I. Introduction
In this chapter we will review of the anatomy of the thorax and discuss traumatic injuries to the chest wall and the thoracic viscera. Chest wall injuries include the skin, subcutaneous tissue, intercostal muscles, ribs, sternum, and parietal pleura. Thoracic visceral injuries include two main categories: (1) Mechanical injuries of the respiratory system, which will include diaphragmatic rupture. The inclusion of diaphragmatic rupture is due to the fact clinically it presents with symptoms analogous to pneumothorax; (2) Mechanical injuries of the cardiovascular system, which will include the mediastinum. This is primarily due to the fact the most common cause of a widen mediastinum is aortic rupture. We will also discuss the mechanisms of chest injury.

II. Musculoskeletal Anatomy of the Thorax

A. Overview: The thorax forms the upper part of the trunk, being separated from the lower part, the abdomen, by the diaphragm. The thorax consists of two parts, the musculoskeletal cage (rib and thorax cage) and an internal cavity that contains the lungs, heart, mediastinum, trachea, esophagus, thymus, vagus and phrenic nerves, the right and left sympathetic trunks, the thoracic duct and major systemic and pulmonary blood vessels (see Figs 1, 2 & 3).
Fig. 1. Anterior view of human skeleton. (Wiki)
Fig. 2. Posterior view of human skeleton. (Wiki)
Posteriorly, the thorax is formed by 12 thoracic vertebrae and the intervertebral disks and the posterior aspect of the ribs (see Fig. 4). Laterally, the chest wall is formed by 11 ribs and their respective intercostal muscles, having a convex shape (see Fig. 5). Anteriorly, the chest is slightly convex formed by the sternum, ribs and their costal cartilages (see Fig. 6). As previously indicated, the diaphragm forms the floor of the chest cavity, separating it from the abdominal cavity.
Fig. 4. Posterior view of the ribs and articulating vertebrae. The inferior thoracic aperture is visible at the bottom. The inferior aperture is bounded by: the 12th thoracic vertebra posteriorly, 11th and 12th pairs of ribs laterally, costal cartilages of ribs 7 through 10 anteriorly, and the xiphisternal joint (i.e.: the articulation between the xiphoid process and the inferior border of the sternal body) anteriorly. (Wiki)
Fig. 5. Anterior view of the rib cage. (Wiki)
The superior border of the chest is represented by the superior thoracic aperture (thoracic inlet). On average it measures 5 cm anteroposterioly (front to back) and 10 cm transversely (side to side). Its upper most posterior border is the first thoracic border. The lateral border is formed by the medial aspect of the first ribs on each side. The anterior border is the manubrium of the sternum (see Fig. 7).

There is an inferior thoracic aperture (thoracic outlet), which unlike the well defined thoracic inlet, is large and irregular in outline. Its overall configuration is oblique due to the fact the posterior wall of the chest is much longer than the anterior wall (see Fig. 4). Structures passing through the inferior thoracic aperture between the thorax and abdomen include the inferior vena cava and esophagus, both of which pass through the
diaphragm, and the abdominal aorta and thoracic duct which pass posterior to the diaphragm through the aortic hiatus.

Fig. 7. The superior thoracic aperture is bounded by: the first thoracic vertebra (T1) posteriorly, the first pair of ribs laterally (more specifically, the first ribs form lateral C-shaped curves posterior to anterior), the costal cartilage of the first rib and the superior border of the manubrium anteriorly. (Wiki)

The posterior border of the inferior thoracic aperture is formed by the twelfth thoracic vertebral body. The twelfth rib and distal end of the eleventh rib form the posterolateral border. The distal cartilaginous ends of the seventh to tenth ribs, which unite and then
continues superiorly, form the anterolateral borders. The sloping costal cartilages of ribs seven to ten in front form the costal arch into which the xiphoid process of the sternum descends in the midline. The xiphoid process and xiphoidsternal junction form the anterior (front) border of the inferior thoracic aperture.

There is an anatomical plane that can further aid you in orienting the components of the thorax (see Figs. 8 & 9). This is the horizontal plane that passes through the intervertebral disc between the fourth and fifth thoracic vertebrae posteriorly and the

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Fig. 8. The above drawing depicts the various planes and their orientations of the human body. (Wiki)
Fig. 9. The above drawing depicts the various planes of the human body. (Wiki)

junction of the manubrium and sternum (angle of Louis/sternal angle) at the level of the second costal cartilage anteriorly. This horizontal plane separates the superior mediastinum from the inferior mediastinum and also defines the upper most anatomic position of the pericardium and pulmonary trunk, the beginning of the origin of the arch of the aorta, the level at which the ascending aorta ends and the descending aorta begins, the level at which the azygous vein arches over the roof of the right lung and opens into the superior vena cava, the level at which the pulmonary trunk divides into right and left pulmonary arteries, the thoracic duct crosses from right to left side and reaches the left side at the level of the sternal angle, the level at which the cardiac
plexus is situated, the level at which the trachea bifurcates into the right and left main bronchi, and the site where the superior vena cava penetrates the pericardium to enter the right atrium. It is located 5 cm below the suprasternal notch (see Fig. 10).

Fig. 10. Location of the angle of Louis/sternal angle at the level of the second costal cartilage and the third and fourth intercostal spaces. (Rev Esp Cardiol. 2008;61:1109-10, vol 61 Num 10 DO1:10.1016/S1885-5857(09)60020-7 Morphologies Suggestive of V₁ and V₂ Lead Misplacement. Javier Garcia-Niebla, Centro de Salud Valle del Golfo, El Hierro, Santa Cruz de Tenerife, Espana.

B. Ribs: There are typically twelve pairs of ribs. All ribs are attached (articulate) posteriorly with the vertebral column forming the greater part of the thoracic skeleton (see Figs. 2 & 4). On occasion you may find a patient with more or less than twelve pairs of ribs. Their number can be increased through the formation of cervical or lumbar ribs. Typically, when there is less than twelve pairs it is through the absence of the twelfth pair. The upper seven pair of ribs are all attached to the sternum through their costal cartilages; due to this connection with the sternum these ribs are called true ribs (see Figs. 1, 5 & 7). The costal cartilages of the eighth, ninth and tenth ribs join with the costal cartilages of the above ribs, hence they are called false ribs (see Figs. 1, 5 & 7). The eleventh and twelfth ribs are also false ribs, but since they have no connection with the sternum they are referred to as floating ribs (see Figs. 5, 6 & 7). Occasionally the
costal cartilage of the tenth rib may not join with the costal cartilage of the ninth rib, thus it may also be a floating rib. Depending upon the race this incidence varies between 35% to 70%.

The space between the ribs are referred to as intercostal spaces; they contain the intercostal muscles, nerves, arteries and associated veins and lymphatics (see Fig.11).
Ribs three through nine are referred to as ‘typical ribs’ due to their commonality in anatomic structure (see Fig. 12). Ribs one, two, ten, eleven and twelve are referred to as ‘atypical ribs’ due to their unique anatomic structure (see Fig. 13).

Fig. 12. The above picture is that of a ‘typical rib.’ A typical rib has the following characteristics: head that articulates with the bodies of the vertebrae; neck; tubercle that articulates with transverse processes of the vertebrae; angle, which is a point just lateral to the tubercle where the shaft bends forward; and the costal groove in which the intercostal arteries, veins, lymphatics and nerves travel. (Wiki)
The anatomic structure of a typical rib (ribs 3 to 9) consist of a shaft with an anterior and posterior end (see Fig. 12). The shaft has a convex curvature externally and is grooved internally next to its inferior border, which is sharp, whereas the upper border is rounded. Posteriorly, these ribs articulate with the vertebral column through two facets. The inferior facet articulates with the body of the corresponding thoracic vertebra, i.e. the third rib articulates with the third thoracic vertebra, whereas the superior facet articulates with the above vertebral body. These articulations with the thoracic vertebrae are called costovertebral joints, i.e. they connect the head of the ribs to the thoracic vertebrae. Directly lateral to the head of the rib is the neck, which is a flattened portion of the rib (see Fig. 12). The neck extends lateralward from the head for about 2.5 cm. Following the neck is the shaft of the rib. At the point of juncture of the neck and shaft is a posteriorly directed eminence called the tubercle, which has two parts. The superior lateral part serves for the attachment of the lateral costotransverse ligament. The inferiormedial part articulates with the transverse process of the vertebra of the same number as the rib, i.e. the tubercle of the third rib articulates with the transverse process of the third thoracic vertebra. Immediately lateral to the tubercle is the angle of the rib, which marks an abrupt change in the curvature of the shaft. The angle is the site of greatest twist in the rib. The weakest part of the rib is immediately anterior to its angle. The anterior end of the shaft is cupped for the reception of the costal cartilage.

The anatomic structure of the atypical ribs are as follows:

1. The first rib is the broadest, shortest, and most sharply curved (see Fig. 13). It has no twist, and its superior and inferior borders are directed inward and outward rather than up and down. Its head has a single facet to articulate with the first thoracic vertebra (T1). The superior surface of this rib has two shallow grooves, which are separated by a small pointed projection, the scalene
tubercle. The groove anterior to the scalene tubercle is for the subclavian vein. The subclavian artery and typically the lower trunk of the brachial plexus travel in the groove behind the tubercle. The under surface has no costal groove and the sternal end is thick. As discussed previously, the first rib forms the superior thoracic aperture together with the manubrium and T1 (see Fig. 7).

2. The second rib is twice the length of the first rib, less curved, thinner and minimally twisted (see Figs. 14 & 15).

Fig. 14. This is an illustration of the second rib. The non-articular portion of the tubercle is only occasionally feebly marked. The angle is slight, and situated close to the tubercle. The body is not twisted, so that both ends touch any plane surface upon which it may be laid; but there is a bend, with its convexity upward, similar to, though smaller than the found in the first rib. The body is not flattened horizontally like that of the first rib. Its external surface is convex, and looks upward and a little outward; near the middle of it is a rough eminence for the origin of the lower part of the first and the whole of the second digitation of the serratus anterior; behind and above this is attached the scalenus posterior. The internal surface, smooth, and concave, is directed downward and a little inward; on its posterior part there is a short costal groove. (Wiki)
Fig. 15. This is an illustration comparing the first (on the right) and second ribs. (Wiki)

The second rib has two facets to articulate with T2 and T1. Its outer surface faces outward and upward. The special feature of this rib is a marked tuberosity approximately at its middle of the outer surface for the origin of a part of the first and the whole of the second digitation of the serratus anterior muscle. Behind the tuberosity is a roughened line of attachment for the posterior scalene and other muscles.

3. The tenth rib has a single articular facet on its head that may articulate with the intervertebral disc above, besides to the upper border of the tenth thoracic vertebra (see Fig. 16). The ninth and tenth ribs are typically united anteriorly by a fibrous joint. However, as has been pointed out previously, the tenth rib may be free, with its anterior end being pointed like the eleventh and twelfth ribs. Otherwise, this rib conforms to the ‘typical’ ribs.

4. The eleventh rib has a single facet on its head, but no neck. It also may have no tubercle or a very small one, but with no facet. It has an ill-defined slight angle and
a shallow costal groove. The anterior end is pointed and tipped with cartilage (see Fig. 17).

Fig. 16. The above is an illustration of the tenth rib. Note its single articular facet on its head.

Fig. 17. The above is an illustration of the eleventh rib. It has a single large articular facet. It has no necks or tubercles, and is pointed at its anterior ends. The eleventh has a slight angle and a shallow costal groove. (Wiki)

5. The twelfth rib is small and slender and may be even shorter than the first. Its tubercle, angle, and costal groove are absent or if present are ill-defined. The shaft tapers off at its sternal end and carries only a small costal cartilage. Both the eleventh and twelfth ribs terminate in the abdominal wall (see Fig. 18).
Fig. 18. The above is an illustration of the twelfth rib. It has a single large articular facet on the head. It has no necks or tubercles, and is pointed at its anterior ends. The twelfth has no angle or costal groove. It is much shorter than the eleventh rib, and its head is inclined slightly downward. Sometimes the twelfth rib is even shorter than the first rib.

C. Costal Cartilages: The costal cartilages are bars of hyaline cartilage that extend from the ribs anteriorly and contribute substantially to the elasticity of the walls of the chest, hence enhancing the mobility of the chest. The upper seven cartilages join the sternum; the next three, eight through ten, articulate with the cartilages just above them; the last two are mere tips which end in the wall of the abdomen. The cartilages increase in length through the first seven and then gradually become shorter. The cartilage of the first rib joins the manubrium without an intervening joint. Costal cartilages two to seven join the sternum by synovial joints (see Fig. 19). In the older age group the costal cartilages tend to ossify superficially, lose their pliability, become brittle and thus susceptible to fracture.
D. Sternum: The sternum is located in the anterior midline of the chest sloping downward and forward. It is slightly convex on its anterior aspect and a little concaved posteriorly. It is somewhat dagger in shape, being broader above and narrow below, with an average length of 17 cm (6.69 inches) in the adult. The sternum consists of three parts: manubrium, body, and xiphoid process (see Figs. 19 & 20).

1. Manubrium: The name manubrium refers to a handle-shaped structure. The human manubrium is quadrilateral in configuration. Its anterior surface is convex from side to side, but concaved from above downward and provides partial origin for the pectoralis major and sternocleidomastoid muscles. Its thick superior border forms the shallow concave floor of the jugular notch (suprasternal notch) (see Fig. 20). To each side of the jugular notch is the clavicular notch for the articulation with the sternal ends of the clavicle. The lateral borders have a depression superiorly for articulation with the first costal cartilages (see Fig. 20). Inferiorly, the lateral borders have a notch for articulation with part of the costal cartilage of the
second rib (see Fig. 20). The inferior border, oval and rough, has a thin layer of cartilage for articulation with the superior border of the body. This cartilage does not ossify until old age. An important point to remember anatomically is the articulation between the manubrium and sternum is not in the flat plane, but instead projects forward forming the sternal angle (angle of Louis) (see Fig. 10). This sternal angle, as previously discussed, denotes the location of the sternal end of the second rib and is the most reliable surface marker of the chest.

2. **Body of the Sternum:** The body of the sternum is long narrow and thinner than the manubrium. The body extends from the fifth to the ninth vertebra. The anterior surface is virtually flat. Three transverse ridges extend across the anterior surface,
which represent the lines of fusion of the embryologic segments of the sternum that formed the body. A sternal foramen, may be found between the third and fourth sternebrae. These three ridges also occur opposite the articular depressions for the third, fourth, and fifth costal cartilages (see Fig. 21). The manubriosternal joint is marked by a posterior transverse groove and is palpable anteriorly as a ridge. On each side of the anterior surface of the body, the pectoralis major muscle takes origin; from the posterior surface arises the transversus thoracis muscle.

Fig. 21. Note the transverse ridges. (Wiki)
3. **Xiphoid Process:** This is the lower portion of the sternum; sword-shaped cartilaginous process, which is cartilaginous in youth; its upper part, especially, becomes ossified in the adult. The xiphoid process is in the epigastrium. It is considered to be at the level of the ninth thoracic vertebra. The upper border of the xiphoid process articulates with the body of the sternum. The xiphosternal junction marks the lower limit of the thoracic cavity. It is an anatomical landmark, in the median line (see Fig. 8), for the upper surface of the liver, the diaphragm, and the lower border of the heart. Anteriorly, it serves for the attachment of the rectus abdominis muscles; laterally, to the aponeurosis of the flat abdominal muscles; and posteriorly, to the transversus thoracis and the sternal slips of the diaphragm. The xiphoid shows considerable variation in form; frequently it is bifid and occasionally perforated (see Fig. 22).
Fig. 22. The above illustration depicts some of the variations in the sternum, such as the apertures (perforations) in the lower body of the sternum and xiphoid process and the bifid xiphoid process. (Wiki)

E. Forensic Anatomy: Although technically the liver, kidneys, stomach and spleen are within the abdominal cavity, anatomically, they are located beneath the costal margin and thus are not uncommonly involved in blunt force traumatic injuries to the chest, as well as gunshot and stab wounds of the chest. In gunshot and stab wounds of the chest it is not uncommon for there to be perforation of the diaphragm and entrance into the peritoneal cavity followed by penetration or perforation of the liver, kidneys, stomach or spleen. A gunshot or stab wound involving the heart, depending on the track, can perforate the diaphragm followed by penetration or perforation (penetration means to enter, whereas perforation means to pass through) of the liver with involvement of the inferior vena cava. This is due to the anatomic position of the heart, which results in the lateral edge of the right ventricle lying on the diaphragm by way of the pericardial sac.

III. Mechanisms of Chest Injury
There are two types of force, which the trunk experiences when blunt force is applied, as in a motor vehicular accident, transient body forces (force/surface/body volume) and transient surface reactions (force/surface area).

Transient body forces lead to distortion of the trunk, which result in some of the viscera and/or vessels within the chest and or abdomen being physically distorted, which may cause lacerations and hemorrhage. The underlying mechanisms for these injuries are due to rotary inertia, sheer distortion, flexion or extension and hydraulic loading.

Transient surface reactions are believed to be responsible for deformation of the chest which causes contusions and lacerations both of the skin and underlying soft tissues and the lungs and or heart, most especially in the area of contact. Experimental recording of intrathoracic pressure and movement of the chest wall shows that the greatest values for transient body forces and surface tractions occur with low velocity impacts rather than high velocity. This is due to the fact in high velocity impacts, peak overpressure occurs before peak deflection. Using the lungs to demonstrate what occurs in a low velocity impact, since the lungs are less dense than
the tissues of the chest wall, there will be a separation of these structures at the impact site. The lungs being less dense will move ahead of the chest wall, which in turn produces a negative pressure. This results in accentuating the difference of tissue density in the components of the lungs, which if the stresses and strains are severe enough, there is disruption of lung tissue.

