatic diaphragmatic hernia

Thoracic injuries account for approximately 25% of trauma-related deaths. They contribute to an additional 25% of deaths in the United States annually. Great vessels injuries, or disruption of the heart, usually result in immediate death. Early deaths, those that occur within 30 minutes to 3 hours, are due to cardiac tamponade, tension pneumothorax, aspiration, or airway obstruction. Although some of these injuries (most of them cardiac) require emergent surgical intervention, most injuries to the lungs and pleura can be treated nonoperatively by applying certain fundamental principles of initial trauma management, which can substantially reduce morbidity and mortality related to these injuries. A thorough knowledge of the pathophysiology of cardiac and pleuropulmonary injuries is of utmost importance for optimal treatment.

A rapid initial assessment should be completed upon arrival at the trauma center. Assessment is based on a series of diagnostic clues obtained from directed data collection. Index of suspicion is the initial data collected to form this diagnostic set. Orderly evaluation will identify injuries that are life threatening, for example, tension pneumothorax, open pneumothorax, and massive hemothorax. The secondary assessment will identify the majority of lung and pleural injuries, which include pulmonary contusions, pneumothorax, and hemothoraces.

Airway Obstruction

The first priority in treating trauma patients is airway management. The cornerstones of airway management are adequate oxygenation, ventilation, and protection from aspiration. Immediate airway control can prevent death. Airway obstruction can be a primary problem or the result of other injury. Although the source of, and approach to, an obstruction may be slightly different, the principles of basic and advanced life support are
fundamental to managing obstructions. Initial priorities with all trauma patients are evaluation and management of the airway, breathing, and circulation, cervical spine with stabilization, and level of consciousness.

The most common causes of airway obstruction are the tongue, avulsed teeth, dentures, secretions, and blood. But, expanding hematomas that cause compression of the trachea, and thyroid cartilage or cricoid fractures resulting in hemorrhage and edema may also be sources of obstruction. Airway compromise can be acute, insidious, progressive, and/or recurrent. The single most frequent indication for intubation is an unconscious patient suffering compromised ventilatory effort. Key issues in managing airway difficulty include the following:

1. Delivering adequate oxygen to vital organs
2. Maintaining a patent airway
3. Ensuring adequate ventilation
4. Protecting the cervical spine
5. Recognizing the need for endotracheal intubation
6. Knowing how to utilize rapid sequence intubation
7. Being proficient in surgical airway techniques
8. Preventing hypercarbia is critical!

Upon clinical evaluation, patients present with signs of anxiety, hoarseness, stridor, air hunger, hypoventilation, use of accessory muscles, sternal and supraclavicular retractions, diaphragmatic breathing, altered mental status, apnea, and cyanosis (sign of preterminal hypoxia). Cyanosis is a very late sign of preterminal hypoxia, since it requires at least 5 g of reduced hemoglobin to be clinically detectable. Trauma patients may be anemic to such a degree that they do not have enough hemoglobin to appear cyanotic. Indications for airway interventions are divided into 3 broad categories outlined in Table 1.

Basic management for airway compromise includes

1. securing an intact airway,
2. protecting the airway if jeopardized, and
3. providing an airway if partially obstructed or totally obstructed.

Basic, advanced, and surgical airway intervention techniques must be performed, even though the patient may have a cervical spine injury. Indications for surgical cricothyroidotomy are edema of the glottis, fracture of the larynx, or severe hemorrhage obstructing the airway. Remember, when in doubt, intubate; protect the cervical spine, and intubate early, especially in cases of neck hematomas or possible airway edema. Airway edema can be insidious and progressive, and may make delayed intubation more difficult, if not impossible.

Three young men, all aged 22 years, were involved in a motor vehicle crash (MVC) while driving in a sports utility vehicle. The restrained driver lost control at approximately 45 mph and hit a car parked on the side of the road. No one was ejected, but the 2 passengers were not restrained and sustained significant injuries. The 3 victims were transported to the emergency department (ED) for treatment.

The driver was awake upon arrival. He had abrasions over his chest and face, was complaining of some chest pain, and was breathing at a rate of 28 breaths per minute (bpm). He stated that upon impact, he hit the steering wheel with his chest. An arterial blood gas on room air reflected a pH of 7.44, PCO2 of 32, Pao2 of 53, and Hco3 of 24. Auscultation revealed decreased breath sounds and rales on the right side. A chest x-ray (CXR) was performed and showed mild pulmonary infiltration in the right lower lobe, consistent with pulmonary contusion.

PULMONARY CONTUSION

Pulmonary contusion is the most common, potentially lethal, chest injury. The resulting respiratory failure develops over time rather than immediately. Pulmonary contusion is the bruising of lung tissue resulting from a shock wave of force through the parenchyma. Diffuse hemorrhage follows, as well as interstitial and alveolar edema, causing impairment of gas exchange at the gas tissue interface. The most common mechanism of injury causing pulmonary contusion is MVC.
Table 1. Indications for invasive airway management*

<table>
<thead>
<tr>
<th>Absolute indications</th>
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<tbody>
<tr>
<td>Obstruction</td>
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<tr>
<td>Apnea</td>
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<tr>
<td>Hypoxia</td>
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<tr>
<td>Expanding neck hematomas</td>
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<table>
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<tr>
<th>Strong relative indications</th>
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<tbody>
<tr>
<td>Significant craniocerebral injury</td>
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<tr>
<td>(coma, increased intracranial pressure, central nervous system trauma, etc)</td>
</tr>
<tr>
<td>Ventilatory compromise</td>
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<tr>
<td>(disruption of the thoracic bellows mechanism)</td>
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<tr>
<td>Massive retroperitoneal hemorrhage/hematomas (causing displacement of the intraperitoneal viscera and upward pressure on the diaphragm)</td>
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<tr>
<td>Combative patient with life-threatening injuries</td>
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<tr>
<th>Relative indications</th>
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<tbody>
<tr>
<td>Severe maxillofacial trauma</td>
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<tr>
<td>Extensive pulmonary contusion</td>
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*Adapted from Phillips.³

Up to 87% of patients with pulmonary contusions have at least one other associated chest injury.⁴ The greater the degree of pulmonary contusion, the greater the degree of ventilatory impairment. Pulmonary contusion is usually the result of a high-velocity-deceleration injury such as an impact with a steering wheel. The initial force of the injury causes tissue rupture. Additional damage occurs as the tissue “springs back.” For this reason, it is more common to see pulmonary contusion injuries in younger patients, because their chest walls are more flexible and stretch due to the external forces, whereas the elderly with stiffer, thinner chest walls are more prone to rib fractures.⁵ Greater contusions are sustained in those individuals with thin-wall chests, because there is less protection provided by muscle and adipose tissue.

Clinically, patients present to the ED with signs of respiratory distress, including dyspnea, and PaO₂ less than 60 on room air. Auscultation of breath sounds may reveal decreased breath sounds, rales, and wheezing over the next 24 hours. Upon assessment, the patient may complain of chest pain or have abrasions on the chest or back. Patients may also exhibit ineffective cough with hemoptysis. None of these signs, or symptoms, are specific for pulmonary contusion, but hemoptysis has been reported in up to 50% of patients.⁴ Chest x-ray reveals consolidation and pulmonary infiltration at the area of injury, but these findings may lag 12 to 24 hours. If abnormalities are seen on the admission x-ray, the pulmonary contusion is severe. For this reason, the cornerstone of diagnosis is clinical suspicion based on the history and mechanism of injury.⁴

Some patients can be treated without intubation and mechanical ventilatory support. The management of patients who sustain pulmonary contusions is based on 3 factors⁵: overall stability of the patient, adequacy of oxygenation, and pulmonary mechanics. For patients who do not need ventilatory support, the following criteria⁵ should be met:

1. PaO₂ more than 60 on 50% inspired oxygen,
2. respiratory rate less than 24 bpm,
3. spontaneous tidal volume more than 5 mL/kg, and
4. vital capacity exceeding 10 mL/kg.