Blunt force injury to the chest can cause disruption of the myocardium in the form of lacerations, which may be superficial involving only the epicardium or extend deeply into the myocardium and on occasion communicating with a ventricular cavity. Typically, the underlying causation is apparent at the time of autopsy either as a fractured rib and/or sternum or foreign objects penetrating the chest on impact. What is less clear as to causation is when there is evidence of cardiac rupture but no apparent bony fracture or penetration by a foreign object. It is true that violent compression of the heart between the anterior chest wall and the vertebral column can cause myocardial rupture. However, the concept of violent compression of the abdomen and or lower extremity causing myocardial rupture is not so clear. The most common explanation for this causation is such violent compression produces a sudden increase in venous return, which results in sudden distention of the right atrium and ventricle, which because of the thinness of their walls, rupture. What is of interest is that in vitro experiments have shown that right atrial pressure is indeed increased in this way, however, ruptures have yet to be produced.

When compressive force is applied to the anterior chest there will be a transient decrease in intrapleural space, the degree of which being determined by the intensity of the force and the resistance to compression. Rib fractures occur when their elastic limits have been exceeded, the broken ends of which may lacerate the lungs and/or the heart. However, both contusions and rupture of the lungs or the heart can occur without evidence of skeletal fracture. Also, injuries to the thoracic viscera can occur following abdominal compression. Blunt abdominal impact can cause the liver and diaphragm to be pushed upward resulting in displacement of the lungs and/or heart, thus leading to blunt force traumatic injuries. It has also been shown that the thoracic viscera move caudally (toward the feet) under forceful acceleration. What must be kept in mind is compression injuries are not simply a matter of squeezing tissues between two firm
surfaces. A pure linear force is seldom applied to the body in a motor vehicular accident. Often there are rotational forces which substantially contribute to the resulting injuries. Compression of the chest and/or shear wave displacement of the heart and aorta result in distortion and twisting of the two structures relative to one another with potentially serious consequences for the integrity of the aorta. For example, in anterior-posterior compression of the chest there is a relative backward motion of the heart. The stresses and strains of this displacement will be concentrated at anatomic points where the aorta’s position is relatively fixed, such as at the level of the ligamentum arteriosum, which is attached to the underside of the arch opposite the origin of the left subclavian artery (see Fig. 23).
Fig. 23. The above represents the anterior view of the heart. 1 represents the right border of the heart composed by the right atrium (RA). 2 represents the inferior border of the heart made up by the RA, right ventricle (RV), and the left ventricle (LV). 3 represents the left border of the heart made up by the LV. AA (ascending aorta), AAR (aortic arch), PT (pulmonary trunk), LA (left atrium), LPV (left pulmonary vein), LPA (left pulmonary artery), Lig A (ligamentum arteriosum), which is the embryonic connection between the pulmonary trunk and the aortic arch. At one stage in development, the lungs were not functional so the blood was shunted into the arterial system at this site. Oxygen exchange in the embryo occurred at the placenta and not the lungs.), LS (left subclavian artery), LCC (left common carotid artery), BC (brachiocephalic artery), SVC (superior vena cava), IVC (inferior vena cava), RPA (right pulmonary artery), and RPV (right pulmonary vein).

The ligamentum arteriosum is a band of connective tissue that is approximately 1.5 cm long and 3 to 5 mm in diameter; it represents the remains of the fetal ductus arteriosus and the dorsal part of the left sixth aortic arch. There is a narrowing of the lumen of the aorta at the level of the origin of the left subclavian artery and the ligamentum arteriosum called the aortic isthmus. Another point of relative fixation for the aorta is its root. The aortic root is in essence the outflow tract of the left ventricle forming a bridge between the left ventricle and the ascending aorta. It also serves as the supporting structure for the leaflets of the aortic valve (see Figs. 24, 25, 26 & 27).
Fig. 24. The above depicts a section through the heart replicating the parasternal long axis, showing how the aortic root is the centerpiece of the heart. The root extends from the basal attachment of the valvular leaflets within the ventricle (yellow arrows) to the sinutubular junction (red dotted line). The compass shows the orientation relative to the remaining thoracic organs. (The surgical anatomy of the aortic root, Anderson RH, 2007;(219):2527, Multimedia Manual of Cardiothoracic Surgery).

Fig. 25. This is a closeup of Fig. 24, which shows the extent of the aortic root, and reveals the semilunar attachments of the valvular leaflets supported by the right coronary and non-coronary aortic valvular sinuses. The red dotted line again shows the sinutubular junction, which is the distal extent of the root, while the red arrow shows the basal attachment of the right coronary aortic valvular leaflet, marking the proximal extent of the root. As shown by the yellow arrow, the anatomic ventriculo-aortic junction is in the middle part of the root, and is crossed by the hing-lines of the valvular leaflets. (The surgical anatomy of the aortic root, Anderson RH, 2007;(219):2527, Multimedia Manual of Cardiothoracic Surgery).
Fig. 26. The aortic root has been opened from behind and spread apart, so that the full width of the cylinder can be seen. The aortic valvular leaflets have then been removed, revealing the semilunar nature of their attachments. The purple dotted line shows the anatomic ventriculo-aortic junction, which is the union between the ventricular musculature and the aortic wall at the bases of the left and right coronary aortic valvular sinuses (1 & 2), but between the aortic wall and fibrous continuity with the mitral valve at the base of the non-coronary sinus (3). Note how the semilunar attachments incorporate muscle at the base of the coronary aortic sinuses, but fibrous tissue within the ventricle as the hinge lines extend distally to reach the sinutubular junction (red dashed triangle). (The surgical anatomy of the aortic root, Anderson RH, 2007;(219):2527, Multimedial Manual of Cardiothoracic Surgery).

The aortic root extends from the basal attachments of the aortic leaflets to the sinutubular junction (see Fig. 25). The root is a cylinder with its walls being made of the aortic valvular sinuses as well as the interdigitating intersinusal fibrous triangle and two small crescents of ventricular muscle incorporated at its proximal end (see Fig. 26). It is the semilunar attachments of the leaflets within the valvular sinuses that form the hemodynamic junction between the left ventricle and the aorta (see Fig. 27).
Fig. 27. The above is an illustration which shows the bisected aortic root, and how the semilunar attachment of the valvular leaflets incorporates aortic wall in the intersinusal triangles, and ventricular tissues at the base of each of the coronary aortic sinuses. (The surgical anatomy of the aortic root, Anderson RH, 2007;(219):2527, Multimedral Manual of Cardiothoracic Surgery).

There is another point of relative fixation, which occurs in the region of the left hilum (root) and its component structures, the left mainstem bronchus, pulmonary artery, pulmonary veins, the bronchial arteries and veins, the pulmonary plexuses of nerves, and the lymphatic vessels and nodes. All of these structures are held together by mediastinal connective tissue surrounded by a reflection of the pleura. The root of the left lung is ventral to the thoracic aorta and inferior to its arch. The root of the left lung serves as a fulcrum, about which the heart moves.

It is thought that rupture of the heart is more likely to occur during systole rather than diastole. However, some believe the cardiac cycle the heart is in at the time of the compressive issue plays little role whether the heart will rupture.
The mechanisms of chest injury can be divided into five categories: blunt force (acceleration/deceleration), crushing injuries, penetrating/perforating injuries (stabbing or gunshot wounds), blast, inhalation burns and foreign body aspiration.

A. Blunt Force (acceleration/deceleration): Typically, these injuries are the result of a fall or a motor vehicular accident. A fall of 15 feet will have an impact velocity of approximately 22 mph, while a fall from 65 feet will produce an impact velocity of approximately 45 mph. What is often forgotten is on impact, the resulting sudden deceleration leads to the intrathoracic viscera being subjected to apparent marked increase in their organ weights. For example, a fall of approximately 30 feet onto a hard surface, will effectively increase the weight of a heart which weighs 350 grams to one that weighs 14,000 grams on impact. This sudden increase in apparent weight has the potential to cause disruption of not only the heart, but its attached vasculature, as well as other structures within the thoracic cavity. The G force generated by a fall of approximately 30 feet is about 40 G, which is analogous to a car traveling at 45 mph striking a concrete wall. To carry this example further, should the same car be traveling at 67 mph, it will generate 90 G on impact with the concrete wall.

Pragmatically, if a person falls a distance of approximately 15 feet, impacting a hard surface, you can anticipate substantive intrathoracic injuries. What is also important to remember is the chest wall has considerable recoil, especially in children and young adults, and has a good “memory” for its normal shape, hence the potential extent of intrathoracic injury is best assessed through examination of the motor vehicle rather than the appearance of the person. For the same reason, in cases of falls it is important that the height of the fall be accurately determined as well as the nature of the impact surface to correctly determine the intrathoracic injuries.

Blunt force trauma to the thorax occurs primarily in one of four directions:

1. **Anterior-posterior:** Anterior-posterior directed force causes a relative backward movement of the heart, which can lead to disruption of the aorta especially at three relatively fixed points previously discussed. The most common point of aortic disruption is at the level of the ligamentum arteriosum, just below the origin of the left subclavian artery. Anterior-posterior directed force can also cause a laceration, the so-called ‘wishbone’ fracture, either of the left and/or right mainstem bronchus,
which is believed due to the backward and upward arc the heart travels in following such AP displacement during deceleration injuries. Injuries to the heart itself are not uncommon in such deceleration injuries, with or without fracture of the sternum. Cardiac injury occurs in approximately 50% of deceleration injuries. One of the intracardiac injuries that can occur with acceleration and or deceleration injuries is destruction of the valvular mechanism of the heart.

2. **Posterior-anterior:** Posterior-anterior directed force is not considered under blunt injuries to the thoracic because relatively few intrathoracic injuries result from impact to the back. It is on this fundamental point that many have considered reversing the direction of the seats within commercial aircraft.

3. **Lateral compression:** Such side to side compression of the chest in deceleration typically causes fractures of the lower ribs, not uncommonly associated with other injuries of the body. For example, with fractures of ribs seven through ten laterally, one should look for injuries of the spleen, liver and kidneys. ‘Flail chest’ can arise from fractures of the ribs laterally, however, when this occurs the portion of the chest involved is small with intrathoracic injury being confined to contusions and/or lacerations of the lung.

4. **Transdiaphragmatic:** Although seatbelt use has led to a significant decrease in injuries due to motor vehicular accidents, there is evidence that substantive deceleration can cause a sudden rise in intra-abdominal pressure, together with shearing and twisting of the upper trunk if only a seat belt is used. Such a sudden increase in intra-abdominal pressure can lead to rupture of the diaphragmatic leafs, especially on the left. Unfortunately, such injuries are not often recognized in the paramedics or ER physicians initial evaluation of such traumatically injured patients.

**B. Penetrating Injuries:** If the lungs are penetrated or perforated by a low velocity missile or a stabbing instrument the resulting parenchymal damage has more to do with the extent of the hemorrhage into and near the wound track. This is primarily due to the fact the lung has a low density, great elasticity and has good properties of healing. However, a high velocity missile, especially if it is designed to flatten or fragment on
impact, will cause extensive lung parenchymal damage on perforation; typically, such injuries are non-survivable.

The heart, as well as the major vessels, will sustain injury with low velocity missiles or a stabbing instrument, the exact nature of which will be determined by the location of the wound track and the anatomic structures involved. In those who have sustained an injury due to a high velocity missile, it is absolutely imperative they receive immediate medical attention and surgery for any hope of survival.

**C. Crush Injuries:** These are injuries of the chest in which its elastic limits and those of the intrathoracic viscera have been exceeded. The resulting severity of the injuries is often determined by the time the crushing forces have been applied. Typically, the person has sustained an anterior-posterior directed crushing force. These patients are often in shock, requiring rapid resuscitation and volume infusion. Most of these patients will present with flail chest due to multiple rib fractures, pneumothorax and/or hemothorax, pulmonary contusions and lacerations, cardiac and aortic injuries, and rupture of the diaphragmatic leafs. These injuries are often associated with rupture of the liver, spleen and kidneys. The mortality rate in such patients is high.

There is another category of crush injury patients which is referred to as ‘**traumatic asphyxia syndrome**’, also referred to as Ollivier syndrome or Perthes syndrome. In these patients the crushing forces are applied over a wide area for a period as short as 2 to 5 minutes. The classic syndrome includes cervicofacial cyanosis, subconjunctival hemorrhage, widespread capillary and venular dilatation and rupture causing petechiae and ecchymosis of the face, neck and upper torso, which is especially prominent over the eyelids, nose and lips (see Fig. 28 & 29). Hemorrhage into the retina is usually present (see Fig. 30). There is often associated bluish discoloration (cyanosis) of the upper part of the chest (see Fig. 28). Periorbital edema and ecchymosis are often present, as well as bilateral optic disc edema. Neurologically, the patients with this syndrome manifest agitation, restlessness, and disorientation. These clinical symptoms are often associated with hemorrhage into the leptomeninges, as well as the cerebral hemispheres (see Fig. 31).
Fig. 28. Patient with traumatic asphyxia syndrome. The head, neck, and upper chest are cyanotic and edematous, with multiple petechiae. He also had bilateral subconjunctival hemorrhages and bilateral hemothorax. (Wiki)
Fig. 29. Subconjunctival hemorrhage with traumatic asphyxia. (Wiki)

Fig. 30. Retinal hemorrhages that can be seen in traumatic asphyxia. (Wiki)
Fig. 31. This is a brain of a sheep showing congestion of the vessels in the leptomeninges and finite hemorrhages (petechial hemorrhages). There is also evidence of cerebral edema (swelling of the brain) manifested by flattening of the gyri and narrowing of the sulci. (Wiki)

Typically, the above injuries are associated with injuries of the brain, thorax, and abdomen. The injuries to the thorax can have significant pulmonary and cardiac complications in the form of severe contusions, and disruption of tissue, as well as a flail chest.

There are a number of hypothesis as to the pathophysiology of traumatic asphyxia syndrome. Tardieu in 1866 offered the first explanation, “the punctiform ecchymosis of the face, neck and chest are caused by the effort in which resistance to suffocation manifest itself.” Heutes in 1874 proposed two hypotheses: first, a mechanical reflux of blood out of the compressed chest into the valveless veins of the head and neck leading to venular and capillary atony and stasis cyanosis, and second, contusions of the abdominal sympathetic nerves causes vasodilation of the head and neck region followed by cyanosis and petechial hemorrhages of the head and neck. Bolt in 1908
postulated four factors: (1) deep inspiration, (2) closure of the glottis, (3) thoracoabdominal effort, e.g. splinting of thoracic and abdominal musculature, and (4) thoracic or abdominal compression that forces blood into the cervicofacial regions producing venular and capillary stasis. In 1951, Reichert and Martin conducting experiments using dogs showed that occlusion of the superior vena cava alone could cause considerable congestion of the upper extremities and head and neck regions, but that in of itself was not sufficient to cause the clinical features of traumatic asphyxia. In 1968, Williams and associates hypothesized that when the person who is experiencing severe chest compressions realizes the potential outcome they elicit a fear response. This fear reflex manifest by the patient taking a deep breath and holding it, bracing them self. This results in a closed epiglottis, which markedly enhances the increase in intrathoracic pressure. In 1989 Thompson and coworkers showed the elevated intra-abdominal pressure developed during the fear response would lead to collapse of the inferior vena cava, which protects the lower torso from venous injury seen in the head and neck and upper torso regions in traumatic asphyxia.

Survivability is determined by the morbidity of the associated injuries. In those with pure traumatic asphyxia syndrome who survive to the emergency room, a full recovery should be expected.

**D. Blast Injuries:** Of the various physical disturbances caused by detonation of a bomb or shell, there are two which are peculiar to explosions in air, which are especially likely to cause internal injuries. One is the instantaneous change from normal to greatly increased atmospheric pressure (the blast wave), and the other is impact against the body of the rapidly displaced air.

The leading edge of a blast wave is called the blast front. When the blast front impacts a person they are immediately immersed in a stratum of atmosphere, which is under greatly increased pressure. High-order explosives produce a supersonic blast front, whereas low order explosives deflagrate (subsonic combustion) and do not produce an overpressure wave.

Deflagration is a term describing subsonic combustion that usually propagates through thermal conductivity; hot burning material heats the next layer of cold material and ignites it. Most “fire” found in daily life, from flames to explosions, is deflagration.
Deflagration is different from detonation, which is supersonic and propagates through a shock wave. For example, C4 explosions can create initial pressures of over 4 million pounds per square inch (psi). An individual in the path of an explosion will be subjected not only to excess air pressure, but to pressure from the high-velocity wind traveling directly behind the shock front of the blast wave. A lethal blast-induced overpressure of 100 psi travels with a velocity of approximately 1500 mph. Typically, a blast wave generating an overpressure of 60 to 80 psi is considered potentially lethal.