The driver was placed on 100% O₂ by face mask and given morphine sulphate (MSO₄), 4 mg, intravenous (IV), for pain. On reassessment an hour later, he continued to exhibit...
dyspnea with weak cough. He had difficulty clearing bloody sputum from his airway. The physician decided to intubate the patient and placed him on mechanical ventilation.

Progressively, the patient may show decreased lung compliance and increased work of breathing, with a stiff, wet lung picture requiring endotracheal intubation and mechanical ventilation. Intubation should be undertaken if a PaO₂ of 60 mm Hg with an FiO₂ of 50% cannot be maintained. When using the PaO₂/FiO₂ ratio to determine whether to intubate, the normal ratio is 500, with intubation being indicated for patients with ratios below 300. While on ventilatory support, the ideal inspired oxygen should not exceed 50% while maintaining a PaO₂ of more than 60 mm Hg. If the level of oxygen cannot be maintained, positive end-expiratory pressure or continuous positive airway pressure is added.

Initial treatment of pulmonary contusion includes supplemental oxygen therapy, monitoring oxygen saturations, aggressive pulmonary toilet to help clear bloody secretions from the airway, and administration of analgesics. Because pulmonary contusion leads to capillary membrane leak, judicious fluid management is essential to minimize the formation of edema in the injured region of the lung.

Hemoptysis, or blood in the endotracheal tube, is a sign of pulmonary contusion. Intubated patients require frequent suctioning to clear blood, tissue, and mucus from the airway. For patients with moderate to severe contusion, intubation and intermittent mandatory ventilation, as well as positive end-expiratory pressure, provides better results if ventilatory assistance is needed. It is the nurse's responsibility to monitor secretions. They should become thinner and contain less blood over the first few days posttrauma. Intubation and mechanical ventilation are not always indicated, especially if the patient is able to maintain spontaneous ventilation with adequate PaO₂, and is able to clear secretions with coughing. For patients who are mechanically ventilated, it is important for the RN to monitor arterial blood gas results, peak inspiratory pressures, and lung compliance. Rising peak inspiratory pressures with decreased compliance may indicate the need for pressure-control ventilation to avoid barotrauma.

Barotrauma results from overdistention and rupture of the alveoli. Secretions trapped in the lower airways may cause obstruction. On inspiration, the ventilator cycles gas past the partial obstruction into the alveoli. Consequently, when the expiration of gas is blocked, the lung remains inflated. When the cycle repeats itself, the alveolus ruptures. Barotrauma can be reduced by maintaining adequate analgesia and sedation levels. If the patient is not responsive to conventional ventilation therapy, and has a catastrophic pulmonary contusion, then pressure-limiting ventilatory modes, such as jet ventilation, are considered. Adult extracorporeal membrane oxygenation is also an option. Pneumonia is the most common complication of pulmonary contusion and, when present, worsens the prognosis.

Fluid management remains controversial. However, if the patient has a significant pulmonary contusion, placement of a pulmonary artery catheter may facilitate the monitoring of fluid resuscitation.

The front passenger was second to arrive at the ED. He was clearly in respiratory distress with a respiratory rate of 34 bpm, and using accessory muscles. He exhibited asymmetric chest wall movement, with left expansion greater than right. Examination showed shallow depth of breathing; lung sounds were diminished bilaterally. He presented with 100% O₂ by nonrebreather face mask. Heart rate was 120 bpm. The nurse palpated crepitus on his right chest wall. As the patient's respiratory status worsened, a significant paradoxical movement of a segment of rib was noticed on his right lateral chest. Recognizing this as a flail segment, the nurse anticipated the need for endotracheal intubation for ventilatory support, and pain control via epidural catheter.
FLAIL CHEST

Flail chest is a common major injury to the chest wall, occurring in approximately 20% of admitted trauma patients. There is an associated mortality rate, as high as 50% in some series. It can be even higher in patients older than 60 years. The diagnosis of flail chest injury is made on the basis of fractures of 2 or more ribs, in 2 or more separate locations, causing an unstable segment. The flail segment classically involves anterior (sternal separation) or lateral rib fractures. Posterior rib fractures rarely produce a flail segment because of the heavy musculature that provides stability. These fractures create a free-floating segment of rib or sternum, resulting in a paradoxical movement relative to the rest of the chest wall during inspiration and expiration. The segment follows pleural pressure instead of respiratory muscle activity, which is sucked in during inspiration and protrudes out during expiration. This movement may not be evident on initial assessment because intercostal muscles in spasm act as a splint for the flail segment. This may be a contributing factor in the failure to identify flail chest within the first 6 hours in up to 30% of patients with this injury. As the patient’s pulmonary status worsens, the paradoxical movement of the flail segment will increase. Although the paradoxical motion of the unstable flail can greatly increase the work of breathing, the main cause of hypoxemia of flail chest is the underlying pulmonary contusion, which invariably occurs with flail chest. Nonetheless, paradoxical movement prevents full expansion of the underlying lung, decreasing minute ventilation. Pain is a contributing factor, preventing the patient from taking full breaths.

As mentioned previously, the diagnosis of flail chest injury is made on the basis of fractures of 2 or more ribs, in 2 or more separate locations, with resultant paradoxical motion of that segment of the chest wall. The chest wall must be observed for several respiratory cycles and during coughing. Patients with flail chest present with asymmetric chest movement and signs of respiratory distress, including increased respiratory rate and work of breathing, and decreased tidal volumes. Crepitus may be palpated around the flail segment, and patients will complain of chest pain. Over time, the patient fatigues and requires mechanical ventilation to maintain adequate oxygenation and minute ventilation.

Treatment of flail chest includes appropriate airway management, supplemental oxygen therapy to maintain the PaO$_2$ at levels of 80 to 100 mm Hg, and pain control. Stabilizing the flail segment to reestablish the thoracic bellows effect, and promoting air exchange is essential. Positioning the patient with the injured side down, as well as IV pain medications, may be temporarily beneficial. Splinting the flail segment with rolled towels or tape may be beneficial if it increases the patient's tidal volumes. Sandbags should not be used, as they add unnecessary resistance to respiratory efforts. Contrary to the standard treatment for trauma patients, fluids should be used cautiously in the setting of flail chest because of the high probability of underlying pulmonary contusion. Excessive IV fluids will contribute to edema of the injured area of the lung.