Although the blast wave generated by an explosion starts with a single pulse of increased air pressure, lasting a few milliseconds, the wave itself is quite irregular, with many eddies within it. This accounts for why two soldiers standing near a bomb when it detonates, one will suffer serious injuries, if not be totally fragmented, whereas the adjacent soldier experiences only a slight compression wave and survives without serious harm. Furthermore, since blast waves are reflected from surfaces, there tend to be areas where the waves meet, and there may be points of reinforcement or of neutralization of the pressure, depending on whether the two waves meet in the same or opposite plane. Typically, explosions near or within hard solid surfaces become amplified two to nine times due to shock wave reflection. Consequently, individuals between the blast and a building generally suffer two to three times the degree of injury compared to open spaces.

You must also remember when a wave of high pressure passes from a medium of high density to a medium of lesser density a disrupting or lacerating effect occurs at the junction of the two media. As a pressure wave passes through a body the sites of maximum injury are produced where a relatively friable tissue is against a substance of low density, especially a gas. Thus, since air is easily compressible by pressure while water is not, the most significant injuries produced by blast occur in gas-containing organs, especially the lungs, walls of the stomach, intestines and the middle ear. Another important factor that plays a significant role in the degree of blast injury is the distance the person is from the detonation site. For example, the detonation of a 125 lb. bomb will generate approximately 200 psi 15 feet from the blast site. At 50 feet the 125 lb. bomb will produce approximately 10 psi.
Thus, the magnitude of damage due to the blast wave is dependent on: (1) the peak of the initial positive pressure; (2) the duration of the overexposure; (3) the medium in which it explodes; (4) the distance from the incident blast wave; and (5) the degree of focusing due to a confined area or walls.

Another important point to remember is that with the sudden displacement of atmosphere in all directions away from the detonation site, there is created a central zone of diminished pressure immediately behind the moving stratum of compressed air. As the expanding force is exhausted leaving a zone of diminished pressure, there is a backward rush of air to restore the normal atmospheric pressure. The resulting wind produced by the backward rushing air will also impact the body, typically on the surface opposite from the detonation center. Injuries related to a blast can be divided into four categories; primary, secondary, tertiary, and quaternary.

**Inhalation Injuries:** Burns of the laryngotracheobronchial tree can follow inhalation of flame, superheated air and hot smoke as the result of an explosion, all of which have grave prognosis. Should the lips, mouth and oral cavity show evidence of burns you can anticipate there will be inhalation injury throughout the respiratory tract. In such fire victims there will be evidence of progressive pulmonary insufficiency as well as cardiovascular dysfunction, the degree of which will have a major impact on morbidity and mortality. Respiratory burns are frequently missed clinically. They have a mortality of approximately 50%. The morbidity and mortality increases when respiratory burns are associated with smoke inhalation.

Part of the increase in morbidity and mortality with smoke inhalation is the many highly toxic elements within the smoke. Combustion of wood and man-made plastic polymers (e.g. polyvinyl chloride, polystyrene and polyurethane) can lead to highly toxic gases in the smoke; these include hydrogen cyanide and many other inorganic and organic hydrocarbons, which are rapidly lethal when inhaled. In addition, enormous amounts of oxygen are consumed, which can lead to death from anoxia or death as the result of lethal levels of carbon monoxide.

The pathophysiological aspects of acute lung injury induced by combined burn and smoke inhalation will be discussed under tissue injuries associated with explosions.
There will be an extensive discussion of both the pathology and pathophysiology of burns in the chapter on thermal injuries.

**F. Foreign Body Aspirations:** Blunt force trauma of the craniofacial region can lead to unexpected aspiration of teeth, fragments of dentures and foreign material. Such aspirated material will typically follow the straightest and most dependent course after they enter the trachea. The right mainstem bronchus follows a more direct route than the left (20° to 30° as compared to 40° to 60° for the left mainstem bronchus). The greater angle assumed by the left mainstem bronchus is to allow it to extend around the heart. Typically, larger more solid objects lodge in the right mainstem bronchus, while smaller solid objects come to rest in the right lower lobe bronchus. This is especially true in the aspiration of foreign objects by children, generally in the age groups of 1 to 3 years. In both children and adults, larger objects are sometimes stopped at the larynx and are expelled by vigorous coughing. Sharper objects may perforate a bronchus, which can lead to hemorrhage or even penetrate the pleural cavity causing a pneumothorax. Also, small foreign bodies may migrate within the bronchi causing wondering infiltrates.

**IV. Blunt Force Traumatic Injuries to the Thorax**

**A. Fractures of the ribs, manubrium, body of sternum and xiphoid process (Adults):**

1. **Fractures of the ribs:** A rib fracture is a break (fracture) of one or more of the bones making up the rib cage. The most commonly fractured ribs are the middle ribs, usually due to a direct blow or as the result of a crushing injury to the thorax. It is more common in adults, especially the elderly, due to decreased rib cage flexibility. The underlying reason for rib fractures being especially common in the elderly is because of osteoporosis. The presents of fractured ribs in children is evidence of a significant amount of force having been applied and not uncommonly is associated with severe thoracic injuries such as contusions and laceration of the lungs and/or the heart. The most commonly fractured ribs are the seventh and tenth. The fracture occurs just anterior to the angle of the rib (see Fig. 12). Displaced rib fractures may be associated with disruption of adjacent structures such as intercostal
vessels, nerves and lymphatics. The displacement of the fractured ends can also lead to lacerations of the lung, heart, and great vessels. Typically, in most cases the lungs and heart escape injury. However, if the thorax has been subjected to severe blunt impact, as in a pedestrian being struck by a motor vehicle, falling from a height or sustaining a crushing injury, the lungs, heart, great vessels, liver, kidneys, spleen, and stomach may be penetrated. Multiple rib fractures result in increased pain and the possibility of ventilation impairment, and increase the likelihood of coexisting serious injury. The most serious blunt chest wall injury is a “flail” (“stove-in”) chest, a clinical condition characterized by paradoxical chest movement with breathing. “Flail” chest results from isolation of a segment of the chest wall by multiple rib fractures typically involving the anterior ribs in at least two places, costochondral separation, sternal fractures, or a combination these fractures. In such instances inspiratory effort resulting from diaphragmatic descent is associated with inward motion of the injured thoracic wall with little or no ensuing ventilatory exchange. The increased work of breathing creates only increased negative intrapleural pressure changes, which, in turn, cause more paradoxical motion of the chest wall. “Flail” chest interferes not only with respiratory exchange, but also with blood return to the right atrium (see Fig. 51, p 68).

The major physiologic consequence in “flail” chest is due to contusion of the adjacent lung (see Fig. 50) and a decrease in vital capacity. It is also important to remember, the paradoxical movement of the “flail” segment may be masked for several hours by intercostal splinting thus, the onset of symptoms may be delayed. Posterior rib fractures typically do not lead to “flail” chest because of effective splinting by the thick musculature of the back.

“Flail” chest is present in 5% to 13% of chest injury patients and is associated with increased mortality.

Rib fractures can occur without direct trauma and have been reported after sustained coughing and in various non-contact sports such as rowing and golf, often in elite athletes. They can also occur because of diseases such as primary or metastatic cancer or infections. Fractures due to a disease process
are referred to as pathologic fractures. The first rib is rarely fractured due to its location behind the clavicle. However, if broken, the fractured ends can be driven into the brachial plexus and the subclavian vessels causing serious complications (e.g., nerve damage and hemorrhage) (see Fig. 13). Typically, fractures of the first and second ribs (see Fig. 14) are associated with head and facial (craniofacial) injuries. Fracture of either the first and/or second rib can lead to a compromise in respiration. Although the first rib has very little movement during respiration, it serves as a base attachment for the intercostal muscles and the ribs below. In other words, during respiration, the muscles in the first intercostal space contract, drawing up on the rib below it and so forth, until all ribs have moved through a small distance. The combined movements increase the transverse and anteroposterior diameters of the thoracic cavity. If the first rib is fractured, especially in combination with a fracture of the second rib, its ability to serve as an anchor for the ribs and their respective intercostal muscles may be compromised, thus leading to decrease in the transverse and anteroposterior diameters of the thoracic cage during inspiration. Fractured ribs can lead to a number of complications to the lungs. The more important complications include lacerations, pneumothorax, interstitial emphysema, intrapulmonic and intrapleural hemorrhage, pleurisy, pneumonia, pulmonary abscess, empyema, and air embolism. In addition, an intercostal or internal mammary vessel may be lacerated causing a hemothorax. Should the traumatized person have preexisting adhesions, they may be torn leading also to a hemothorax (see Fig. 49, p 66).

There are occasions in which the immediate effects of the blunt force thorax injury are without any significant symptoms, but several hours later the seriousness of the injury becomes apparent. It is important to remember, especially in children and young adults, that even though there has been great displacement of the fractured end of the rib at the moment of fracture, the bone usually returns to an approximately normal position. The point is, when there is no obvious displacement at the time the patient is initially evaluated does not preclude the possibility of displacement.
2. **Fractures of the Manubrium**: Fractures of the manubrium typically require enormous force and are quite rare, although there is at least one published report of a fracture occurring in a non-contact sport, which will be discussed. The incidence of fractures of the manubrium is approximately one in twenty fractures of the sternum with sternal fractures themselves occurring in 5% to 8% of people who experience significant blunt chest trauma. The manubrium is a very rigid and strong bone, firmly held to the spine due to its attachments to the clavicles and the first two ribs bilaterally (see Figs. 21 & 32).

![Diagram of the thoracic cavity and ribcage](image)

**Fig. 32.** The above illustration shows the anatomical connections of the manubrium. Remember, the manubrium and the cartilage of the first rib form the anterior boundary of the superior mediastinum. (Wiki)
The clavicle forms a very strong and stable joint at the top of the manubrium. The first rib is attached directly to the body of the manubrium, whereas the second rib has strong ligamentous connections to the manubriosternal joint, ensuring its strength and stability. These attachments cause the manubrium to move backward when the spine is flexed. Some authors believe that although these attachments are important, more important is the forward displacement of the sternum produced by the lower ribs. It is believed there are two factors involved in the production of fractures of the body of the manubrium or the sternum. As the manubrium is forced backwards a sudden increase in intrathoracic pressure occurs, which will thrust the lower sternum forwards and upwards. The site at which the sternal injury occurs will depend on two variables: the type of manubriosternal joint and the type of articulation with the second rib.

There are three types of sternomanubrial joints: synovial, synchondral (a union between two bones formed by either hyaline cartilage or fibrocartilage) or synostoeal (the osseous union of bones which are usually distinct). A mobile synovial joint will be the weakest point of the sternum and displacement will occur at this site. If however, the joint is synchondrial or synostoeal and the manubrium is firmly anchored by the second and first ribs then the upper pieces of the sternum will break. If the manubriosternal joint is firm and the second rib articulation is mobile, the stress will pass through the body of the manubrium, which will than fracture.

As pointed out earlier, the manubrium can be fractured without contact. For example, a 26 year-old male who was in poor physical condition, embarked on a body building program. He performed body building exercises every second day for six months. He had exercised the pectoralis muscles each time, using four different sessions inclusive of bench pressing. The underlying causation for the transverse fracture just below the attachment of the first ribs to the manubrium was due to the muscle attachments and the direction of their forces on the manubrium and sternum. The attachment of the manubrium to the spine through the first and second ribs and the attachment to the shoulders through the clavicles was also part of the mechanism of the transverse fracture. The sternal head of
the sternocleidomastoid and part of the pectoralis major are attached anteriorly to
the manubrium and sternum. The sternohyoid and sternothyroid muscles are
attached posteriorly to the manubrium, whereas the transverse thoracis is
attached to the posterior part of the sternum. The very strong rectus abdominis is
attached to the distal part of the sternum. During his sit up exercises, in which
he raised the upper part of the body from the lying position, is when the fracture
occurred. This was because during the sit up exercise, hyperflexion of the spine
presses the upper part of the manubrium anteriorly, and the contraction of the
abdominal and thoracic muscles at the same time added further to the fracture
angulation producing a transverse fracture just below the attachment of the first
ribs to the manubrium.

It is believed the repetitive strenuous training sessions in this formerly untrained
patient led to a stress fracture in the manubrium, because the strength of the
muscles build up faster than the strength of the bones. Fracture-dislocation of the
manubriosternal joint has been described as a complication of seizures, the mech-
anism of which is believed similar to this case.

3. Fractures of the body of the Sternum: Fractures of the body of the sternum are
produced by direct blows, indirect injury, falls, crushing injuries, muscular action
such as in grand mal seizures and patients with tetanus, or spontaneously due to
progressive kyphosis of the thoracic spine. Also, high thoracic spinal fractures
may cause the sternum to fracture, usually due to hyperflexion.

Blunt force trauma applied anterior-posterior to the midline of the chest typically
displaces the lower fragment of the sternum posteriorly. Injury from indirect
violence is usually due to flexion-compression or flexion-rotation of the upper
thorax or vice versa, resulting in combined injury to the vertebral column and
sternum.

Seventy percent of sternal fractures are located in the body and 17.6% at the
manubriosternal joint. Fractures of the sternum most often occur following direct
trauma to the chest, such as in steering wheel injuries or falls from heights, but
they are also seen following hyperflexion trauma of the torso, an example of which
was discussed above. Fractures of the sternum are often accompanied by rib or
spine fractures. It has been postulated by Fowler (1957) that the force is transmitted to the sternum mainly through the ribs and to a lesser extent through the clavicles and chin, causing a forward buckling which tends to displace the upper fragment behind the lower. This is opposite displacement from that usually seen in direct injury to the sternum. This is due in part to the rigid attachment of the upper portion of the sternum to the vertebral column.

Another example of indirect violence causing a fracture of the sternum is a football player who receives a blow to the top of his head while he is bent forward with his head down. The player’s position causes the blow to be absorbed by the chest, leading to a fracture of the sternum.

Most sternal fractures occur in the midbody, and are usually transverse. Stress fractures are occasionally seen in athletes, such as wrestlers and body builders, an example of which was given above. They can also occur in women with osteoporosis and kyphotic thoracic spines.

Most sternal fractures are caused by blunt force trauma to the anterior chest. In the United States, motor vehicular accidents account for 60 to 90% of all sternal fractures. Sternal fractures have a 18 to 62% risk of associated thoracic, mediastinal or cardiac injury. As indicated above, sternal fractures are also associated with compression fractures of the thoracic spine secondary to hyperflexion of the spine at the time of injury.

Sternal fractures are associated with a 25 to 45% mortality rate due to the frequent associated chest injuries, such as cardiac contusion, myocardial rupture, aortic rupture, pulmonary contusion, and thoracic spine compression fractures, especially if the fracture is above T6; such fractures can lead to the development of autonomic dysrhythmia. However, if the sternal fracture occurs in isolation, their outcome is good.

5. **Fractures of the Xiphoid Process:** Fractures of the xiphoid process (see Fig. 32) typically results from blunt force trauma applied to the lower midline of the chest, such as can occur in a motor vehicular accident, contact sports, blows by a fist or other hard object or improperly performed chest compressions during cardiopulmonary resuscitation. Such anterior-posterior direct trauma can
fracture the xiphoid process propelling it into the liver causing a lethal hemor-
rhage. Most fractures of the xiphoid process are not lethal. For example, the
fractured xiphoid process may penetrate the diaphragm causing clonic diaphrag-
matic spasm. There is also a rare condition in which the fractured xiphoid process
can be pulled down inferiorly by the rectus abdominis muscles. This can lead to
elongation of the xiphoid process with reattachment to the sternum by a distraction
tissue neogenesis. Distraction tissue neogenesis manifest by an abnormal sensa-
tion, tenderness, and pain in the middle of the chest. These symptoms are
elevated by surgical removal of the xiphoid process.

B. Fractures of the ribs, costochondral, sternum and clavicles (Infants and
Children)

1. Rib fractures: Although rib fractures are not uncommon in adults they are a rarity
in healthy infants; the presence of rib fractures without adequate history are
important diagnostic signs of abuse. Rib fractures are seen in 5 to 27% of abused
infants and children. The greatest incidence of rib fractures in the pediatric age
group is in the younger-than-1 year-old age group. Overall, close to 90% of
abusive rib fractures are seen in infants younger than 2 years of age. What must
be understood is due to the flexibility of the thorax in infants and young children,
substantive force is required to fracture ribs. For example, in one study child
abuse was the underling causation for rib fractures in 32 of 39 infants (82%).
When an infant is squeezed side-to-side, such as when the hands are clamped in
each axilla or on the lower lateral chest wall, the resulting pressure causes flexion
of the ribs, causing a fracture in their posterior segments, typically near their necks
(see Fig. 12). The underlying mechanism is believed to be due to the levering of
the neck region of the ribs against the transverse process of the vertebrae, which
serves as a fulcrum causing excessive anterior flexion (see Fig. 33). What must
be remembered is in order for this to occur the ribs must be in the horizontal plane
relative to the transverse process, i.e., infant is held upright with the hands of the
abuser clamping the lateral wall of its chest followed by substantive compression
or shaking. Some believe if the infant is lying supine on a flat surface, as in
Fig. 33. The above illustration shows the costovertbral articulation of a typical rib, left posterolateral view. (Wiki)

cardiopulmonary resuscitation or being stepped on, it does not experience posterior rib fractures. However, Kleinmen believes if there is substantive anterior-posterior compression, the stress over the ventral cortex of the posterior rib, where the tuberosity articulates with the transverse process (see Fig. 33), can cause a fracture. The important point here is the anterior-posterior force must be substantive. Such anterior-posterior compression also stresses the lateral aspects of the ribs and can thus cause lateral rib fractures. More than 80% of fractures induced by child abuse occur in the posterior position. In the majority of cases of posterior rib fractures, there are not overlying bruises (contusions). Some believe the presents of posterior rib fractures, most especially in an infant under one year-of-age is virtually pathognomonic of child abuse.

Typically, rib fractures are often clinically occult, most especially posterior rib fractures and often do not show external evidence of trauma, rarely are associated with intrathoracic visceral injury, are usually bilateral, often involving multiple ribs.
Posterior rib fractures are especially difficult to see in the acute phase, i.e., before callus formation for several reasons: (1) the transverse process is frequently superimposed over the fracture site; (2) the fracture line often crosses obliquely to the radiographic beam; and (3) there is no displacement of rib fragments due to the preservation of the posterior periosteum.

Lateral and anterior rib fractures may result from direct blows or squeezing, the latter causing out-bending and tension. They are usually caused by substantive compression of the rib cage. It should be understood that substantive compression of the chest does not include CPR (cardiopulmonary resuscitation). Feldman studied the incidence of rib fractures in children emphasizing children receiving CPR and comparing the result with rib fractures associated with child abuse. Despite varying degrees of skill in administrating CPR, no rib fractures were identified in the non abused child. The flexibility of the ribs and sternum are so great that rib fractures seem to occur in extremely high-velocity injuries, such as motor vehicular accidents or through sustained compressive deforming forces. In lateral and anterior rib fractures the inner surface of the rib buckles and the outer surface may break in a manner similar to the greenstick fracture of long bones. Such acute lateral or anterior rib fractures can be difficult to see radiographically unless there is displacement of the fragments. Thus, lateral and anterior rib fractures in an infant or child may go undetected on plain radiography unless callus formation has occurred, which is typically between 7 and 10 days.

2. **Costochondral fractures:** These fractures are rare. In one study they constituted 4% of rib fractures due to abuse and all were associated with severe intra-abdominal injuries (see Fig. 34). These fractures are also very difficult to visualize by plane x-ray, usually being seen after callus formation.
Fig. 34. The blue portion of the above shown rib cages represent the costal cartilages. The point of juncture with the ribs, which are osseous, is the costochondral junction. (Wiki).

3. **Clavicular fractures:** Although from an anatomical perspective the clavicle is considered the strut of the upper limb and not part of the skeletal make-up of the chest, we will briefly review the significance of clavicular fractures in an infant and childhood (see Fig. 35).

In childhood the most frequent site of fracture in the clavicle is the midshaft. Fractures of the clavicle are seen in 2 to 6% of abused children. Violent shaking of an infant or child can cause both medial and lateral fractures; fractures of the medial clavicle, unless having a reasonable explanation, should be regarded as suggestive of child abuse until proven otherwise. One of the reasonable explanations that must be considered in an infant a few weeks to 2 months of age is birth trauma. Birth trauma and its relationship to fractured ribs will be discussed shortly.
Fig. 35. The clavicle is a doubly curved short bone that connects the arm to the body (trunk), located directly above the first rib (see Figs. 19 & 32). It acts as a strut to keep the scapula in position so the arm can hang freely. Medially, it articulates with the manubrium of the sternum at the sternoclavicular joint. At its lateral end it articulates with the acromion of the scapula at the acromioclavicular joint. It has a rounded medial end and a flattened lateral end. (Wiki)

4. **Fractures of the sternum**: Fractures of the sternum are rare and when present suggestive of child abuse unless there is a reasonable plausible explanation. The underlying foundation for this position is that to fracture the sternum in a child requires an enormous amount of force.

**C. Dating of Fractures in Infants and Children**

Although the dating of fractures is not in the category of mathematical exactness, some general information can be acquired:

1. In the living infant or child dating of the fractures should be left to the pediatric radiologist and to the pediatric pathologist, usually the pediatric orthopedic pathologist and radiologist in the deceased.

2. Typically, if there is no callus, the fracture is less than seven to fourteen days. If
there is a callus, the fracture is at least seven days old.

3. In the deceased infant or child, dating of the histologic features at the fracture site is best left to an orthopedic pathologist, preferably a pediatric orthopedic pathologist. This is due to the fact that the age of the infant affects the healing process. Callus develops in neonates sooner than in the older infants. Dynamic changes occur in the skeleton itself as an infant matures, and compact bone increases. You must also consider the history of the injury for in child abuse accurate histories are not forth coming. Lastly, many fractures due to child abuse are subjected to repetitive trauma, either affecting the same site or same bone, or because medical attention was not sought there is prominent pathology at the fracture site.

4. Some of the histological features which can be observed are: (a) periosteal bone formation usually indicating the fracture is less than 7 to 10 days old, seldom greater than 20 days old; (b) a fracture with an exuberant periosteal reaction or callus formation is greater than 14 days old; (c) a fracture with slight periosteal formation may be as recent as 4 to 7 days old; and (d) loss of the fracture line definition takes longer than new bone formation, approximately 14 to 21 days.

Considerations to think about when assessing rib fractures in a child under 2 years-of-age are as follows:

1. Injuries that are acquired during birth rarely cause rib fractures, however, there are some reported cases. The reason the birthing process is rarely associated with rib fractures is due to the fact that typically, the largest part of the fetus’s body, the head, is delivered first. The next largest body part to be delivered is the shoulders, with the rest of the fetus being easily delivered. In those few cases which have been reported in the literature involving rib fractures from birth trauma most involved posterior rib fractures that occurred during difficult vaginal deliveries and often involved infants large for their gestational age. It is believed direct compression of the infant’s chest from the mother’s symphysis pubis during the delivery is the underlying cause. Other conditions said to be associated with rib fractures occurring during birth are traumatic deliveries involving shoulder dystocia and breech deliveries.
To further underscore the rarity of rib fractures occurring during delivery, Rubin did a study involving 15,485 consecutive deliveries. In this study birth injuries were found in 108 newborns. Injuries included fractured clavicles, facial nerve injuries, brachial plexus injuries, intracranial nerve injuries, and assorted other fractures and soft tissue injuries. No infants with rib fractures were identified.

What should be remembered is fractures identified after the second month of life are not due to birth injuries.

2. Accidental injuries typically do not cause posterior rib fractures unless the dynamic of the force being applied causes the rib cage to be rotated beyond the horizontal plane of the transverse process of the thoracic vertebrae. Also, substantive compressive anterior-posterior application of force, such as occurs in motor vehicular accidents, heavy objects falling on the chest or prolonged pressing on the chest can cause rib fractures.

3. Infants with osteogenesis imperfecta (OI) or rickets can have rib fractures due to increase in bone fragility. However, OI is usually diagnosed by family history, an distinct clinical, radiological and laboratory features. OI is the most common genetic cause of bone fragility. It is a heterogeneous disorder caused by a molecular defect in collagen. Clinical features of OI include blue sclera, recurrent fractures, dentinogenesis imperfecta, short stature and bowing of the extremities. Radiographs typically show excessive wormian bones of the skull, long bone deformities, and osteopenia (see Figs. 35, 36, 37, 38 & 39).

Clinically, rickets manifest itself by growth retardation, metaphyseal faying, prominence of the costochondral junctions (rachitic rosary), and frontal bossing. Rickets is due to dietary deficiency of vitamin D. Laboratory analysis typically shows a low to low-normal serum calcium, low serum phosphorous, and elevated alkaline phosphatase activity (see Figs. 40, 41, 42, 43, 44, 45 & 46).

4. There is an entity, “temporary brittle bone disease,” which was originally described by Peterson. This entity has no scientific basis and is regarded as “court room science.”
Fig. 35. The above pictures show the classic blue sclera of osteogenesis imperfecta (Lobstein syndrome). People with OI are born with defective connective tissue, or without the ability to make it, usually because of a deficiency of type-I collagen. This deficiency arises from an amino acid substitution of glycine to bulkier amino acids in the collagen triple helix structure. The larger amino acid side-chains create steric hindrance that creates a bulge in the collagen complex, which in turn influences both the molecular nanomechanics as well as the interaction between molecules, which are both compromised. As a result, the body may respond by hydrolyzing the improper collagen structure. If the body does not destroy the improper collagen, the relationship between the collagen fibrils and hydroxyapatite crystals to form bone is altered, causing brittleness. Another suggested disease mechanism is that the stress state within collagen fibrils is altered at the locations of mutations, where locally larger shear forces lead to rapid failure of fibrils even at moderate loads as the homogeneous stress state found in healthy collagen fibrils is lost. These works suggest OI must be understood as
a multi-scale phenomenon, which involves mechanisms at the genetic, nano-, micro- and macro-level of tissues.
Historically, OI has been viewed as an autosomal dominant disorder of type I collagen. In the past several years, there has been the identification of autosomal recessive forms. There are 8 different types of OI, type 1 being the most common, though the symptoms vary from person to person. (Wiki)

Fig. 36. The above is an x-ray of the left forearm showing a fracture of the ulna and radius in abnormally shaped bones. They are bent and have thin shafts (gracile). These findings are consistent with a diagnosis of osteogenesis imperfecta. (Wiki)
Fig. 37. This is a photo of a child with dentinogenesis imperfecta, which is seen in OI. Dentinogenesis imperfecta is due to opalescent dentin resulting in discoloration of the teeth, ranging from dusky blue to brownish; poorly formed dentin with an abnormally low mineral content; obliterated pulp canal; normal enamel; the teeth usually wear down rapidly, leaving short brown stumps. (Wiki)
Fig. 38. The above is a picture of an infant with the most severe nonlethal form of OI, type III disease, which is characterized by severe bone fragility; multiple fractures; progressive marked deformity of the spine and long bones; and severe short stature, associated with vertebral compression, kyphosis, deformity of the limbs, and disrupted growth. (Wiki)
Fig. 39. The above is an example of a wormian bone, which is also known as an extra sutural bone. These are extra bone pieces that occur within a suture in the cranium. These are irregular isolated bones which appear besides the usual centers of ossification of the cranium and, although unusual, are not rare. They occur most frequently in the course of the lambdoid suture, as is shown above. They are also occasionally seen within the sagittal and coronal sutures. A large wormian bone at lambda, such as in the above photograph, is often referred to as an Inca bone (Os Incae), due to the relatively high frequency of occurrence in Peruvian mummies. Another specific wormian bone, the pterion ossicle, sometimes exists between the sphenoidal angle of the parietal bone and the great wing of the sphenoid bone. Wormian bones are a marker for various diseases, such as OI. Wormian bones may also be seen in: Pyknodysostosis, Rickets, “Kinky-hair” Menke’s syndrome, Cleidocranial dysositosisis, Hypoparathyroidism and Hypophosphatasia, otopalatodigital syndrome, primary acro-osteolysis and Down’s syndrome. (Wiki)
Figs. 40 (left) and 41 (right). These two figures depict growth retardation in a child with rickets. The picture on the left shows the child’s stature before treatment with calcium, whereas the one on the right shows the same child 2 years after treatment with calcium. (Wiki)
Fig. 42. X-ray of a child with rickets demonstrating classic metaphyseal fraying and physeal widening. (Wiki)
Fig. 43. A child with rickets showing rib beading (rachitic rosary). (Wiki)
Fig. 44. This is an illustration of the appearance of the inside of the thorax of a normal rat (left) and one suffering from rickets (right). Note the smooth and symmetrical form of the normal animal. On the right the rickety rat is seen to have prominent deformity of the thorax. The shoulders are rounded and the sternum is misshapen. There are large knobs on the ribs due to spontaneous fracture and attempts at healing. The costochondral junctions (junction of the osseous rib with its cartilage) are enlarged, and the ribs are bent inward at their ventral extremity. The beaded condition of these ribs is analogous to the “rachitic rosary” seen in children with rickets (see Fig. 43). (Wiki)
Fig. 45. Frontal bossing in a child with rickets. (Wiki)
D. Penetrating Injuries of the Thorax: Pulmonary injuries due to gunshot and knife wounds differ in several respects from those wounds caused by fractured ribs. Penetrating/perforating wounds of the chest wall caused by gunshot or knife wounds are more susceptible to infection. Another difference is those injuries associated with external wounds are typically more extensive than those produced by fractured ribs. Lastly, the pneumothorax produced by penetrating/perforating wounds is usually non-compressive, whereas the pneumothorax produced by fractured ribs is usually
compressive (see Figs. 47 & 48). This is due to the fact in the former the normal negative intrathorax pressure is replaced by atmospheric pressure. Loss of negative pressure causes the lung in the pleural cavity with the external wound to collapse. However, with expiration there is no increase in pressure within the pleural cavity, thus, the mediastinum does not shift to the opposite non-involved pleural space resulting in compression of that lung, as occurs in compressive pneumothorax. Such shifting of the mediastinum compromises blood return to the heart and thus cardiac function. This will be discussed further on pages 71 to 73.

Fig. 47. The above is an example of an open pneumothorax produced by an open/sucking wound which allows the free passage of air into and out of the pleural space. If the opening in the chest is two-thirds the diameter of the trachea, air passes through the chest wall defect with each respiratory effort, because air tends to follow the path of least resistance through the chest wall defect. This leads to impaired ventilation causing hypoxia and hypercarbia. (Wiki)
Fig. 48. Tension pneumothorax (compressive pneumothorax) develops when a “one-way valve” air leak occurs either from the lung or through the chest wall. Air is forced into the thoracic cavity without any means of escape. This causes a collapse of the affected lung, displacement of the mediastinum to the opposite side, decrease in venous return and thus, compromise of cardiac function, and compression of the opposite lung leading to further embracement of respiratory function. (Wiki)

Another complication of gunshot wounds is the missile producing the wound may carry pieces of clothing or fragments of bone into the pleural cavity and or lung enhancing the propensity for infection.

Although, the entrance wound produced by a missile may only measure 4 to 10 mm in its greatest dimension, the wound it produces in the lung may be considerably larger due either to the missile tumbling or its deformation (i.e., flattening-out or fragmentation)
coupled with the cone of released kinetic energy, which is primarily a function of velocity square of the missile.

The stab wound on the surface of the skin may be well-defined, however, due to the expansion and partial deflation of the lung during inspiration and expiration, it may show an extensive laceration rather than a well defined wound track.

Either a missile or knife wound may involve a pulmonary vessel causing a hemothorax, or pulmonary infarction distal to the wound track. (see Fig. 49).

A bronchus may be injured leading to interstitial emphysema.

Fig. 49. The above is an illustration of a hemothorax. (Wiki)
E. Non-penetrating Injuries of the Thorax: Although the lungs, because of its elastic structure, can undergo considerable gradual compression or absorb a strong impact to the overlying chest wall without evidence of injury to the underlying lung, sudden violent compression of the chest, such as in crushing or a forceful blow, as in a motor vehicular accident or assault can produce lung contusions and or lacerations. This is especially true if the glottis is closed at the moment of impact of violent blunt force trauma.

**Lung contusions** are the most common injury to the lungs caused by blunt force trauma. They may be single or multiple, finite or spread over a wide area, and they may be peripheral or deep within the parenchyma or both (see Fig. 50). They are also the most common lung injury, which can lead to death. Although contusions of the lung in adults are commonly associated with rib fractures, as in a “flail” chest, (see Fig. 51), they can be produced without them, especially in children and young adults. This is due to the flexibility of the chest wall and the elastic costal cartilages in this age group.

![Fig. 50. The above is a picture of a lung with multiple diffuse acute contusions. (Wiki)](image-url)
Fig. 51. This is an illustration of a “flail” chest. The major difficulty in a “flail” chest stems from the injury to the underlying lung in the form of a pulmonary contusion (see Fig. 50).
In a “flail” chest a segment of the chest wall does not have continuity with the rest of the thorax cage. Two or more ribs are fractured in two or more places. (Wiki)

These contusions are produced by rupture of the small peripheral blood vessels beneath the visceral pleura. This leads to bleeding into the subvisceral pleural alveoli (air sacs). How rapid these contusions form and how much area they occupy is determined by the number of vessels disrupted as well as the size of the vessels.
Typically, contusions are seen almost immediately. However, they may not be seen on x-ray for several hours. One of the points to remember for the forensic pathologist is that although contusions may be identified radiographically in the hospital record, they may not be seen at the time of autopsy. This is because contusions, especially smaller ones, may be cleared in 3 days.
Typically, pulmonary contusions serve as a red flag for the presence of extrathoracic injuries or some other intrathoracic injuries. In one study, those with pulmonary contusions showed extrathoracic injuries in 67% of the cases.
Pulmonary contusions are associated with a mortality of 16%, however, when they occur with “flail” chest (see Figs. 50 & 51), the mortality is 42%.
What must not be forgotten, the presence of blood in the alveoli seen with the microscope, in of itself does not automatically mean blunt force trauma. In deaths due
to anoxia, whatever the cause, alveolar capillary permeability is markedly increased
causing leakage of red blood cells into the alveoli, grossly seen as profuse hemorrhage.
Severe passive congestion can also cause intraalveolar hemorrhage due to leakage of
red blood cells and/or rupture of capillaries. Lastly, if the person sustained oral-facial
injuries, they may aspirate blood, which can end up in the alveoli.
Severe blunt force trauma to the chest can cause lacerations of the lung, which are
due to shearing forces or violent sudden compression (see Figs. 52 & 53).

Fig. 52. This is a photography of a laceration of a lung. (Wiki)
Fig. 53. This is a coronal CT showing lucencies (pale areas in radiography) in the lung caused by pulmonary lacerations on the left of the image (black arrows). (Wiki)

Typically, lacerations of the lung due to blunt force trauma occur as small, scattered lacerations, either on the surface of the lung or deep within the parenchyma. However, as shown in Fig. 52 they may occur as a single, large, non-communicating defect in the central portion of the lung. Lacerations lead to leakage of blood, as well as air, into the injured parenchyma, which in turn leads to the development of a hematoma that may also contain air. Hematomas of the lungs are more common in children and young adults due to the flexibility of the chest wall.