Intubation/tracheostomy and mechanical ventilation are indicated if the respiratory rate is faster than 35 bpm, or less than 8 bpm, if PaO$_2$ is below 60 mm Hg on supplemental oxygen at 50%, if PaCO$_2$ is acutely above 55 mm Hg, or if vital capacity is less than 15 mL/kg. For segments larger than 4 to 6 in, or multiple flail segments, positive-pressure ventilation is the optimal solution. Internal splinting through positive-pressure ventilation not only corrects paradoxical chest movement but it also decreases the work of breathing and pain. It is not uncommon for patients to be mechanically ventilated for up to 3 weeks as the fracture heals. Surgical internal stabilization is a quicker treatment.
Table 2. Ventilatory therapy*  

<table>
<thead>
<tr>
<th>Choice of therapy depends on:</th>
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<tr>
<td>Size of the flail</td>
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<tr>
<td>Degree of pulmonary dysfunction</td>
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<tr>
<td>Effort of breathing and fatigue</td>
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<tr>
<td>Presence of concomitant thoracic injuries</td>
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<tr>
<td>Pulmonary contusions</td>
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<tr>
<td>Need for general anesthesia</td>
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<tr>
<td>Surgical procedures related to associated</td>
</tr>
<tr>
<td>traumatic injuries</td>
</tr>
<tr>
<td>Risk of posttraumatic respiratory</td>
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<tr>
<td>insufficiency</td>
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*From Sherwood and Hartsock.  

Permanent ventilatory impairment may require long-term management. Supplemental oxygen therapy and vigilant pulmonary toiletting, including postural drainage, chest physiotherapy, and use of the incentive spirometer, are used to prevent pulmonary infection, strengthen respiratory muscles, and to maintain a clear airway. Some patients continue to experience intractable intercostal pain after the flail chest has healed. Nonpharmacologic treatments such as transcutaneous electrical nerve stimulators may also be helpful for pain control when pain medicines are no longer effective or the patient cannot tolerate its side effects. Other adjunctive therapies include massage and positioning.  

The patient was intubated, sedated, and placed on mechanical ventilation to manage his flail chest. As the nurse continued to monitor the patient, be noticed that the patient's blood pressure (BP) dropped to 40/20 and heart rate increased to 135 bpm. Pulses, upon palpation, were weak and thready. Breath sounds, on auscultation, were significantly diminished on the left side. The left chest was dull to percussion. The patient's oxyhemoglobin saturation measured 85%. Upon examination, the trachea was deviated to the right. Knowing this is a life-threatening change in the patient's status, the nurses suspected a massive hemothorax and notified the physician while immediately setting up for chest tube insertion.  

HEMOTHORAX/MASSIVE HEMOTHORAX  

Hemothorax is the accumulation of blood in the pleural space caused by bleeding from the chest wall, lung parenchyma, or major thoracic vessels. It occurs in roughly 25% of patients with chest trauma. Patients with hemothorax typically present with decreased breath sounds unilaterally or bilaterally with dyspnea, tachypnea, and dullness to percussion over the affected side. The primary cause of hemothorax is either a laceration to the lung, an intercostal vessel, or an internal mammary artery because of blunt or penetrating.
trauma. Bleeding in these types of cases is usually self-limiting and does not require surgical intervention. Radiographic films may not reveal a fluid collection of less than 300 mL. Bleeding from parenchymal lacerations often stops on its own because of the low pulmonary pressures and high concentrations of tissue thromboplastin in the lung. Small hemothoraces usually seal themselves within a few days.

Accumulation of greater than 1500 mL of blood is considered a massive hemothorax that can have disastrous results. A left-sided massive hemothorax is more common than the right-sided one, and is typically associated with aortic rupture. A massive hemothorax is commonly due to penetrating trauma with hilar or systemic vessel disruption.

Because the chest cavity can hold most of a patient's circulatory volume, it is possible for the patient to become hemodynamically unstable emergently in the case of a massive hemothorax. Signs of shock, like hypotension, decreased venous return, and cyanosis are common because of hypovolemia and increased pressure in the thorax. Neck veins may be flat, secondary to hypovolemia, or distended because of the mechanical effects of intrathoracic pressure. A mediastinal shift and/or tracheal deviation is a classic sign caused when the contents of the chest cavity are shifted away from the blood accumulation because of increased intrathoracic pressure.

Treatment of acute hemothorax includes supplemental oxygen therapy and, in most cases, the insertion of a large (36-38 French) chest tube (tube thoracostomy) just anterior to the midaxillary line at the fourth or fifth intercostal space to allow for chest decompression. A moderate-size hemothorax (500-1500 mL) that stops bleeding after a thoracostomy is usually treated with a closed drainage system. It is the responsibility of the RN to maintain and monitor the chest tube drainage system, and the color and amount of the drainage. The RN should have blood available before decompression and be prepared to autotransfuse the blood.

### Table 3. Indications for exploratory thora-cotomy*

<table>
<thead>
<tr>
<th>Condition</th>
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<tr>
<td>&gt;1500 mL of blood evacuated after initial chest tube insertion</td>
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<tr>
<td>&gt;200 mL/hour for ≥4 consecutive hours</td>
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<tr>
<td>&gt;150 mL/hour for 3 hours for the elderly</td>
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<tr>
<td>Hemodynamically instability systolic blood pressure &lt; 80 mm Hg despite aggressive blood/volume resuscitation</td>
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<tr>
<td>At least 2 functioning chest tubes are in place and signs of exsanguination occurs</td>
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*From Powell et al and Sherwood and Hartsock.

In the case of a massive hemothorax, intubate the patient. Shock is a compelling indication for intubation. A chest tube is also placed, and urgent thoracotomy is performed. There are 5 indications for performing an urgent thoracotomy (Table 3) when there are signs of exsanguination. However, if it is greater than 1500 to 2000 mL, or continues to bleed more than 100 to 200 mL per hour, then an exploratory thoracotomy must be performed to find the source of hemorrhage and to remove clots from the mediastinum. Surgery is emergent in these patients because insertion of a chest tube drainage system prevents any tamponade of the hemorrhage and rapid exsanguination is possible.

A chest tube was inserted at the fourth intercostal space with the immediate evacuation of 1600 mL of blood. The patient regained symmetric expansion of his chest wall and his tidal volumes doubled on the ventilator. His BP increased to 70/50 mm Hg. Transfusion therapy was initiated according to hospital protocol. The nurse assisted in transporting the patient to the operating room (OR) for an emergency thoracotomy.

Almost simultaneously, the third passenger arrived in the ED. He too was unrestrained in the sports utility vehicle and arrived in respiratory distress. Prehospital providers initiated 100% O₂ by nonrebreather facemask. Oxyhemoglobin...
saturation measured 89%, respiratory rate was 38 bpm, heart rate was 120 bpm, and BP was 80/60 mm Hg. Examination also revealed asymmetrical chest wall movement, labored breathing, and absent breath sounds on the right side. He was immediately intubated and bag ventilated. Chest x-ray showed a right pneumothorax with near complete collapse of the right lung.

PNEUMOTHORAX

Pneumothoraces in blunt thoracic trauma are most frequently caused when a fractured rib penetrates lung parenchyma. But it can also be caused by deceleration or barotrauma without associated rib fractures. The loss of negative intrapleural pressure results in the partial, or total, collapse of the lung, with a resulting accumulation of air in the pleural space. Typically, a loss of more than 40% lung volume because of a pneumothorax precipitates respiratory distress in the patient unless there is preexisting lung disease, in which case an even smaller loss in lung volume may not be tolerated. Most often, intrapleural air leak is self-limiting because the progressive collapse and decreasing ventilation of the affected lung seal the leak. In other cases, the lung may collapse completely.

Patients will present with respiratory distress, including dyspnea, tachypnea, and tachycardia. Decreased, or absent, breath sounds can be heard over the affected area with hyper resonance to percussion if the pneumothorax is large. Patients may often complain of chest pain, but it may not develop for hours. Chest x-ray demonstrates a pleural stripe fallen away from the chest wall, with absence of lung markings beyond the stripe. Small pneumothoraces may not be visible with x-ray. A small pneumothorax (less than 1-2 cm) can be observed with follow-up x-rays, taken within 6 to 8 hours, and by monitoring the patient’s respiratory status, and often, needs no further treatment. Larger pneumothoraces require the insertion of a chest tube. The classic chest tube insertion site at the second intercostals space is a less optimal site for trauma patients whose injuries are typically a combination of pneumothorax and hemothorax and require a more distal site.

As the x-ray technician removed the film from under the patient’s back, she noticed an approximately 3-cm small wound on his right posterolateral chest wall with bubbles around the site and an apparent “sucking sound.” Suspecting this was the cause of his pneumothorax, the nurse prepared an occlusive dressing and prepared for chest tube insertion.

OPEN PNEUMOTHORAX

Open pneumothorax is caused when a penetrating chest trauma opens the pleural space to the atmosphere, leading to a collapsed lung and a sucking chest wound. Historically described as early as the 13th century, open pneumothorax is a common combat injury, but in civilian life, it is most likely caused by penetrating injury or impalement. Injuries where the wound is greater than two third of the diameter of the trachea, the air will preferentially enter the wound during respiration, thereby, inhibiting normal ventilation, and lead to profound hypoventilation and hypoxia. The increased intrathoracic pressure can also cause the mediastinum to shift to the opposite side. This, in combination with reduced venous return, can lead to decreased cardiac performance and hemodynamic instability.

Signs and symptoms are proportional to the size of the defect. Signs and symptoms include, but are not limited to, visible defects, restlessness, dyspnea, tachypnea, cyanosis, asymmetrical chest expansion, hypoxia, and reduced venous return. Respiratory distress is a common finding with these patients, as is decreased breath sounds on the side of the injury. The presence of a thoracic injury with apparent sucking action and possibly gas bubbles at the wound site is most obvious. It is important to note that wound appearance can be deceiving. A large, menacing-looking wound may prove superficial, whereas a small, otherwise unassuming wound may
actually be responsible for the patient's injuries.

Treatment is aimed at returning normal ventilation and closing the wound. The first step is to assure an adequate airway, and intubate, if necessary. Then, locate the wound and place a sterile occlusive dressing over it to promote normal ventilation. A standard method involves placing a nonporous dressing (eg, petroleum-impregnated dressing) over the wound and taping it on 3 sides. This acts as a 1-way valve, allowing air to escape during expiration but becomes occlusive during inspiration. The RN must monitor the patient carefully for signs of a developing pneumothorax, such as hypotension, respiratory difficulty, decreased pulse pressure, hypoxia, and jugular venous distention (JVD). To help prevent this, a chest tube is inserted at another site to treat the pneumothorax after the dressing is applied. These patients are often intubated, and mechanically ventilated, to ensure control over ventilation. The wound should be cleaned and debrided prior to simple closure. Surgical exploration and closure may be required for larger wounds.

The patient was placed slightly on his left side and supported with pillows to visualize the wound better and to facilitate the insertion of a chest tube. The nurse placed a dressing over the site and taped it on 3 sides while waiting for the physician to return to insert the chest tube. Inadvertently, the patient rolled onto his back again while the nurse documented his assessment. Suddenly, the high-pressure alarm sounded on the ventilator. The RN noticed that the patient's BP dropped to 60/45 mm Hg. Further assessment revealed distended neck veins. The dressing had become occlusive because the surrounding pillows prevented air from escaping. The nurse suspected that his patient had developed a tension pneumothorax.

TENSION PNEUMOTHORAX

Tension pneumothorax is a life-threatening situation that requires immediate recognition and treatment. It is the accumulation of air, or blood, in the pleural space without an exit, causing an increase of intrathoracic pressure, and compression of the great vessels, lung, heart, and trachea. The results of this compression are a failure to ventilate and decreased cardiac output. This injury may be an immediate result of the primary trauma, a delayed complication from an undetected injury, or an undesirable result from treatment such as mechanical ventilation or an inadvertently clamped chest tube. Patients will exhibit signs of shock, including hypotension and decreased cardiac output, and severe respiratory distress due to compression of the heart and lungs. Asymmetric chest wall movement may be visible, as well as distended neck veins. Tracheal deviation is a classic sign due to increased intrathoracic pressure causing mediastinal shift, but may be difficult to detect in intubated patients. Patients may also become cyanotic from prolonged hypoxia.

Treatment is aimed at decompressing the chest to release the trapped air or blood. Supplemental oxygen is provided, and immediate decompression is accomplished by inserting a 12- or 14-gauge angiocatheter into the second intercostal space in the midclavicular line of the affected hemithorax. As air or blood is released, ventilation should improve, and the cause of injury should then be investigated and treated appropriately. A chest tube is inserted in the fourth intercostal space in the midaxillary line, to allow lung re-expansion, and to prevent further episodes. Pain management and pulmonary toileting are also initiated at this point. It is imperative for the RN to closely monitor patients who are at high risk for tension pneumothorax (Table 4) to quickly recognize and treat the patient if the situation arises.

The dressing was released on one side to relieve the pressure in his chest cavity and the patient's BP returned to baseline. A chest tube was inserted at a more lateral site and the patient was transported to the OR for wound debridement and closure.
Table 4. At risk for developing a tension pneumothorax*

<table>
<thead>
<tr>
<th>Inadequately resolved pneumothorax</th>
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<tbody>
<tr>
<td>Bronchial tear</td>
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<tr>
<td>Lung contusion</td>
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<tr>
<td>Pulmonary cyst</td>
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<tr>
<td>Use of moderate to high levels of positive end-expiratory pressure</td>
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</tbody>
</table>

*From Sherwood and Hartsock.

CARDIAC TAMPONADE

Cardiac tamponade is a life-threatening, slow, or rapid, compression of the heart, which eventually prevents the heart from beating. It is caused by the accumulation of blood and blood clots, compressing and preventing the heart from filling with blood with each contraction. Constriction of the heart leads to a decrease in cardiac output, perfusion, and eventually venous return. Without intervention, the heart is unable to fill and/or pump effectively, and cardiac arrest occurs. The leading cause (80%-90%) of cardiac tamponade in trauma patients is penetrating injuries such as stab wounds, bullets, or rib fractures, which produce lacerations of the pericardium that seal from fatty tissues or by the formation of clots. Once the pericardium is sealed, blood continues to collect in the pericardium, setting the stage for tamponade. The size of the perforation, and the amount of blood collecting in the pericardium, directly affects the mortality and morbidity of the patient. Because the pericardium is inflexible, small changes in volume can have considerable effects on the pressure within the pericardium; as little as 50 mL of blood can decrease cardiac output. Tamponade also occurs in about 2% of patients with penetrating trauma to the chest and lower abdomen, and occurs in 10% of blunt chest trauma injuries. In a recent review, the most common site of cardiac penetration was the right ventricle (43%) because of its maximal anterior exposure. The second most common site is the left ventricle (33%), followed by the right atrium (15%), and then the left atrium (6%).

Clinical presentation of cardiac tamponade can be hidden by, or misdiagnosed as cardiogenic shock (tachycardia and hypotension with cold extremities due to vasoconstriction), and the diagnosis may initially be subtle. Classic signs of tamponade include JVD due to increased venous pressure or lack of cardiac filling. Although classic to tamponade, JVD still may not be noted because of profound hypotension. Especially in rapid tamponade, JVD is not present because of insufficient time for blood volume to increase, once again attributable to hypotension.