Hematomas of the lung undergo progressive shrinkage over time, which is usually evident by 6 weeks radiographically, however, they may still be recognized up to 4 months of the traumatic event. Especially large hematomas may persist up-to-a-year. Severe blunt force trauma of the lower chest of a child can result in torsion of the lung in a 180° arc such that the apex of the lung comes to rest next to the diaphragm. Torsion of a lung or lobe of a lung is extremely rare, but serious condition, that rarely is due to blunt force trauma to the chest, except in children. When it occurs in children it is typically associated with severe trauma, such as the child being run over by a car. In
adults, torsion of the lung is usually associated with thoracic surgery, spontaneous pneumothorax induced by a needle biopsy, pleural effusion or neoplasm.

V. Complications of Blunt Force Trauma to the Lungs

A. Pneumothorax: Although briefly mentioned above this subject will be discussed in greater detail. Pneumothorax is air gaining access to the pleural cavity. In order for this to occur there must be a communication between the alveolar air sacs within the lung and the pleural space through a defect in the visceral pleura or a communication between the air (environment) and the pleural space through a defect in the chest wall. Another mechanism which can cause the development of a pneumothorax is the leakage of air within mediastinal emphysema into the pleural space. Such leakage typically occurs into the left pleural space.

There are several types of pneumothoraces. A *spontaneous pneumothorax* is one that occurs without antecedent trauma to the thorax. A *primary spontaneous pneumothorax* occurs without underlying lung disease, while a *secondary pneumothorax* occurs in its presence. A *traumatic pneumothorax* results from penetrating or non-penetrating chest injuries. A *tension pneumothorax (compressive pneumothorax)* is a pneumothorax in which the pressure in the pleural space is positive throughout the respiratory cycle. *Primary spontaneous pneumothoraces* are usually due to rupture of apical pleural blebs, which are small cystic spaces that lie within or immediately under the visceral pleura. This type of pneumothorax typically occurs in smokers. Spontaneous pneumothorax can also occur as the result of an abscess cavity communicating with the pleural space, or over expansion of the alveolar air spaces, as can occur in asthma, which rupture.

Most *secondary pneumothoraces* are due to chronic obstructive lung disease with either blebs and or bullae due to emphysema which rupture. Other causes of secondary pneumothoraces are abscess cavities which communicate with the pleural space, or over expansion of the alveolar air spaces, as can occur in asthma, which rupture. A rare cause of secondary spontaneous pneumothoraces occurs in women with pleuropulmonary endometriosis. *Traumatic pneumothoraces* can result both from penetrating and non-penetrating trauma.
Tension (compressive) pneumothoraces are pneumothoraces in which positive pleural pressure is life-threatening both because ventilation is severely compromised and because the positive pressure is transmitted to the mediastinum, which causes mediastinal shifting, decrease in venous return to the heart with consequent reduced cardiac output and compression of the non-involved lung further compromising respiration.

Defects within the visceral pleura or in mediastinal emphysema, as discussed above, leads to a compressive pneumothorax. This is because with a defect in the visceral pleura, air continues to accumulate with each inspiration, however, due to the valve-like action of the visceral pleural defect, air cannot egress the pleural space with expiration because the defect closes. Likewise, air entering the pleural space through leakage of air from mediastinal emphysema has no egress point. Also, narrow slit-like or circular defects, such as those produced by an ice pick in the chest wall, will allow air to gain access to the pleural space during inspiration, but close off during expiration allowing the air to accumulate creating a compressive (tension) pneumothorax (see Fig. 48). It is important to remember that compressive pneumothorax is not only produced by trauma, they can also be spontaneous or therapeutic in origin as discussed above.

The result of a pneumothorax causes progressive collapse of the lung on the affected side to the point it ceases to expand at all, eventually becoming atelectatic (see Fig. 54). As the pneumothorax continues to expand it shifts the affected lung to the opposite side as well as the mediastinum and the trachea. With the shifting of the mediastinum, the contained heart is also shifted, which compromises the return of blood flow to the heart. This results in the distention of the neck veins and a decrease in cardiac output. The shifting of the affected lung and mediastinum into the uninvolved pleural space compromises the function of that lung. Such shifting ultimately leads to hypoxia and shock manifested by a decrease in blood pressure and tachycardia. At autopsy not uncommonly you will find subcutaneous emphysema due to air leaving the chest cavity and accumulating in the subcutaneous tissue.

A large defect in the chest wall will typically allow for communication between the air on the outside and the pleural space. This communication results in a non-compressive
type of pneumothorax. It is non-compressive because the defect allows for the free flow of air on inspiration and expiration. Ultimately this can result in paradoxical respiration as described under “flail” chest. The end result may be partial or complete non-aeration of the affected lung (see Figs. 47 & 48, p 63-64).

If there is evidence to suggest the deceased may have a pneumothorax, the chest should be opened under water. This is done by reflecting the skin and subcutaneous tissue of the lateral chest wall to the posterior axillary line. At this point fill the space between the reflected skin and subcutaneous tissue and the exposed rib cage with water. Then incise an intercostal space to expose the pleural space below the water level. If a pneumothorax is present, the air within the pleural space will escape through the incised defect as air bubbles in the water.

**B. Interstitial Emphysema:** The entrance of air into the connective tissue is called interstitial emphysema (see Fig.55). The most common cause is rupture of dilated alveolar air sacs in an emphysematous lung. Other causes are penetrating/perforating
wounds of the chest produced by a missile or a stab wound. Another cause is penetration of the lung by a fractured end of a rib. Rupture of the alveoli can also occur with the combination of vigorous coughing and bronchiolar obstruction, which causes a sharp rise in the pressure within the alveolar air sacs. In that vein, children with whooping cough and bronchitis are susceptible to interstitial emphysema. Ulcers of the stomach or duodenum may perforate leading to mesenteric or retroperitoneal interstitial emphysema, which in the case of the latter may spread to the mediastinum. A fracture of the larynx, missile or knife injuries to the larynx, trachea, or bronchi may cause interstitial emphysema. Obstruction to the airways by blood clots, tissue or foreign bodies, tracheotomy, jugular puncture, operating on the neck, chest or abdomen or unusual intense physical exercise, exertion such as lifting weights, in which before the lift, the person takes a deep inspiration and than closes his glottis all can cause interstitial emphysema.

Fig. 55. This is an example of interstitial emphysema involving the lung. Note the presence of air bubbles rupturing the interlobular space. (Faculdade de Medicina Veterinára. Universidade Técnica de Libboa) (Wiki)
The mechanism appears to be rupture of the marginal alveoli, which are those in which their base is against the bronchi. The air then enters the interstitium and spreads toward the hilus of the lung. From the hilus it then extends into the mediastinum. The interstitial emphysema can continue its spread by air dissecting along fascial planes and vascular spaces into the neck or downward into the retroperitoneal space and into the pelvis and lower extremities.

There are several potential lethal complications of interstitial emphysema. One is the interstitial emphysema may spread through the stroma of the lung and on reaching the visceral pleura form blebs. These in turn may rupture causing a pneumothorax. A pneumothorax can also occur through rupture of the mediastinal pleura. When this occurs it is typically on the left side. In both cases the resulting compression pneumothorax can lead to death.

The second, potentially lethal consequence of mediastinal emphysema is interference with blood flow through the pulmonary veins or the superior and inferior vena cava, thus compromising the return of blood to the heart leading ultimately to cardiac failure. Mediastinal emphysema can also cause air tamponade and decreased coronary blood flow, leading to cardiac failure.

**C. Hemothorax:** This refers to blood in the pleural cavity. It is believed by a number of experts that hemothorax is quite common in blunt force trauma to the thorax, being frequently found with air and serous fluid. There is a point I need to make here, and it primarily applies to the pathologist doing the autopsy on a patient who’s death is directly related to the hemothorax. In such a patient, it is essential that you do a hematocrit on what you believe to be blood in the pleural cavity. For if the hematocrit is more than half of the peripheral blood, the patient is considered to have a hemothorax. This recommendation is based on 37 years of experience as a Forensic Pathologist. The determination of the hematocrit on pleural fluid will substantially aid you under cross examination in either a criminal or civil case (see Fig. 49).

The primary concern of blood within the pleural cavity has little to do with compromising the mechanics of respiration, but a great deal to do with the volume of blood lost.
Hemothorax leading to death need not be the result of substantive injury, such as a traumatically induced rupture of the thoracic aorta, or for that matter, a rupture of a thoracic aortic aneurysm. A small vascular defect can lead to a fatal hemothorax. This is because during inspiration the negative pressure within the intrapleural space is accentuated, which in turn increases the flow of blood from the defect. Also, there is a tendency for blood to remain fluid longer in the intrapleural space.

Blunt force trauma to the chest without penetration or rib fractures can cause a fatal hemothorax through tearing of pleural adhesions followed by hemorrhage. As has already been discussed, blunt force trauma to the chest may fracture ribs, which in turn can lacerate intercostal or internal mammary arteries. The laceration of the vessel need not be large, for a needle puncture of an intercostal artery can lead to a fatal hemothorax.

Typically, lacerations of the lung do not lead to a fatal hemothorax. This is primarily due to the low pressure of the pulmonary circulation, retraction of the very elastic lung tissue and the inherent coagulation properties of lung tissue.

If a review of the medical records shows evidence of a hemothorax, but the patient lived for several weeks before dying, you may not see evidence of a hemothorax at the time of the autopsy. This is due to the fact the blood may have been absorbed by the pleural lymphatics.

Lastly, the presents of a small to moderate amount of blood within the pleural space may have a cause other than trauma. Small to moderate amounts of blood may be seen in asbestos-induced pleural disease, tuberculosis, and neoplastic disease, such as mesothelioma and metastatic or primary lung cancers invading the pleura.

**D. Pleuritis:** Pleuritis is an inflammation of the pleura (see Figs. 56, 57 & 58). It invariably will follow a penetrating wound of the chest wall.
Fig. 56. Fibrinous pleuritis of an eight-week-old lamb with pasteurelosis. Photo: Donal Toolan. (Wiki)
Fig. 57. The above photo is an example of fibrinous pleuritis and marbling of lung tissue in a ten-week old calf with *Mannheimia haemolytica* associated pneumonia. Photo: Donal Toolan. (Wiki)

Fig. 58. The above is another example of a pleuritis involving the upper lobe with extension into the lower lobe. This serves as an example of a diffuse spreading pleuritis. (Wiki)

Typically, in penetrating wounds the inflammatory reaction is bacterial and if the patient survives but does not receive appropriate medical care, the bacterial inflammatory reaction will develop into an empyemia (purulent [pus] pleural exudate).
A crushing injury of the chest, even though the chest wall is not penetrated, can lead to a pleuritis if there is already an existing pneumonic process. Also, if the person already has an existing localized pleuritis, blunt force trauma to the chest may cause a diffuse spread of the localized pleuritis (see Fig. 58).

There are a number of fundamental points you need to be aware of when accessing the pleura and fluid within the pleural cavity. Normally, the pleural cavity contains approximately 15 cc of serous, relatively acellular, clear fluid, which lubricates the pleural surface. There are several types of pleuritis, which are divided into categories based on the appearance of the pleura and the resultant appearance of the pleural fluid. These categories are serous (see Fig. 59), serofibrinous (see Fig. 60), fibrinous (see Figs. 56 & 57), suppurative (empyemia) (see Figs. 61 & 62), and hemorrhagic pleuritis (see Fig. 63).

Fig. 59. This is an example of serous pleuritis. Often serous and serofibrinous pleuritis exist together as depicted in Fig. 60. (Wiki)
Fig. 60. This is an example of serous (pale pink areas) and serofibrinous pleuritis in a pig. The more red blotches are the serofibrinous pleuritis. (Aleksandar Masic, Western College of Veterinary Medicine). (Wiki)
Fig. 61. Visceral pleura of the right lung showing the adherent yellow-white pus of a pleural empyema. (Humpath.com-Human pathology) (Wiki)
Fig. 62. The thick yellow-white exudate adherent to the visceral pleural surface of the right lung is an example of empyema. (Humpath.com-Human pathology) (Wiki)
The amount of the fibrinous component depends largely on the stage and severity of the inflammation. Fibrinous exudations generally reflect a later and more severe exudative reaction, which in an earlier developmental phase might have presented a serous or serofibrinous exudate (see Figs. 59 & 60). A suppurative pleuritis (empyema) is a purulent (pus) pleural exudate, which implies bacterial or mycotic (fungal) seeding of the pleural space. A point you need to remember, if the pleurisy is limited to the diaphragmatic surface on the right side, it may be secondary to perihepatitis, or to an abscess or other inflammatory conditions in the liver. Such inflammatory conditions may extend by continuity through the diaphragm into the pleural spaces, most often the
right side. However, a perisplenitis or peritonitis in relation to a gastric ulcer may extend through the left leaf of the diaphragm to cause a localized pleuritis on the pleural surface of the left leaf of the diaphragm.

Hemorrhagic pleuritis is manifested by sanguineous (bloody tinged) inflammatory exudate (see Fig. 63). These are not common. When they occur they are frequently found in hemorrhagic diatheses, rickettsial diseases, acute influenza pneumonia, and metastatic involvement of the pleural cavity. The sanguineous exudate must be differentiated from hemothorax.

Lastly, the pleural ligament, which is a bilaminar reflection of the pleura, passes from the mediastinal surface of each lung, below the root, to the pericardium downward to the diaphragm (see Fig. 64). It is more prominent on the right. On occasion this can be

Fig. 64. Mediastinal surface of the right lung. (Pulmonary ligament labeled at lower right). (Wiki)
mistaken for an adhesion.

**E. Pneumonia:** This may develop from blunt force trauma to the chest or as a consequence of an injury elsewhere in the body. Both non-penetrating and penetrating chest injuries are frequently followed by the development of pneumonia (see Figs. 65, 66 & 67). However, it is often difficult to establish a cause and effect relationship unless there is objective evidence of blunt force trauma such as fractured ribs, laceration of the lung, or contusions of the skin and underlying subcutaneous tissue. Where causation becomes problematic is if the person has lived for a time following the injuries, allowing for the pathologic features of the injuries to be mitigated. The underlying foundation of this complication appears to be immobility due to confinement in bed and compromise of respiration manifested by diminished rate and amplitude of respiration and

![Fig. 65. This is a photomicrograph of a normal lung. (Wiki)](image)
unconsciousness. Compromise of respiration is often associated with blunt force trauma, which interferes with the movement of the thorax and abdomen. When a

Fig. 66. This is a photomicrograph of a lobar pneumonic process involving the lung. Lobar pneumonia is a disease characterized by inflammation and congestion of the lungs (see Fig. 68). Infection with any of a variety of microorganisms is the typical cause, but sometimes the condition is related to an allergic reaction or inhalation of fluid, food, or certain gases. When pneumonia is restricted to a single lobe of the lung or to a portion of a lobe, it is referred to as lobar pneumonia. Pneumonia that has a more patchy extensive spread through the lungs is known as bronchopneumonia (see Fig. 67). The gram-positive bacterium Streptococcus pneumoniae (also known a pneumococcus) is responsible for most cases of lobar pneumonia. (Wiki)

person is unconscious the coughing reflex is often inhibited, which allows mucus and fluid to accumulate in the tracheobronchial tree. Such material serves as an excellent
cultural medium for bacteria to proliferate, thus leading to the development of pneumo-
nia. Often because of blunt force trauma the victim may aspirate fragments of broken
teeth, dentures, food, blood, vomitus, etc, which further enhances the development of
aspiration pneumonia (see Fig. 69).