Other, more common, signs are known as Beck’s triad. Beck’s triad is a complex of 3 associated symptoms that are classic to the diagnosis of cardiac tamponade. These signs include increased central venous pressures, widening pulse pressure, and muffled heart tones. These could be hard to recognize, or may be completely absent, because of hypotension. Since tamponade can be masked by other symptoms or injuries, it should be ruled out in any patient who has obvious penetrating trauma to the chest and abdomen, or if blunt trauma is suspected.

Other, less specific, signs that are common in tamponade include chest discomfort, pleuritic pain, tachypnea, and dyspnea on exertion. Another key sign is called pulsus paradoxus, an inspiratory systolic fall in arterial pressure of 10 mm Hg or more during normal breathing. Pulsus paradoxus is caused by a fall in cardiac output, resulting from increased negative intrathoracic pressure during inhalation. If the patient has an arterial line, pulsus paradoxus is very well noted, and is also called pulsus alternans, where every other arterial pulsation is weak. Kussmaul’s sign is a rise in venous pressure with inspiration when breathing spontaneously, and is a true paradoxical venous pressure abnormality associated with tamponade. This classic sign is rarely seen.

Diagnosis of tamponade is not easy. An echocardiogram is most useful in the diagnosis of tamponade, and is considered the
most readily available and efficient tool, if timing permits. Echocardiogram may characteristically demonstrate right atrium or ventricle chamber collapse.

Treatment of cardiac tamponade hinges on aggressive airway control, oxygenation, circulatory support, and rapid transport to a trauma center for definitive treatment. Rapid infusion of IV fluids will increase venous pressure and improve cardiac output. It also allows time for needed interventions. The ultimate treatment of tamponade is drainage or removal of blood and clots from the pericardial sac, preferably by needle paracentesis guided by echocardiography, fluoroscopy, or computerized tomography (CT). The needle tip would be evident on imaging, which helps ensure safety when penetrating the pericardium. Surgical drainage is desirable in patients with intrapericardial bleeding, and in those with clotted hemopericardium. Trauma patients who undergo a pericardiocentesis will usually require surgical inspection, and possible repair, of the heart. Medical treatment of tamponade remains controversial, but should include inotropic support, as well as supporting the body’s compensatory mechanisms to reduce the elevated vascular resistance. Cardiac tamponade is a life-threatening emergency, and removal of blood from the pericardium is the only intervention that will sustain life.

Nursing management of a patient with cardiac tamponade would include ventilatory support/airway protection, assistance with emergent pericardiocentesis, if indicated, and hemodynamic support, including fluid resuscitation. The patient would most likely be recovered and monitored in the intensive care unit (ICU) for several days, and then transferred to a telemetry floor for further monitoring before discharge. Patients would be discharged home with individualized follow-up instructions if they are without complications during hospitalization.

Prognosis of tamponade is excellent as long as the trauma patient is treated promptly, before hypovolemic or cardiogenic shock occurs, or pulseless electrical activity. Pulseless electrical activity caused by tamponade in trauma patients carries a poor prognosis and requires critical thinking for recognition and immediate life-saving interventions.

AORTIC DISRUPTION

Aortic disruption, resulting from blunt trauma, is the leading cause of immediate death. Disrupting the blood flow in the aorta impedes perfusion to vital organs and to extremities. Depending upon where the disruption is in the aorta, the size, or entirety, of the disruption determines the significance of the prognosis. Disruption of the aorta is any interference within the aorta, such as a small tear, laceration, occlusion, or complete rupture. An occlusion of the aorta carries a good prognosis if it is diagnosed quickly and interventions are immediate. Each year, 5000 to 8000 people in the United States die as a result of aortic or great vessel rupture.

The most common mechanism is rapid deceleration from a high-speed impact such as that experienced by the patients whose case study is described in this article. Rapid deceleration can cause shearing forces on the aorta and other great vessels. Depending on the degree of force, it can cause either partial or complete tears. If the tear is complete, it is usually fatal at the scene. Patients who survive the initial injury have a high likelihood of survival if the disruption is identified and treated rapidly. If the injury is not identified, one third of the patients die within the first 6 hours.

The most common site of tears or rupture in the aorta is in the descending portion distal to the left subclavian artery at the ligamentum arteriosum. The descending aorta, which is fixed to the vertebrae, decelerates at the same rate as the body would decelerate in a high-speed accident. The heart and the aortic arch often continue to move laterally and inferiorly into the left hemithorax. The resultant force is a combination of shearing, torsion, and bending that produces maximal stress. There are also 2 other sites in the ascending aorta where rupture occurs: where the aorta leaves the pericardial sac and at the
entry to the diaphragm. Other less common mechanisms include falls from a great height or sport-related impacts (ie, football, hockey).

The clinical presentation of a disrupted aorta can be very specific, but some patients remain symptom free. In those cases, critical thinking doctors and nurses are required to recognize it before too much time has elapsed. Up to 50% of patients will have no external signs of chest trauma, so the single most important factor in establishing the diagnosis is a high index of suspicion based on mechanism of injury.

Possible signs include a pulse deficit in any area, particularly lower extremities or the left arm, hypotension, unexplained by other injuries, or hypertension of the upper extremities compared with the lower extremities. The patient may experience sternal or posterior interscapular pain and may have dyspnea or respiratory distress. Auscultation of a precordial or interscapular systolic murmur is also very specific. Depending on the location of the disruption, the patient may have hoarseness, attributable to a hematoma causing pressure around the aortic arch or complete lower extremity paralysis.

Diagnostics include plain film x-ray, which would show a widened mediastinum (>8 cm) or an obscured aortic knob. Other findings that are suggestive of an aortic disruption are left apical pleural cap, tracheal deviation to the right, and elevation and rightward deviation of the right mainstem bronchus. A CT scan is done, particularly if the CXR is questionable, but its overall accuracy is approximately 53%. Aortography remains the gold standard for detecting aortic injuries. Aortography allows visualization of the aorta and any tears, aneurysms, occlusions, or hematomas that may be present. Ultimately, the disruption will require surgical intervention, but until the patient is ready for surgery, the major focus is to keep the blood pressure in a specified range, usually less than 90 mm Hg. Blood pressure is of utmost importance. If pressure is too high, it could cause the aorta to completely rupture and the patient to exsanguinate.

Nursing management includes airway protection, intubation, oxygen delivery, and careful administration of fluids. Hypotension is acceptable as long as the patient can tolerate it. Stimulation is reduced; pain control is a top priority to aid in keeping BP low. Continuous monitoring for hemodynamic stability is key.

Prognosis completely depends on early recognition and prompt treatment, but many patients who survive the operative period still die within the first week after injury. Postinjury complications that require monitoring include hypertension, paraplegia, bowel ischemia, renal failure, graft leaks, and infection. It is important for the nurse to remember that any organ system below the level of disruption may be damaged during the period of hypoperfusion. Renal and bowel function need to be monitored. Blood pressure needs to be kept low to aid in healing of new grafts and to prevent rebleeding. The patient’s rehabilitative plan will need to be tailored to his or her particular disabilities, whether permanent or temporary.

Two young men were involved in a high-speed MVC during which their vehicle plunged off an interstate freeway, down a 20-ft embankment, across a second highway, and then crashed into a retaining wall. Per paramedics, there was considerable vehicle damage. Both airbags deployed.

The driver R.G., reportedly unrestrained, arrived at the trauma center, awake, alert, and cooperative. He denied pain, but did complain of thirst. His vital signs were stable. Both primary and secondary examinations failed to reveal any significant injuries. However, the history of a high-speed crash, coupled with possible ejection, raised the level of suspicion for a potentially serious or a life-threatening injury. Per protocol, a portable CXR was obtained in the trauma resuscitation room. The CXR image demonstrated a widened mediastinum. The trauma surgeon ordered a CT scan of the chest. The 16 array helical CT scanner images demonstrated a traumatic transection of the descending thoracic aorta.
The patient was taken to the OR emergently for thoracotomy and aortic repair. Postoperatively, he was taken to the surgical ICU, intubated on ventilator support, with chest tubes, an arterial line for BP monitoring, central venous pressure catheter, and a pulmonary artery catheter. He spent 4 days in the ICU for careful monitoring and BP control. He continued to progress on the surgical ward. He went home postinjury day 7 in good condition.

The passenger E.L. arrived awake and alert, complaining of mild tenderness to his chest. His chief complaint was left lower extremity pain. His vital signs were stable. Primary examination revealed no life-threatening injuries. Secondary examination revealed a gross deformity of his left hip, left knee, and left ankle. Despite the extremity injuries, E.L. had a good pulse as well as movement. Chest x-ray demonstrated a widened mediastinum. Again, secondary to the high-energy event of a high-speed MVC, there was suspicion for aortic injury. CT scan confirmed a mid-descending thoracic aortic tear. The patient remained hemodynamically stable, but was taken to the OR emergently for aortic repair, followed by orthopedic fixation of his lower extremity injuries.

The patient tolerated the surgeries well and recovered uneventfully in the ICU for 3 days. He was discharged 5 days later without complications, with instructions to follow-up with the trauma service in the clinic.

**MYOCARDIAL CONTUSION**

Myocardial contusion, unlike concussion, results in actual histopathological changes. Upon trauma to the myocardium, cellular injury occurs with extravasation of erythrocytes into the muscle wall, along with necrotic areas of myocardial fibers, myocardial edema, interstitial, and subendocardial hemorrhage. With or without necrosis, or even contusion, it is believed that injured myocardial cells contract less forcefully, contributing to diminished cardiac output. The reported incidence of myocardial contusion ranges between 3% and 75% in studies of blunt chest trauma. High-speed deceleration collisions are the most common cause, but speeds less than 35 mph have also contributed. Myocardial contusion should be suspected in all patients involved in MVCs of 20 mph or greater, especially if there is damage to the steering wheel or the patient complains of chest pain. It is estimated that at least 20% of patients with steering wheel impact sustain cardiac contusions, 16% of which are fatal. The most frequent scenario is an unrestrained driver in a high-speed crash, hitting the steering wheel with his chest. Other, less common, mechanisms include falls from a height and direct blows to the chest, particularly in sport-related incidents. Sometimes, direct trauma to the abdomen has been found to generate enough upward force into the chest cavity to produce blunt cardiac injury. The clinical significance of cardiac contusion is directly related to its complications and associated injuries, including dysrhythmia, traumatic myocardial infarction, thromboembolus, reduced left ventricle ejection fraction and/or cardiac output, valvular injuries, congestive heart failure, ventricular aneurysm, pericardial effusion, coronary artery laceration, and cardiac rupture.

Clinical presentation of myocardial contusion is the presence of dysrhythmias. Sinus tachycardia is the most common dysrhythmia. Other dysrhythmias include premature atrial contractions, atrial fibrillations, right bundle branch block, ST elevation and T-wave flattening, and premature ventricular contractions. Dysrhythmias in a chest trauma patient should always indicate cardiac contusion, until proven otherwise. If evidence of external chest trauma (such as fractures, the imprint of a steering wheel, or complaints of angina-like pain despite the absence of dysrhythmias) exists, one should suspect cardiac contusion. Tenderness, ecchymosis, or swelling of the chest wall are also diagnostic. Other findings that may suggest contusion include chest pain (usually unrelieved by nitrates), auscultation of a pericardial friction rub, or an S3 gallop. But once again, myocardial contusion may be
unrecognized and may only be detected when serious complications develop.

Diagnosis of a cardiac contusion relies mainly on an admission electrocardiogram (ECG). As previously stated, if cardiac/chest trauma is even suspected, the patient must be ruled out for cardiac contusion. If an admission ECG is abnormal, the patient should be admitted for continuous heart monitoring and a follow-up ECG 24 to 48 hours later. Most arrhythmias usually occur within that time frame. An echocardiogram, CK and CKMB, and troponin levels are frequently assessed as a secondary tool once an abnormal ECG has been obtained. CK and CKMB are nonspecific to myocardial injury and are usually elevated in trauma patients because of skeletal muscle injury. Associated with elevated troponin levels, they depict a cardiac event of some sort. Troponin measures myocardial contractile proteins (not found in skeletal muscle). They are released into circulation only after loss of membrane integrity. The latest studies show that positive cardiac enzymes offer the highest accuracy in the diagnosis of cardiac injury. However, there is no one diagnostic method that is unanimously accepted at this time.

Treatment of cardiac contusion is still very controversial, as are the related diagnostics. It is recommended the patient be monitored continuously for symptomatic arrhythmias, especially ventricular irritability or conduction defects. If arrhythmias occur, then the patient is treated per the algorithms in advanced cardiac life support.

Prognosis of cardiac contusion is very promising. Despite the fact that the injury is usually not clinically significant, complications can occur. Patients older than 50 years and those with preexisting cardiac disease are at higher risk for significant cardiac complications following blunt chest trauma. Without serious complications, or any major associated injuries, there is complete recovery.

**MYOCARDIAL RUPTURE**

Myocardial rupture is an acute traumatic tear of the ventricles, atria, intraventricular septum, intraatrial septum, chordae, papillary muscles, or valves. Rupture of the myocardium is rare, but usually fatal. Patients who survive rupture of the heart usually have sustained either right- or left-sided atrial rupture. Mortality rates range from 50% to 85%. Most survivors of myocardial rupture survive because of cardiac tamponade.

Myocardial rupture occurs when blood-filled chambers are compressed with enough force to generate a tear in the chamber wall or septum, or to rupture a cardiac valve. Rupture of the heart is usually caused by blunt chest trauma from an MVC or a fall. Just as a contusion of the heart occurs, sufficient force can completely rupture the heart depending upon the phase of the cardiac cycle at the time of impact.

Clinical signs include all signs of cardiac tamponade and hypovolemic shock due to extreme hemorrhaging. Patients may also have evidence of sternal fracture, as well as numerous rib fractures, including the first rib. Tamponade symptoms include muffled heart tones, JVD due to increased venous pressure, hypotension unrelieved by fluid resuscitation, tachycardia, and arrhythmias. The patient may also be cyanotic from the upper chest to the head.

Treatment of myocardial rupture is immediate surgical intervention to repair the heart. One third of patients quickly exsanguinate and the remaining two thirds have an intact pericardium, which ends up protecting them from immediate exsanguination. Consequently, the only survivors paramedics would likely encounter are those with cardiac tamponade.

**TRACHEOBRONCHIAL DISRUPTION**

Traumatic disruption of the tracheobronchial tree is a rare, but potentially life-threatening, injury caused by blunt or penetrating trauma. Blunt injury occurs in about 1% of cases. This is due to large amount of thoracic bony protection surrounding the major airway structures. Direct trauma to the trachea, including the mainstem bronchus,
may be blunt or penetrating. Injury to a major bronchus is an unusual, and fatal, injury that is frequently overlooked. The injury may occur at any level, but the majority of such injuries result from blunt trauma, and occur within an inch of the carina. In blunt trauma, the impact creates a sudden increased pressure in the airway against the glottis. Approximately 80% of patients with major airway injuries die at the scene as a result of asphyxia due to interruption or airway obstruction compounded by aspiration of blood and intrapulmonary hemorrhage.\textsuperscript{13} Thirty percent of the remaining patients who reach the hospital alive do not often survive because of associated injuries.