Fig. 67. This is a photomicrograph of bronchopneumonia represented by a focal area of
alveolar filling by polymorphonuclear leukocytes and fibrin. In bronchopneumonia the
alveolar inflammation is initially localized around a terminal or respiratory bronchiole
(not seen in this image), which is the initial site of infection from which it spreads into
peribronchiolar alveoli. (Wiki)
Fig. 68. Note the patchy acute congestion of an organizing pneumonia. (Wiki)
F. Pulmonary abscess: There are several mechanisms by which pulmonary abscesses may develop following blunt force trauma.
A penetrating injury, such as a stab wound, may deposit pathogenic bacteria either in the pleural cavity and or the lung. Aspiration of foreign material, such as fragments of broken teeth or dentures may lead to the development of not only aspiration pneumonia, as shown in Fig. 69, but also an abscess (see Fig. 70) and pulmonary gangrene (see Figs. 71, 72, 73 & 74).
Pulmonary gangrene is a necrotizing, sometimes putrid, pneumonia, which leads to massive necrosis and sloughing of the lung. Typically, it involves the upper lobes, usually on the right side with exception in those cases of aspiration, in which it will generally involve the right lower lobe, the reasons for have already been discussed.
Fig. 70. Pulmonary abscess in right upper lobe. Image courtesy of Yale Rosen, MD. (Wiki)
Fig. 71. The above is a picture of a lung involved with pulmonary gangrene. Note the sharp transition from normal lung to the blackened area. The blackening is caused by oxidative changes that occurred during life in the infarcted, infected tissue. Pulmonary gangrene is caused by a combination of infection and infarction. It differs from necrotizing pneumonia or abscess (Fig. 70) in its extent—usually involving a whole segment or lobe. It is usually caused by bacteria (mostly gram negative), but fungi, tuberculosis, and radiation can also cause it. In one series involving 25 patients, 76% of the patients were males with the upper lobes involved in 80% of cases. Predisposing factors included lung disease, alcoholism, diabetes mellitus, and nutritional deficiency. (Wiki)
Fig. 72. Histologically, pulmonary gangrene is characterized by consolidated air spaces with preserved outlines, which is consistent with coagulative necrosis (note the alveoli are filled with necrotic exudate), as well as variable liquefaction parenchymal necrosis in a setting of pneumonia and thrombosis of a large artery (Fig. 73). (Wiki)
Fig. 73. The above is a photomicrograph of an artery showing arteritis and thrombosis in pulmonary gangrene. The artery (between the arrows) has a necrotizing, destructive angitis and luminal thrombosis. The underlying causative organism may not be visible in the affected artery. However, in this case a Gomori methenamine silver (GMS) stain demonstrated a fungal organism as the underlying cause (see Fig. 74). (Wiki)

Fig. 74. The organisms seen in this photomicrograph are septate hyphae that branch dichotomously at an acute angle. The most likely diagnosis is aspergillus, but because *Pseudallescheria boydii* and some other fungi have a similar appearance, cultures are necessary to confirm the diagnosis. Cultures in this case showed *A. fumigatus*. (Wiki)

**G. Aspiration:** There has been some discussion of aspiration in pages 18 & 19.

Aspiration is the inhalation of liquid or solid materials into the lower respiratory track, usually from the oral or nasal cavities, oropharynx, esophagus or stomach. Once within the lung, the finer and more fluid ingredients flow into the first dependent orifices. If the aspiration should take place with the person lying on their back (supine), the finer and liquid material will most often end-up in the posterior segment of the upper lobe and the superior segment of the lower lobe. If the person is sitting or standing the aspirated material will most often be found in the basilar segments of the lower lobes (see Fig. 75). If the person should be lying on their right side when the aspiration occurs, the aspirated material will typically be found in the apical and posterior upper lobe bronchi. The typical microscopic appearance of aspiration pneumonia is seen in Fig. 69.

Besides blunt force trauma, especially to the craniofascial region, as a cause of aspiration, other etiologies are impaired consciousness, typically due to alcohol, drugs or anesthesia, seizure activity, strokes, dementia and neuromuscular disorders. Surgery involving the nose or oropharyngeal region can result in postoperative bleeding leading to aspiration of blood into the laryngeotracheobronchial tree. Myerson did a study on the incidence of postoperative bleeding following tonsillectomy and found it
occurred in 79 to 100% of patients. Radiologist occasionally observe aspiration while performing upper gastrointestinal tract barium studies.

Fig. 75. The above is an example of aspiration pneumonia involving the right lung. Note the foci of aspiration pneumonia and abscess formation in the right lower lobe. (Free Image Finder) (Wiki)

Huxely et al. demonstrated that most normal people who enter a deep sleep aspirate. Normal individuals typically will clear this material without difficulty or complications. This is because of our acute cough reflex, muco-ciliary activity and alveolar
macrophage response. The induction of pathology due to aspiration is determined by the character, volume and frequency of the aspirated material. Pulmonary changes following aspiration can be divided into two categories, aspiration pneumonitis and aspiration pneumonia. Aspiration pneumonitis refers to acute chemical lung injury due to aspirated gastric acid with or without injury due to aspirated particulate matter, whereas aspiration pneumonia is an infectious process due to inhalation of oropharyngeal secretions, which have been colonized by pathogenic material.

H. Air Embolism: When blunt force chest trauma causes disruption of the pulmonary parenchyma or that of the tracheobronchial tree, there is an avenue for air to enter the circulation. The traumatic injury need not be massive, for it can occur with a needle puncture or stab wound of the lung. There are two types of air embolism, venous and arterial.

Venous air embolism occurs when air enters the systemic venous circulation and travels to the right heart and then to the pulmonary arterial system. To be clinically significant a relatively large amount of air must enter the venous circulation. Other clinically significant factors are the rate of infusion, the position of the person at the time of the venous air embolism and their cardiac status. The minimal amount of air that is needed to enter the venous system to become lethal has not been clearly established. However, most agree, 200 to 300 cc of air rapidly infused into the venous system is sufficient to cause death. Other, however, believe that death can occur with a rapid infusion of as little as 100 cc of air.

Venous air embolism can be the result of trauma, especially knife wounds of the neck. Other causes of venous air embolism the Forensic Pathologist need be aware of are orogenital sexual activity, especially in a pregnant women when the vagina is orally inflated by exhaled air; iatrogenic procedures such as neurosurgery; orthopedic surgery; head and neck surgery; obstetrics and gynecology; positive pressure ventilation; placement of a central venous catheter; and some radiographic procedures. Although, venous air embolism is relatively common in some of these procedures, for example, up to 40% of posterior cranial fossa neurosurgical procedures accomplished while the patient is in the sitting position experience venous air embolism, death is uncommon.
A fundamental point you need to remember is the higher the open vein is above the right atrium, the greater the pressure gradient and the more likely air will enter the vessel. This is also why the head of the bed should be tilted down when inserting or removing a central venous catheter from the jugular or subclavian vein. Thus, in those who have either undergone a neurosurgical procedure, especially involving the posterior fossa with the patient in the sitting position or the victim has sustained knife wounds of the neck, the Forensic Pathologist must be aware of air embolism as a cause of death (see Fig. 76).

![Diagram of a catheter disconnection leading to venous air embolism](image)

Fig. 76. The above is an illustration of a catheter disconnection leading to venous air embolism. Ten minutes after a patient began receiving a hemodialysis treatment, his venous blood line separated from the catheter. Shortly after the venous blood line was clamped, the patient complained of shortness of breath. His respirations were labored at a rate of 28 to 32 breaths/minute. He was immediately placed on his left side in the Trendelenburg's position, started on 10 liters/minute of oxygen by face mask, and transported to the hospital. Despite these measures, he died of an air embolism. (U.S. Food and Drug Administration/U.S. Department of Health and Human Services) (Wiki)
For venous air embolism to occur requires some communication between atmospheric air or some gas and an open venous system, where the pressure gradient favors the entrance of air or gas into the systemic venous system (see Fig. 76). **Arterial air embolism** typically evolves from air within the pulmonary venous circulation entering the left heart and then into the systemic arterial circulation. There are occasions in which air within the systemic venous or pulmonary arterial system enters the systemic arterial system through a right to left shunt via a patent foramen ovale (see Fig. 77) or pulmonary arteriovenous malformation or by simply overwhelming the filtering capacity of the lung. Unlike venous air embolism it only requires a minuscule

![Diagram of heart with patent foramen ovale](image)

**Fig. 77.** The above drawing shows a patent foramen ovale. (Archives of Disease in Childhood) (Wiki)

amount of air (a fraction of a cc) in the systemic arterial circulation to have catastrophic effects, most especially for the heart and brain.

In the living patient the onset of clinical symptoms and signs is typically immediate, with dyspnea (short of breath) being most common. Other manifestations may include
evidence of central nervous system involvement, especially in arterial air embolism, such as fainting, light headedness, anxiety and stroke. The patient may experience chest pain. The specific “mill-wheel” murmur may be heard, however, this is easily missed due to its transient nature. The “mill-wheel” murmur is described as a “characteristic splashing auscultatory sound due to the presence of gas in the cardiac chambers.” Often the heart sounds are normal or there is a loud systolic murmur. At autopsy venous air embolism is evident on opening of the pericardial sac and noting the beaded appearance of the epicardial veins. Remember, a couple of bubbles in the epicardial veins does not make for a diagnosis of air embolism. In venous air embolism air bubbles are seen throughout the length of the epicardial vessels. To insure the diagnosis, insert a syringe needle into the right ventricle, the syringe of which is half filled with water. If air is present, it will bubble through the water in the syringe. Some also advocate filling the pericardial sac with water before insertion of the needle into the right ventricle. This is because when the needle is passed through the wall of the right ventricle, you may see air bubbles in the water filling the pericardial sac. Following this, you can open the right atrium and ventricle. If venous air embolism has taken place the blood will have a frothy appearance (see Fig. 78).
Fig. 78. The above is an autopsy photograph demonstrating another procedure used to confirm the presence of an air embolism. After the pericardium has been opened the pericardial space is filled with water to totally cover the heart. The right ventricle is then punctured with a scalpel, followed by turning the scalpel, which produces ascending air bubbles (arrow). (RadioGraphics-Virtopsy: Minimally Invasive, Imaging-guided Virtual Autopsy) (Wiki)

Regarding air entering the coronary arteries, this is most difficult to demonstrate at the time of autopsy for the simple reason you cannot see through the arterial walls. There is another fundamental point you need to remember, if the patient underwent extensive cardiopulmonary resuscitation, especially if open chest massage was used, it may not only be difficult, but impossible to determine the presents of air embolism, venous or arterial. Also, should the person survived the air embolism but died a short time later you will not be able to demonstrate anatomically air embolism. Likewise, should the person undergo postmortem decomposition, you will not be able to determine air embolism as a cause of death, in part due to postmortem decomposition.
I. Rupture of the diaphragm: Traumatic rupture of the diaphragm is relatively uncommon in those who have sustained blunt force trauma to the trunk (see Figs. 79 & 80). When it occurs it usually is due to trauma to the lower anterior chest most commonly as the result of a motor vehicular accident. Typically, such MVA which cause rupture of the diaphragm are associated with an increase in intra-abdominal pressure. In a study by Shorr et al. MVAs were responsible for 78.0% of ruptures. Other causes were motorcycle accidents, pedestrian accidents, and a kick from a horse. In a study by Yamamoto et al. (2005), rupture of the diaphragm was caused 75% of the time by blunt force trauma, however, in 25% of the cases the rupture was due to penetrating trauma. The rupture usually occurs through the weakest points of the diaphragm, which are in the posterolateral positions along the embryonic fusion lines. However, diaphragmatic rupture has occurred in all areas of the diaphragm.

Fig. 79. The above illustration shows both normal anatomy of the trunk and the anatomy of a tear in the left leaf of the diaphragm causing herniation of the stomach & spleen into the left pleural cavity. The transected aorta was not the direct cause of the rupture of the left leaf of the diaphragm. (Wiki)
Violent upward displacement of the abdominal contents can also rupture the diaphragm, typically posteriorly and centrally. As previously discussed, the left leaf of the diaphragm is most often torn. However, as this entity is being more aggressively pursued clinically, right sided lesions are being more frequently identified. In a study by Shorr et al. 69.5% of their cases had a ruptured diaphragm on the left side and 30.5% were on the right side.

When diaphragmatic tears occur they are most frequently associated with fractures of the ribs and other thoraco-abdominal injuries. What is unfortunate clinically, 90% of the tears in the diaphragm are missed due to the clinical presentation of other injuries. This has serous consequences, because rupture of the diaphragm in-of-itself is associated with a 10% to 40% mortality. However, death is often due to the associated injuries, such as in Fig. 79, which besides the tear in the left leaf of the diaphragm, there was also a laceration in the descending thoracic aorta. In Shorr’s et al. study, of the patients
with ruptured diaphragms, 60.9% had two or more systems involved, with a mortality of 26.9%.

Clinically, there may be a delay in the development of signs and symptoms relative to rupture; the delay being determined by the size and location of the rupture. Again, in Shorr’s et al. study, two of their cases (one with a rupture on the right and the other on the left), the diagnosis was delayed more than a week after the injury. Not only may there be a delay in manifestation of the signs and symptoms, but they may be intermittent. This is due to the intermittent herniation of the abdominal organs, most especially on the left side.

The most serious complication is the development of a tension viscerothorax due to herniation of the abdominal organs through the rupture in the diaphragmatic leaf. This can lead to death. For example, as in Fig. 79, the stomach may herniate into the left pleural space, develop a volvulus (obstruction of the stomach due to the stomach twisting on itself), which in turn leads to prominent distention of that portion of the stomach above the twist as in Fig. 80. This eventually can lead to compression of the left lung and a shift of the mediastinum to the right with attendant cardiac consequences.

**J. Rupture of extrapulmonary airway (Trachea or Bronchi):** Blunt force trauma or penetrating trauma may disrupt the trachea or bronchi (see Fig. 81). However, such ruptures are rare, but when they occur they are serious injuries. Such disruption is associated with an overall mortality rate of 30%, with death often caused by other injuries. Approximately 50% of these patients die within one hour. Penetrating trauma of the tracheobronchial tree typically involves the cervical trachea in 75% of the cases. Clinically it is usually recognized with frothy blood-tinged fluid flowing from the wound. Tracheobronchial rupture due to non-penetrating blunt force trauma are quite rare. When they do occur they are usually related to MVAs. When tracheobronchial rupture does occur it is typically due to compression of the mainstem bronchus against the spine; direct compression by the sternum against a closed glottis; forceful compression to the thorax, which results in the lungs being pulled apart; sudden increase in intraluminal pressure against a closed glottis; transhilar shearing forces on the
tracheobronchial tree between the relative stationary areas of the cricoid cartilage and carina with deceleration forces.

In 80% of the cases with rupture due to non-penetrating blunt force trauma they involve the bronchi, usually the right and within 1 inch of the carina. These ruptures appear as tears, which are parallel to the cartilage rings. When the ruptures involve the trachea they also occur within 1 inch of the carina and are also parallel to the tracheal cartilagenous rings. When non-penetrating blunt force trauma causes tracheobronchial rupture it is often associated with fractures of ribs one through three.

Fig. 81. The above illustration shows the anatomic relationships of the trachea and bronchi. (Wiki)

Tracheobronchial rupture usually leads to a pneumothorax, pleural emphysema and pneumomediastinum. To give you an idea of the seriousness of these injuries, some
studies report an on-scene mortality rate of 80%, with the remaining 20% having a mortality of 30% after arrival at the hospital. Tracheobronchial rupture is often associated with obstruction of the airway. The underlying causes for this airway obstruction are the tongue, teeth or fragments thereof, blood, secretions, dentures, foreign material, fractures of the larynx or cricoid, or a paratracheobronchial expanding hematoma.

**K. Pleural reflex (shock):** Pleural reflex (shock) is a rare phenomenon, which is clinically characterized by a sudden onset of difficulty in breathing, feeling faint, cool pale clammy skin, cyanosis of the lips and nail beds, dilated pupils, distended neck veins, weak pulse, slowing of the heart rate that may culminate into complete heart block. These symptoms and signs can progress to unconsciousness and on occasion sudden death. Pleural reflex is associated with diagnostic needle biopsy of the pleura or lung, pneumothorax therapy, lung operations and thoracentesis. It has also occurred spontaneously in the course of a disease process either involving the pleura alone or involving the pleura and lung.

One of the first reviews of pleural reflex was that by Chabaud, who reported on 3 cases of “nervous accidents” occurring in pleuropulmonary interventions before 1850. In 1864, Roger described what had been known as pleural shock or pleural reflex as an “eclamptic fit,” characterized by cardiorespiratory embarrassment, tonic and clonic contractions of the muscles, loss of consciousness, and in some cases, sudden death. Anderson in 1936 estimated the pleural reflex occurs in approximately one in every 2000 procedures involving the pleura with a fatality rate varying between 15% to 50%. Ormond in 1942 theorized there are two-forms of pleural reflex, one being a reflex syncope and the other due to air embolism.

**Reflex syncope** resembles clinically emotional fainting otherwise referred to as **vasovagal syncope**, which is a vasodilatory sensation. In this reaction the muscle vasodilatory system becomes activated, and at the same time, the vagal cardioinhibitory center transmits strong signals to slow the heart rate markedly. The arterial blood pressure falls rapidly, which in turn reduces blood flow to the brain that causes the person to lose consciousness. This is called **vasovagal syncope**. The pathway probably then goes to the vasodilatory center of the anterior hypothalamus, then
continues to the vagal center in the medulla, after which the pathway goes to the heart through the vagus nerve. The pathway also goes through the spinal cord to the sympathetic vasodilator nerves of the muscles (see Fig. 82).

Remember, parasympathetic innervation of the heart is controlled by the vagus nerve. It is the parasympathetic innervation of the heart which is responsible for slowing the heart rate. The right vagus nerve innervates the SA node (sinuatrial node), the stimulation of which causes bradyarrhythmias (slow heart rate). The left vagus innervates the AV node (atrioventricular node), the stimulation of which predisposes the heart to atrioventricular blocks (see Fig. 83).
Fig. 82. The above is an illustration showing that the sympathetic nerve fibers arise in segments T2 to T4 of the spinal cord and are distributed through the middle cervical and cervicothoracic (or stellate) ganglia and the first four ganglia of the thoracic sympathetic chain. The sympathetic fibers pass into the cardiac plexus and from there to the SA node (Fig. 83) and the cardiac muscle. The effect of the sympathetic nerves at the SA node is an increase in heart rate. The effect on muscle is an increase in rise of pressure within the ventricle, thus increasing stroke volume.