The incidence of tracheobronchial injuries is 3%, with a mortality rate of 30%; 50% of whom die within the first hour.\textsuperscript{4} Tracheal injuries are devastating and are frequently caused by severe rapid deceleration injuries or compressive forces. Traction is produced during deceleration and results in the lung being pulled away from the mediastinum. When the amount of traction overcomes the elasticity of the trachea, the trachea ruptures.\textsuperscript{4}

Three fourths of all tracheobronchial injuries in penetrating trauma involve the cervical trachea, and one fourth involve the thoracic and bronchus. Cervical tracheal injuries present with upper airway obstruction, unrelieved by oxygen, and cyanosis. Symptoms may include local pain, dysphagia, hemoptysis, subcutaneous emphysema, and cough. Bronchial injuries frequently present with hemoptysis, subcutaneous emphysema, or tension pneumothorax with a mediastinal shift.

Eighty percent of major bronchial injuries occur within 2 cm of the carina.\textsuperscript{14} Tracheobronchial injuries often leak into the pleural space, mediastinum, and/or the lung parenchyma. An intrapleural lesion results in a massive air leak, and is the most devastating of the tracheobronchial injuries. Clinical signs of a mediastinal rupture are pneumomediastinum and subcutaneous emphysema. Intraparenchymal injuries usually seal spontaneously if the lung is adequately expanded.

Penetrating trauma is overt and is often associated with esophageal, carotid artery, and jugular vein trauma and requires immediate surgical repair.

Clinical findings depend on the type and extent of the injury. Penetrating wounds to the neck or chest usually present with an associated air leak, such as subcutaneous emphysema, or bleeding, such as hemoptysis. Penetrating injuries to the tracheobronchial tree are usually associated with major intrathoracic vascular, esophageal, and/or pulmonary injuries. This can usually be detected if the patient presents with a pneumothorax, and is relieved by chest tube insertion. The presence of a large air leak, which persists and requires multiple chest tubes to expand the lung, should alert you to the diagnosis. The patient must undergo immediate bronchoscopy to detect the bronchial tear.

Patients with severe blunt trauma must be examined with a high index of suspicion, especially when the first 3 ribs are involved. Fractures of the larynx are a rare injury and are indicated by the following triad: (1) hoarseness, (2) subcutaneous emphysema, and (3) palpable fracture crepitus. Other signs of tracheobronchial disruption are dyspnea, cough, hemoptysis, sternal tenderness, and noisy breathing. Hamman’s sign (crunching sound during systole) may be heard on auscultation of the heart because of air in the mediastinum.\textsuperscript{4} The patient may also present in severe respiratory distress or with total airway obstruction. Observations of labored respiratory effort may be the only clue to tracheobronchial injury and/or airway obstruction. Noisy breathing indicates partial airway obstruction that suddenly may become complete and the absence of breathing suggests that complete obstruction already exists. A persistent pneumothorax despite a functioning chest tube is an important clue to a tracheobronchial injury.\textsuperscript{14}

Ninety percent of patients will have an abnormal admission x-ray.\textsuperscript{1} Chest radiograph may reveal subcutaneous or mediastinal emphysema, pleural effusion, pneumothorax, or fractures of ipsilateral ribs 1 through 5, and
mediastinal hematoma. More specific signs are peribronchial air, deep cervical emphysema, and dropped lung (lung apex rests at the level of hilum). Stabilization of the airway is of utmost importance. Treatment of tracheobronchial injuries may require only airway maintenance until the acute inflammatory and edema processes resolve. Fiberoptic bronchoscopy may also be helpful in diagnosing an injury or facilitating airway placement. If a distal injury is present, intubation can best be accomplished by visualization of the bronchi with a flexible bronchoscope, and then passing an endotracheal tube over the scope. If the patient is stable, administer oxygen at 100% and perform an immediate bronchoscopy.

An attempt to intubate is warranted if the patient’s airway is totally obstructed or if the patient is in severe respiratory distress. Place an endotracheal tube into the mainstem bronchus of the uninjured side to improve ventilation of the uninjured lung. An emergent tracheostomy must be performed if the airway is obstructed, secondary to a ruptured cervical trachea, or intubation was unsuccessful. This is usually followed by operative repair.

DIAPHRAGMATIC RUPTURE

Approximately 0.8% to 1.6% of patients with blunt trauma have a rupture. Blunt trauma accounts for 75% of cases while penetrating trauma accounts for 25% of cases. Since no distinctive signs and symptoms are associated with penetrating diaphragmatic injuries, a high index of suspicion is required for diagnosis.

The left side of the diaphragm is more prone to injury (65%-85% of cases) because it is not protected by the liver. The left postero-lateral portion of the diaphragm is the weakest, and therefore, the most common site of rupture and herniation. The size of the disruption is determined by the mechanism of injury. In blunt trauma, the defect in the diaphragm is typically between 5 and 15 cm. In penetrating trauma, the defect is usually between 3 and 2.6 cm. A rupture or tear of the diaphragm may allow herniation of abdominal contents, such as the stomach, small bowel, or spleen, into the thorax. Herniation may result in respiratory compromise because of impairment of lung capacity, and displacement of normal lung tissue. Penetrating injuries below the nipple line should be evaluated for the potential of diaphragmatic injury, and concurrent abdominal stab wounds to the lateral chest walls and flanks can be associated with diaphragmatic lacerations. Delay in diagnosis is common because of attention to associated injuries and because many ruptures are not recognized on CXR or CT.

The clinical picture depends on the size and site of injury, the presence of the herniation, and associated injuries. Diaphragmatic injuries are difficult to diagnose at first because bleeding is minimal and the patient is often asymptomatic. Physical examination does not reveal diaphragmatic lacerations unless associated injuries exist. In the acute phase, patients may demonstrate decreased breath sounds on the side of the injury and bowel sounds may be heard in the chest. The latent phase is characterized by intermittent visceral herniation of any abdominal structure through the defect. In the latent phase, patients complain of belching, nausea, vomiting, and vague abdominal pain. Common organs that herniate include the liver on the right and the colon, stomach, small bowel, and spleen on the left. Tension viscerothorax is a complication of diaphragmatic rupture; usually the stomach herniates and undergoes volvulus, massively dilates, and causes left lung collapse and mediastinal shift to the right.

Clinical findings include marked respiratory distress, dyspnea (decreased breath sounds on the affected side), palpation of abdominal contents upon insertion of a chest tube, auscultation of bowel sounds in the chest, and paradoxical movement of the abdomen with breathing.

Chest x-rays are usually normal or nonspecific unless a substantial injury exists. In blunt trauma, CXRs are diagnostic initially in only 25% to 50% of cases. The presence of a
nasogastric tube within the chest or the presence of abdominal viscera denotes a perforated diaphragm. Diaphragmatic injuries are not clearly identified, or are missed initially, if the chest film is misinterpreted as showing an elevated left diaphragm, acute gastric dilatation, loculated pneumo-hemothorax, or a subpulmonary hematoma. The following studies\textsuperscript{11} may help in diagnosing diaphragmatic injury:

- Chest radiograph may reveal blurring of the diaphragm, arched diaphragmatic shadow, bowel or extraneous bubbles in the chest, nasogastric tube in the left chest.
- Ultrasound is used to visualize large disruptions or herniation. However, it may miss small tears from penetrating injuries.
- Computerized tomography, with fine cuts through the diaphragm, may detect small ruptures of the diaphragm.
- Magnetic resonance imaging may aid in the diagnosis because it can accurately visualize the diaphragm's anatomy.