The vagus provides the parasympathetic control to the heart. The effect of the vagus at the SA node is the opposite of the sympathetic nerves, it decreases the heart rate. It also decreases the excitability of the junctional tissue around the AV node (Fig. 83) and this results in slower transmission. Strong vagal stimulation here may produce AV block. (Wesley Norman, PhD, DSc) (Wiki)

![Diagram of the heart with labeled structures](image)

Fig. 83. The above is a drawing showing the location of the SA (sinuatrial) and AV (atrioventricular) nodes. The AV bundle (Bundle of His) leaves the AV node near the lower part of the interatrial septum and splits over the upper part of the interventricular septum into a left bundle branch (LBB) and a right bundle branch (RBB). The cardiac muscle is supplied by branches of these two bundles. (SVC-superior vena cava, IVC-inferior vena cava, AO-aorta, RA-right atrium, RV-right ventricle, LV-left ventricle, and CS-opening of the coronary sinus) (Wesley Norman, PhD, DSc) (Wiki)
The visceral pleura (see Fig. 84) is supplied by the pulmonary branches of the vagus nerve and the sympathetic trunk via the pulmonary plexus, which is an autonomic plexus. The pulmonary plexus also supplies the bronchial tree.

Fig. 84. The above is an illustration of a cross section through the thorax showing the various parts of the parietal and visceral pleura. As shown above the lungs occur within a closed space called the right and left pleural cavities. The inside of the chest wall is lined by the parietal pleura and the lungs by the visceral pleura. Note the visceral and parietal pleura are continuous at the root of the lung. (Wesley Norman, PhD, DSc) (Wiki)

The other form of pleural reflex is due to an **air embolism**. That air embolism serves as one of the underlying causes of pleural reflex has been documented through clinical evidence of cerebral symptoms and signs such as hemiplegia, with the autopsy on such patients showing air in the cerebral arteries and occasionally in the coronary arteries. Also, in those who have survived, air embolism has been seen in the retinal arteries. Rukstintat was one of the first to express the opinion in 1931 that air embolism was generally the cause of pleural reflex.
L. **Adult Respiratory Distress Syndrome (ARDS):** Following severe lung injury, such as severe blunt force trauma to the chest or the victim’s chest being exposed to blast injury from an explosion or aspiration of gastric contents, the lung may experience diffuse alveolar damage. At autopsy the lungs are hard to palpation, often having a gray-red coloration, exuding edema fluid on palpation (see Fig. 85).

![Fig. 85. Heavy red-gray lungs of showing congestion and edema of ARDS.](Arcot J. Chandrasokhar, M.D., Loyola University Medical Education Network) (Wiki)

Microscopically, ARDS is characterized by diffuse alveolar damage manifested by expansion of the interstitium by fibroblastic proliferation with an accompanying mixed inflammatory cellular infiltrate. There is hyperplasia of type 2 pneumocytes. The bronchiolar epithelium may show squamous metaplasia. In the exudative phase, hyaline membranes and patchy alveolar hemorrhage is nearly always found (see Figs. 86, 87 & 88). The organizing phase is characterized by an organizing pneumonia, with thrombi, which are often seen in small pulmonary arteries.

ARDS develops in 82% of patients having more than 20% of their lung tissue contused, whereas only 22% develop ARDS if less than 20% of the lung is contused.
Fig. 86. The above is a photomicrograph of a lung with ARDS. Note the hyaline membranes lining the alveoli and the expansion of the interstitium by fibroblastic proliferation accompanied by a mixed inflammatory infiltrate. (Arcot J. Chandrasokhar, M.D., Loyola University Medical Education Network) (Wiki)
Fig. 87. The above is a photomicrograph showing the pronounced intra-alveolar edema seen early in acute lung injury of ARDS. (Wiki)

Fig. 88. This is another photomicrograph of ARDS. (Arcot J. Chandrasokhar, M.D., Loyola University Medical Education Network) (Wiki)

From a pathophysiologic standpoint in ARDS, there is diffuse loss of surfactant causing alveolar atelectasis. The lungs become stiff and less compliant. Lung volumes decrease and minute ventilation increases as a compensatory phenomenon. Tremendous intrapulmonary shunt develops because of alveolar atelectasis, where there is no ventilation with respect to perfusion.
VI. Complications of Explosions

The anatomic structure most sensitive to air blast wave of an explosion is the eardrum (tympanic membranes). As little as 5 psi in excess of atmospheric pressure can damage the tympanic membrane. Higher pressures can cause damage to the middle ear structures.

The middle ear is that portion of the ear internal to the eardrum, and external to the oval window of the cochlea. Within the middle ear is a cavity, the tympanic cavity, in which there are three minute bony structures called the ossicles (malleus, incus, and stapes; also called the hammer, anvil, and stirrup). These structures transfer the vibration of the eardrum into waves in the fluid and membranes of the inner ear to the oval window of the cochlea.

Following an explosion, if the victim does not show evidence of rupture of the eardrum, it is unlikely other organs will show barotrauma.

Barotrauma to the lungs, which are the second most susceptible tissue to an air blast wave, is responsible for most deaths following an explosion. The resulting blast wave causes air-space enlargement and disruption of alveolar walls. Damage at the alveolar/capillary interface can lead to hemorrhage, air embolism, pneumothorax, hemothorax, pneumomediastinum and subcutaneous emphysema all of which have been previously discussed.

Air embolism is one of the major factors in causing deaths immediately following an explosion. Blast induced intra-alveolar hemorrhage also plays a role in morbidity and mortality. It can also be responsible for massive intraparenchymal hemorrhage leading to an immediate death. On occasion the death of the victim may be delayed, with their eventual death due to progressive pulmonary insufficiency caused by intra-alveolar hemorrhage and edema due to damage to the capillary/alveolar interface or diffuse alveolar damage as in ARDS.

Fat embolism is also commonly seen in victims of blast injury and is believed to make a contribution to death.

There is also a concept brought forth by Elsayed and others that blast injuries can cause loss of pulmonary antioxidant reserves, which leads to free radical oxidative
stress which in turn leads to further lung injury. Also, hemoglobin released from ruptured red blood cells further enhances pulmonary injury due to oxidative stress. Deaths due to pulmonary injury can be due to penetration and/or perforation by projectiles following explosions due to bombs. In point-of-fact, most deaths following explosions due to bombs are due to projectiles. In such deaths, it is imperative that a careful autopsy be done, rather than just an external exam listing the cause of death as due to severe blunt force trauma. Recovery of these projectiles, which may include fragments of the wires, timer, blasting cap, concealment container, shrapnel from antipersonnel devises and fragments of material surrounding the devise at the time of the detonation may offer significant help in identifying the sources of material to construct the bomb as well as the bomb maker.

Explosion related thermal injury may also lead to death or severe injury. These are most commonly the result of structural fires or the ignition of clothing or adjacent materials. The exception of course is an explosion due to an incendiary device, which cause primary thermal injuries. Other explosion related respiratory deaths are smoke inhalation, inhalation of toxic gases (underground mine explosions), compressive asphyxia (structural collapse), and fire related oxygen atmospheric depletion.

**VII. Complications of Blunt Force Trauma to the Heart**

Traumatic injuries to the heart resulting in sudden death was first reported by Fabricius (1537-1619) in the 16th century. Fabricius was a pioneering anatomist and surgeon, who was also known as “The Father of Embryology.” The first case with myocardial contusion was reported by Mark Akenside (1721-1770) in 1764. Mark Akenside was an English poet and physician. Pericardiocentesis as a possible treatment for cardiac injury was suggested by Riolanus in 1649. Joannes Riolanus (1580-1657) was a distinguished professor of anatomy in the University of Paris (Fig. 89). The first successful pericardiocentesis was performed by Dominique Jean Larrey (1766-1842). Dr. Larrey was a French Surgeon in Napoleon’s Army and an important innovator in battlefield medicine (see Fig. 90). In 1897 Ludwig Wilhelm Carl Rehn (1849 -1930) was the first to successfully conduct cardiac surgery, when on September 9,1896 he repaired a stab wound suffered by a 22 year-old Gardner, Wilhelm Justus (see Fig. 91).
Fig. 89. This is a picture of Joannes Riolanus, professor of anatomy, University of Paris. (Wiki)
Fig. 90. The above portrait is of Dominique Jean Larrey, a French Surgeon in Napoleon’s Army. (Wiki)

Fig. 91. This is a portrait of Ludwig Wilhelm Carl Rehn, who performed the first successful cardiac surgery in 1897. (Wiki)

The pathological studies by the German physician Christen Thorel (1880-1935) in 1915 further demonstrated the heart may be damaged by non-penetrating thoracic injuries and that cardiac contusions may be sustained without injury of the skin or fracture of the ribs. As an interesting side note, Thoral identified a muscle bundle in the heart, which connects the sinuatrial and atrioventricular nodes passing medial to the orifice of the inferior vena cava. This bundle of muscle fibers is called Thoral’s bundle.
Cardiac injuries occur in approximately 64% of cases of thoracic organ injuries. In other studies myocardial injury was identified between 15% to 75% of cases with blunt thoracic trauma. These injuries manifest in a variety of pathological conditions including arrhythmias, anomalies of the conduction system, congestive heart failure, cardiogenic shock, hemopericardium, pericardial tamponade, cardiac rupture, valvular rupture, intraventricular thrombus (see Fig. 92), thromboemboli, air emboli, coronary artery occlusion or laceration, ventricular aneurysm and constrictive pericarditis. Cardiac injuries may be more common in the absence of broken ribs or sternal lesions, especially in younger patients with a less rigid chest wall.

The most common causes of blunt force traumatic cardiac injuries are high velocity impacts of MVAs, falls, blows sustained during physical altercations and those sports in which substantive physical impact can occur, as in football, rugby and soccer. Cardiac injury should be suspected when there is evidence of rib or sternal fractures involving the anterior chest wall, contusions of the anterior and lateral chest wall, evidence of seat belt and/or steering wheel injury.

In those cases in which there is penetration of the chest wall, penetration of the heart should be considered if the penetration occurs in an area defined by the right sternal border, left anterior axillary line, 3rd intercostal space, and a line drawn from the xiphoid process to the left anterior axillary line.

The more common cardiac injuries associated with blunt force trauma to the chest are as follows:

A. **Myocardial Concussion**: The heart may sustain a myocardial concussion in which there is no evidence of visible myocardial injury, however, clinically they may show any type of arrhythmia and even experience sudden cardiac death. The ECG may show ST segmental elevations and branch blocks. The ST segmental elevations are thought to be due to transient myocardial ischemia due to coronary arterial spasm.

B. **Myocardial contusion**: The heart may be contused either anteriorly or posteriorly (see Fig. 92). The contusion will manifest as a dark red hemorrhagic appearing area. This gross appearance is due to microscopic interstitial hemorrhage. Contusions of the anterior surface of the heart are due to blunt impact to the chest with the sternum being focally deformed, impacting the pericardium and the epicardial surface of the heart.
Such contusions may involve either the left or right ventricle and/or the interventricular septum. If neither the sternum nor ribs are fractured the area of impact between the heart and chest wall is likely to be large involving much of the anterior wall of the heart. If the sternum and/or ribs are fractured, the anterior surface of the heart may show a focal contusion and/or laceration caused by the fractured ends of the bones (see Fig. 92). Sudden deceleration may also cause a myocardial contusion.

Fig. 92. The above image is from Wounds of the Heart (Including Retained Foreign Bodies), Mediterranean (Formerly North African) Theater of Operations. (Lyman A. Brewar III, M.D., and Thomas H. Burford, M.D., U.S. Army Medical Department) (Wiki)

Posterior contusions of the heart are not uncommonly seen in crushing injuries to the chest, which result in the posterior wall of the heart impacting the vertebral column. Should the posterior surface have a diffuse hemorrhagic appearance, it is very important you take samples of tissue to demonstrate interstitial hemorrhage. If there is no evidence of interstitial hemorrhage and/or structurally damaged myocytes, than the diffuse hemorrhagic appearance of the posterior wall is due to the postmortem settling
of blood (livor mortis). It is also important that you consider the possibility that the cardiac injuries were the result of cardiopulmonary resuscitation, open cardiac message or intracardiac injection. This is one of the reasons it is important for you to review the available medical records.

One of the complications of cardiac contusions is the interstitial hemorrhage and structural damage to the cardiac myocytes may involve the conduction system, which can lead to arrhythmias and sudden death. In any type of arrhythmia ST segment changes may be seen. Sinus tachycardia, atrial flutter or atrial fibrillation are the most common. Clinically you may also see a rise in cardiac enzymes. Typically, a rise of more than 6% in CPK and CK-MB suggest a cardiac contusion. Cardiac troponins (CTI) is considered the gold standard for detecting myocyte injury. It is more valuable than CK-MB. Increase in CTI begins within the first hour and continues for 4 to 7 days. In addition, echocardiographics may show evidence of a pericardial effusion and slowing of the myocardial contraction.

In a study of 31 cases of blunt injury to the chest by Cobanoğlu, 3.8% had pericardial effusion, 3.2% had atrial flutter and 61.5% had various cardiac arrhythmias. Histologic examination may reveal only interstitial hemorrhage with minimal myocyte structural damage either subepicardially or extending deep into the myocardium. If sufficient time has elapsed, typically 30 minutes, you may see leukocytic infiltration and edema. If several days have transpired before death, you may see necrosis. Should the patient die 4 to 6 weeks after the trauma, you may see scar formation. However, in most cases contusions of the heart heal without any evidence of residual injury.

C. Lacerations: Myocardial lacerations may occur anywhere in the heart: ventricles, atria, valves, papillary muscles, chordae tendinae and septae. In one study lacerations involved the right ventricle in 93% of their cases. The right atrium was lacerated in 11.4% and both atria in 11.4%. The right ventricle and right atrium were lacerated together in 17% of cases, while both ventricles were lacerated in 5.7%. Extensive lacerations of both ventricles and atria were seen in 8.57% of the cases. In another study involving 14 patients with cardiac lacerations, four involved the right ventricle, three involved the right atrium, two injuries to the left atrium and one injury to the left ventricle. Four patients had injuries involving a combination of two injured chambers.
Seven of the 14 patients had two or more associated injuries. The lacerations in all of these cases were seen on the anterior surface of the heart. The one case of lacerations of the right atrium occurred without any evidence of external chest trauma. In this latter case, the blunt impact to the chest occurred when the right atrium was filled with blood. Thus, the laceration of the right atrium was due to hydrostatic effects of a sudden increase in intraatrial pressure. The preponderance of the lacerations involving the right ventricle as compared to the left is due to the fact the sternocostal surface of the heart is mainly formed by the right ventricle (see Fig. 93).

The primary cause of lacerations of the heart is severe crushing force impacting the anterior chest wall. Such cardiac lacerations from blunt trauma are usually lethal injuries. Although, there are a few reports in the literature of survival from such injuries, typically, the lacerating injuries to the heart in concomitant association of other visceral injuries most often results in death.

Fig. 93. The above photo shows a laceration of the right ventricle. The patient died fifteen minutes after intubation in the OR. (Trauma.ORG) (Wiki).
D. Rupture: The common sites for non-penetrating traumatic cardiac rupture in decreasing order of frequency are: right atrium, right ventricle, left ventricle, left atrium, interventricular septum and valves. The propensity for rupture to involve the right atrium and right ventricle is not only due to the fact the sternocostal surface of the heart is primarily formed by the right atrium and right ventricle, but also due to the thinness of their walls (see Figs. 94, 95 & 96).

Fig. 94. The truck is wedge under the bridge by its trailer. (www.ispub.com) (Wiki)
Fig. 95. Side view of the truck shown in Fig. 94. (www.ispub.com) (Wiki)

Fig. 96. Myocardial rupture of the right ventricle. (www.ispub.com) (Wiki)
What is of interest clinically is that patients with cardiac rupture, who reach the hospital alive, are often saved by prompt diagnosis and immediate surgical treatment. Survival is more common with right-sided injuries, especially right atrial lacerations. Ventricular septal defects (VSD) are uncommon traumatic heart lesions. If the trauma occurs during the late diastolic isovolumic contraction phase of the cardiac cycle or during early systole when the valves are closed and the ventricles and septum are in a high-pressure state the septal wall can be damaged. The muscular part of the interventricular septum near the apex of the heart, is most likely to be injured. A delayed VSD can occur due to a contusion overlying the anterior or posterior surface of the interventricular septum. Such a contusion can cause an alteration in the microcirculation in the underlying septum leading to necrosis and ultimately a VSD. At the moment of chest impact, the sudden increase in pressure, associated with torsion or pulling effect, may lead to a laceration of the heart including VSD or a burst-lesion of the ventricle with a septal aneurysmal appearance.

Post-traumatic VSD may also occur due to reopening of a healed congenital VSD. Remember, congenital VSDs are more frequently located in the membranous septum, whereas traumatically induced VSDs are located near the apex.