Diagnostic procedures

- Diagnostic peritoneal lavage—peritoneal lavage in the presence of diaphragmatic rupture may be falsely negative as much as 25\% of the time.\textsuperscript{11} It is still the most sensitive test for detecting diaphragmatic injury in penetrating lower thoracic trauma (red blood cell [RBC] threshold of 5000 or 10,000 RBC/mm\textsuperscript{3}). In blunt trauma, the standard RBC threshold is 100,000 RBC/mm\textsuperscript{3}. Diagnostic peritoneal lavage is insensitive to diaphragmatic injury at this threshold.\textsuperscript{9} The obvious need for laparotomy is an absolute contraindication to diagnostic peritoneal lavage. Relative contraindications include morbid obesity, a history of multiple abdominal surgeries, and pregnancy.
- Diagnosis can be made by return of lavage fluid from a thoracostomy tube.\textsuperscript{4}

General supportive measures are the mainstay of medical therapy. A confirmed diagnosis, or the suggestion of blunt diaphragmatic injury, is an indication for surgery. While surgery is the only definitive treatment, placement of a nasogastric or orogastric tube can be done to decrease the amount of bowel gas and limit the hernia's size, in addition to decreasing the ileus and reducing the obstruction. Blunt diaphragmatic injuries typically produce large tears measuring 5 to 10 cm or longer. Persons with traumatic hernias frequently have concomitant injuries who require emergent exploration. Surgical repair is necessary, even for small tears, because any defect will not heal spontaneously.

Complications may occur in up to 64\% of patients with blunt diaphragmatic injury and include intra-abdominal abscess and pneumonia.\textsuperscript{10} Paralysis of the diaphragm is common. The late complications of an undiagnosed traumatic hernia include all of the following: bowel herniation, incarceration, and strangulation; tension hemithorax secondary to massive bowel herniation; pericardial tamponade from herniation into the pericardial sac; and diaphragmatic paralysis that may recover after repair.\textsuperscript{16}

Early deaths usually are a result of associated injuries, not due to the diaphragmatic injury. There is a 25\% to 40\% mortality rate associated with diaphragmatic tears, primarily because of the severity of associated injuries.\textsuperscript{14}

ESOPHAGEAL INJURY

Esophageal rupture is the most rapidly fatal injury. Mortality rate is 18\% and increases significantly if the diagnosis is delayed or missed. Death is imminent if diagnosis is delayed. Most esophageal injuries are caused by penetrating trauma. Cervical esophageal injury is more common than thoracic esophagus. Blunt injury is rare (0.1\% incidence),\textsuperscript{1} but is lethal if unrecognized. It is usually caused by a forceful injection of gastric contents into the esophagus from a severe blow to the upper abdomen, resulting in a linear tear in the lower esophagus allowing leakage into the mediastinum. The esophagus has no serosal covering and any perforation results in direct drainage into the mediastinum.\textsuperscript{4} Contaminated material is pulled into the pleural space by negative intrathoracic pressure and results
in subcutaneous or mediastinal emphysema, pleural effusion, retro esophageal air, and unexplained fever within 24 hours of injury.

Esophageal injuries should be considered in any patient who presents with a hemothorax or pneumothorax without any rib fractures, or in any patient who sustains a severe blow to the lower sternum or epigastrium, who presents with pain and/or shock out of proportion to the clinical findings.\(^4\)

Clinical symptoms are subtle and vague, and vary according to the site of rupture and the degree of contamination. Substernal pleuritic chest pain, with radiation to the neck or shoulders, is the most common symptom of esophageal injury. Swallowing and neck flexion may cause pain. There are many other signs and symptoms depending on the location of the injury (Table 5).\(^7\)

Diagnosis requires correlation of the index of suspicion with physical findings, radiological studies, and fiber optic examination. Typical CXR findings are mediastinal emphysema, pneumothorax, hydrothorax, and/or widened mediastinum. Because of concomitant injuries, the CXR may not demonstrate any abnormalities and an esophagogram should be performed. The presence of mediastinal air, usually on the left side, is basis for diagnosis. Esophagoscopy or an esophagogram reveals the site of esophageal tear and whether it connects to one or both pleural spaces.\(^13\) Esophagoscopy is reliable in 60% of injuries, but with esophagoscopy combined with an esophagogram, esophageal injuries are detected 90% of the time.\(^4\) Recognizing injury to the esophagus is difficult because of its rarity, insufficient clinical signs in the initial 24 hours, and the presence of other injuries. Missed injuries and delay in treatment results in the rapid development of sepsis and associated high risk of death. Therefore, prompt aggressive investigation, including radiography, endoscopy, and thoracoscopy, must be performed.

Nursing management begins with attention to airway, ventilation, oxygenation, and circulatory support. The general treatment for any esophageal tear or perforation is suspension of all oral intake, placement of a nasogastric tube with continuous suction, and antibiotic therapy.\(^4\) A large-bore IV catheter should be placed for volume replacement and administration of IV medication therapy. Continuing

Table 5. Signs and symptoms according to location of esophageal injury*

<table>
<thead>
<tr>
<th>Injury</th>
<th>Signs and Symptoms</th>
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<tbody>
<tr>
<td>Cervical perforation</td>
<td>Crepitus</td>
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<tr>
<td>Cervical esophageal perforation</td>
<td>Cervical tenderness</td>
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<tr>
<td></td>
<td>Pain</td>
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<td></td>
<td>Resistance of passive range of motion of the neck</td>
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<td></td>
<td>Dyspnea</td>
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<td></td>
<td>Hoarseness</td>
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<td></td>
<td>Bleeding from mouth or nasogastric tube</td>
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<tr>
<td></td>
<td>Cough</td>
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<td></td>
<td>Stridor</td>
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<td></td>
<td>Neck crepitus</td>
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<tr>
<td></td>
<td>Mediastinal emphysema</td>
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<td></td>
<td>Mediastinitis</td>
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<tr>
<td>Intrathoracic perforation</td>
<td>Peritoneal irritation</td>
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<tr>
<td>Abdominal esophageal tear</td>
<td>Dyspnea</td>
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<tr>
<td></td>
<td>Pleuritic pain</td>
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*From Mason.\(^7\)
monitoring for signs of peritonitis and respiratory insufficiency is necessary.

If pneumothorax or hydrothorax is present, then a chest tube is placed. Medical management is gastric decompression, antibiotic therapy, combined with operative repair consisting of bypassing the affected region, and drainage of the pleural space.13

SUMMARY

Major thoracic injuries are known as the "Deadly Dozen." Each of these injuries may present as immediate life threatening or potentially life threatening. The potentially life-threatening nature of these injuries demands an organized approach by skilled and knowledgeable physicians, nurses, and therapists. Applications of certain fundamental principles of initial trauma management must be applied. Assessment should be based on a high index of suspicion and a thorough knowledge of pathophysiology is important for optimal treatment. Recognizing the signs and symptoms of these traumatic injuries as well as accurately diagnosing and managing these injuries are crucial for better patient outcomes.

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