E. Valvular injuries: Valvular injuries due to non-penetrating blunt force trauma to the anterior chest wall are not commonly seen. Valvular insufficiencies occur due to injuries to papillary muscles, leaflets or chordae. According to some of the literature, it is most commonly seen in the aortic valve (see Figs. 97 & 98), then in the tricuspid and mitral valves in decreasing frequency. However, some of the literature list the tricuspid valve as the most common site. Severe mitral insufficiency due to rupture of the chordae (see Fig. 99) and or the papillary muscle is rarely a complication of blunt force trauma. The mechanism of death whenever a cardiac valve, chordae or papillary muscle is lacerated is due to cardiac failure. Some of the literature suggest lacerations of the aortic or mitral valves required more immediate surgical intervention than tricuspid valve injuries, papillary muscle and chordae ruptures, which can be surgically repaired later.
Fig. 97. A 65 year old male truck driver was involved in a high velocity road accident, sustaining a chest contusion and left tibulofibular fractures. The above image contains two photographs. (A) Shows the manubrium-sternum fractures. (B) Shows the intraoperative view of the aortic valve. Avulsion of the left coronary cusp (LCC) through the mid portion (white arrow) was seen. NCC, non-coronary cusp. (Traumatic aortic and mitral valve injury following blunt chest trauma with variable clinical course. C-D Kan and Y-J Yang, Heart 2005;91:568-570) (Wiki)
Fig. 98. A 50 year old male worker fell down from scaffolding about 8 m high. The above image is an intraoperative view of the aortic valve. An intimal tear was noted at the aorta above the NCC area (dotted arrow). There is an avulsion of the left side of the right coronary cusp (RCC) about 8 mm along where the annulus (large arrow) was found. (Traumatic aortic and mitral valve injury following blunt chest injury with a variable clinical course. C-D Kan and Y-J Yang, Heart 2005;91:568-570) (Wiki)
Fig. 99. A 64 year old male worker fell from a 2 m height. He noted chest tightness at the time but the patient did not pay attention to it. One month later, he had dyspnea and a gradually worsening cough with sputum. There are two images. A Cardiac catherization shows severe mitral regurgitation on left ventriculography. (B) Introperative view of the mitral valve through the septum. Primarily, chordae of the A2-A3 segments were ruptured at the level of the tip of the posterior papillary muscle (arrow). (Traumatic aortic and mitral valve injury following blunt chest injury with a variable clinical course. C-D Kan and Y-J Yang, Heart 2005;91:568-570) (Wiki)

F. Coronary artery injuries: Coronary artery injuries as lacerations or rupture are infrequent. In a series of more than 500 autopsies of non-penetrating trauma to the heart, Parmley and colleagues reported only ten cases. The coronary vessel most commonly involved is the descending branch of the left coronary artery (see Fig. 100). Heyndrickx and colleagues reported a case of rupture of the right coronary artery due to non-penetrating chest trauma, which lead to an inferior wall myocardial infarction with inclusion of the posterior septum and a complete atrioventricular block.
G. Comotio Cordis: This is a disruption in cardiac rhythm caused by impact directly over the heart, sternal region, during a particular phase of the cardiac cycle, which is the ascending phase of the T wave, when the ventricular myocardium is repolarizing, moving from systole to diastole. The mechanism at the cellular level is still not completely understood, but is probably related to the activation of mechano-sensitive proteins, ion channels.

It is estimated that impact energies of at least 50 joules are required to cause cardiac arrest, when applied as described above.

A joule is a derived unit of energy or work in the international system of units. It is equal to the energy expanded in applying a force of one newton through a distance of one meter, or in passing an electric current of one ampere through a resistance of one ohm for one second. From a more pragmatic standpoint, utilizing every day life examples, which we can all identify with, one joule is the energy required to lift a small apple one
meter straight up; the energy released when that same apple falls one meter to the
ground; the kinetic energy of a tennis ball moving at 14 mph, and the energy released
as heat by a person at rest every 1/60th of a second. Impacts of up to 180 joules have
been recorded with hockey pucks and lacrosse balls, 450 joules in karate punches and
1028 joules on a punch delivered by a professional boxer.
Commotio cordis typically occurs in boys and young men, average age 15, usually
during sports, more often baseball, however, it can also be caused by a blow from an
elbow or other body parts as indicated above. Commotio cordis is the leading cause of
fatalities in youth baseball in the US, with 2 to 3 deaths per year.
Other sports in which commotio cordis can be seen are football, ice hockey, polo, rugby
football, cricket, softball, fencing, lacrosse, boxing, karate and the martial arts.
Most cases are fatal with fewer than 1 in 5 surviving in some series, whereas other
report a survival of 35% with the more common use of automated external defibrillation.
However, defibrillation must be started within 3 minutes, and can only sometimes
restore heart activity.

VIII. Complications of Blunt Force Trauma to the Great Vessels
A. Aorta: Rupture of the thoracic aorta may occur from direct injury or from impact
applied to the abdomen, which is transmitted along the column of blood in the aorta in
accordance with Pascal’s law.
Pascal’s law, also known as the “The Principle of transmission of fluid-pressure” states
that “pressure exerted anywhere in a confined incompressible fluid is transmitted
equally in all directions throughout the fluid such that the pressure ratio (initial
difference) remains the same.” This law was established by the French mathematician
Blaise Pascal.
Eight-five percent of traumatic injuries to the great vessels in non-military circumstances
are due to penetrating trauma according to Parmley et al. and Sailer. Fifty-seven
percent of penetrating chest injuries are caused by gunshot wounds and 25% by stab
wounds. Among the remaining 15% of great vessel traumatic injuries, most are blunt
force traumatic ruptures of the aorta and the majority of these occur just distal to the
origin of the descending thoracic aorta (see Fig. 101).
Fig. 101. The above is an illustration of the aorta. It is usually divided into five segments.
Ascending aorta: the section between the heart and the arch.
Arch of aorta: the peak part of the aorta that looks somewhat like a “U.”
Descending aorta: the section from the arch of the aorta to the point where it divides into the common iliac arteries.
   Thoracic aorta: the half of the descending aorta above the diaphragm.
   Abdominal aorta: the half of the descending aorta below the diaphragm.
(Wiki)

It is estimated that blunt aortic rupture occurs in 12% to 23% of deaths from blunt trauma. It represents the second leading cause of death from motor vehicular accidents, representing 15% of motor vehicular deaths. Motor vehicular drivers, passengers, or pedestrians hit by vehicles represent 73% to 92% of all cases. Ejection from a vehicle doubles the risk of aortic rupture, with the use of seat belts alone
reducing the mortality risk by a factor of four. The majority (70% - 80%) of victims are male with an average age of 39 (range 3 - 88). Eighty percent of the cases of aortic rupture are due to motor vehicular accidents, with 72% head-on collisions, 24% side impact, and 4% rear impact.

In 1951, Teare showed traumatic aortic rupture to be the cause of death in 8 of 28 victims of an aircraft disaster due to acute flexion of the body over a seat belt. Falls causing aortic rupture are typically from greater than 3 m.

Those who survive aortic transection typically have two or fewer associated serious injuries. For example, according to Parmley’s original report, 42% of patients with aortic rupture had an associated cardiac injury.

According to autopsy series, 36% to 54% of traumatic rupture of the thoracic aorta occur at the aortic isthmus (see Fig. 101). Eight to twenty-two percent involve the ascending aorta, 8% to 18% occur in the arch, and 11% to 21% involve the distal descending aorta. However, surgical series show 84% to 100% of ruptures occur at the isthmus, and only 3% to 10% occur in the ascending, arch or distal descending aorta. The aortic isthmus is 1.5 cm distal to the aortic attachment of the ligamentum arteriosum, just distal to the origin of the left subclavian artery, at the junction of the aortic arch and descending thoracic aorta. The high incidence of rupture at this site depends on the fixation of the aortic arch by the great vessels arising from its convexity, the attachment of the arch to the pulmonary artery by the ligamentum arteriosum, the paired intercostal arteries and the left main stem bronchus. Also, experiments have suggested the aorta can be displaced in a longitudinal direction sufficient to cause traction tears.

As previously discussed the forces applied to the anterior or lateral chest walls are seldom purely linear. Often there are rotational forces which contribute to the resulting injuries. Compression of the chest and or shear wave displacement of the heart and aorta cause distortion and twisting of the two structures relative to one another. Another significant factor in motor vehicular accidents is the sudden deceleration of the victim accompanied by forceful compression of the anterior chest wall, which causes the heart and intrathoracic great vessels to pull away from the posterior chest wall. The deceleration forces can reach several hundred times the force of gravity. The thoracic
aorta is firmly attached to the posterior wall and thus is fixed in position, which leads to
the tearing of the thoracic aorta just below the origin of the left subclavian artery.
Having said above, which is the conventional accepted explanation for tearing or
rupturing the aorta, not everyone agrees with this explanation. Crass et al. argue that
the forces of differential deceleration, torsion, or hydrostatics have inadequate
magnitude in vehicular accidents to result in aortic tearing due to the inherent properties
of the aorta. Several studies have demonstrated that the gravitational forces of
vehicular trauma do not approach the tensile strength of the aorta. Oppenheim and
Zehnder showed the normal aorta can withstand pressure of 2000 mmHg before
bursting. Considering these studies, Crass has proposed a different mechanism to
account for aortic tears, which is based on thoracic compression. In this hypothesis the
anterior thoracic osseous structures (manubrium, first rib and clavicular heads) rotate
posteriorly and inferiorly about the axes of the posterior rib attachments. When the
force is large enough, these anterior bony structures impact the vertebral column and
the portion of the aorta overlying the spine (the isthmus and proximal descending aorta),
which are pinched between the bones. This causes direct shearing of the aorta.
Crass’s group demonstrated in their research using dogs that a blunt force as little as
20,000 N can cause the rotation posteriorly and inferiorly about the axes of the posterior
rib attachments and thus transect the aorta. In comparison, a 38 mph collision
produces a force of 198,000 N in an average sized adult.
Another theory was proposed by Rice and Wittstruck in 1951, who envisaged the
central portion of the descending aorta snapping forward by the momentum of the
decelerating force of the mass of blood within the vessel. As the upper part of the aorta
is fixed by the great vessels arising from the arch and the ligamentum arteriosum, it
would decelerate at the same rate as the whole body. However, because of its less
rigid fixation, the descending aorta decelerates at a different rate, the difference
constituting a force concentrated at the point of maximum fixation. This hypothesis has
been somewhat disproven by the failure to find fairly constant intercostal bursting and
laceration of the intercostal arteries should this theory have any validity.
When assessing ruptures of the ascending aorta or arch of the aorta there are three
lines of thought. The first, expressed by Vince and Dominick DiMaio in their book,
“Forensic Pathology,” occurs when severe blunt force trauma impacts the anterior chest wall and compresses the heart and intrapericardial portion of the ascending aorta, producing sudden rise in intracardiac and intraluminal pressure, that results in a transverse tear of the aorta immediately above the cusp of the aortic valve. The second line of thought, expressed by Bennett and Cherry in their article “The natural history of traumatic aneurysms of the aorta,” is that the anterior location of the ascending aorta and the weight and ease of displacement of the heart downward and to the left facilitate traction stress on and immediately above the aortic root. The third hypothesis for transverse rupture of the ascending aorta is the valve commissures may exert a rocking force and pull on the aortic wall as the valves are closed in diastole by the column of blood above it, the aorta being also firmly attached to the underlying pulmonary artery. More recently, aortic rupture of both the ascending and descending aorta have been attributed to the deployment of air bags in which in some cases the cars were moving at speeds less than 10 mph. Rupture of the descending thoracic aorta can occur with hyperextension of the spine with its attendant shearing forces. As shown above, there are many different mechanisms of trauma, i.e., front and side impacts, falls, crushing injuries and blasts, which can cause aortic disruption, each with its own application of blunt impact forces. Thus, it is not surprising that there are a variety of different mechanisms, which are responsible for the resultant morbidity and mortality.

It is important to remember survival rates are based on both autopsy and operative series; autopsy series tend to underestimate the rate of long-term survival. It is also important you note when the reviewed study or series was accomplished. For example, in 1958 Parmley and colleagues reported that 86% of patients died at the scene, and 11% survived longer than 6 hours. In-point-of fact, the only survivors in Parmley series were operated on. In more recent surgical series, mortality rates varied from 11% to 40%.

B. Traumatic induced aneurysms of the Thoracic Aorta: This is a much rarer injury than traumatic rupture. Its underlying genesis, however, is closely related to traumatic rupture, as its formation depends on the tensile strength of the adventitial tissues, which
prevent complete rupture at the time of injury, thus leading to survival for sufficient time to cause aneurysm development. Both fusiform and saccular aneurysms can be formed, depending on whether an entire segment of the arterial wall is involved or only a small segment (see Fig. 102). The rapidity of the development of the aneurysm is a function of the extent of damage to the outer coats of the aorta (medial and adventitia) of the aorta.

One of the first reports of traumatic induced aneurysms was by Rindfleisch in 1893. In 1961, Spencer and colleagues reported eight instances of traumatic aneurysm in a series of 15 cases of aortic “rupture” with a total of 60 cases reported in the English literature to that date. In 1962, Bowen and Teare reported two cases. The first was a pedestrian aged 45 years, who died five hours after being struck by a motor vehicle and the second a 21 year old motor cyclist who survived multiple skeletal injuries and
hemorrhage, dying unexpectedly 13 days later from a ruptured traumatic aneurysm of the aorta.

It is important to keep this entity in mind especially in the first few weeks following injury. Spencer and colleagues reported no sudden deaths following ruptured traumatic aneurysms more than a few months old. In seven out of eight patients aneurysms were successfully repaired, but there was only one survival in seven cases of aortic rupture.

C. Great Vessel injuries: Injuries to the great vessels arising from the arch of the aorta (left brachiocephalic, left common carotid and left subclavian), axillary artery, azygos vein, superior and inferior vena cava and pulmonary hilar vessels are life threatening (see Figs. 103, 104, 105 & 106). Most of these patients also have severe concomitant injuries and die at the scene or are dead on arrival at the hospital. Clinical diagnosis of great vessel injury is made by having a high index of suspicion from the mechanism of injury, associated injuries, physical examination and a widen mediastinum shown on the chest x-ray. An aortic arch arteriogram will confirm the clinical suspicion.
Fig. 103. The above illustration depicts the great vessels arising from and going into the heart and the immediate branches thereof. (Wiki)

Fig. 104. The above illustration shows the systemic circuit, venous side. (Wiki)
Fig. 105. The above illustration is the systemic circuit, arterial side. (Wiki)
The Shock Trauma Center of the Maryland Institute for Emergency Medical Services Systems did a retrospective analysis of 515 cases of blunt chest trauma. Nine of these patients presented with great vessel injuries other than aortic tears. These included three patients with innominate (brachiocephalic) artery injuries, two patients with subclavian artery avulsions, two patients with axillary artery injury, one patient with an injury of the azygos vein, and one patient with an avulsed pulmonary artery and vein. There were two deaths and three major complications. One death occurred 48 hours after injury and was due to significant preoperative hypotension and low flow state. The second death occurred over 4 weeks after the incident and was due to sepsis. The complications included empyema, brachial plexus injury, and sternal dehiscence.

Regarding associated injuries, data from the American Association for the Surgery of Trauma Trial (AAST) showed 51% of patients had an associated closed head injury, 46% had multiple rib fractures, and 38% had pulmonary contusions. Although many other autopsy studies demonstrated the majority of patients had associated cardiac
contusions, the AAST study suggested the incidence was only 4%. Orthopedic injuries remain common, occurring in association with aortic rupture in 20% to 35% of cases.

**D. Chronic Traumatic False Aneurysms:** Approximately 2% to 5% of patients with aortic disruptions survive without operations but go on to form traumatic false aneurysms. It appears these aneurysms form, as do false aneurysms typically form, as a communicating hematoma. A false aneurysm differs from a true aneurysm in that the former is not enclosed by any of the normal coats of the vessel (see Figs. 107 & 108).

![Diagram of false aneurysm](image)

Fig. 107. A false (pseudoaneurysm) is usually caused by an injury to the aortic wall leading to blood clot formation outside the outer layer of the aortic wall where there is containment of the blood by the adjacent organs or structures. This is otherwise known as a pseudoaneurysm. In false aneurysms there is no involvement of any layers of the aortic wall, which differs from the true aneurysms and dissecting aneurysms (see Figs. 108 & 109). In the chronic phase, the fibrous tissues surrounding the blood clot thicken and may resemble the aortic wall. False aneurysms may be associated with aortic ulcerations and traumatic aortic injuries. Patients may present with pain or symptoms related to compression of adjacent organs. (The Mount Sinai Hospital) (Wiki)
Fig. 108. The above illustration shows the classification of aneurysms. All three tunica layers are involved in true aneurysms (fusiform and saccular). In false aneurysms, blood escapes between tunica layers, which separate. If the separation continues, a clot may form, resulting in a dissecting aneurysm. (From Copstead and Banasik, 2000) (Wiki)
Fig. 109. In the above illustration the normal aorta is on the left and the aorta with the dissecting aneurysm is on the right. (Wiki)
Fig. 110. The above illustration shows the origin of the dissecting aneurysm. The aortic dissection creates an aneurysm in which a tear develops in the intima (inner lining of the aorta) allowing blood to leak into the media (middle layer). This creates two passages for blood: a true lumen, which is the norma passageway of blood, and a false lumen, which is not lined by the intima but is a newly created passageway. (Wiki)

The formation of a false aneurysm requires first the formation of a hematoma in association with an underlying defect in the intima of the aorta as depicted in Figs. 107 and 108. For the false aneurysm to form there must be continued patency of the arterial defect, so that pressure is transmitted through the defect in the aorta into the perivascular blood clot. This is followed by clotting of the blood in the perivascular hematoma. However, because of the continued patency of the defect in the aorta, the central portion of the perivascular clotted hematoma is hallowed out by the pressure transmitted from the lumen of the aorta. At the same time as the central portion of the hematoma is being hallowed out, the peripheral portion is penetrated by fibroblasts and undergoes organization to form a fibrous wall. As the central canal continues to enlarge, the entire mass forms a sac, the lining of which is continuous with the endothelium of the aorta, with the outer surface of the sac becoming attached to the
surrounding structure. This differs from the dissecting aneurysms in that there is a tear in the intima of the aorta allowing blood to flow within the layers of the aorta, typically the media (see Figs. 109 & 110). These false aneurysms may evolve into a saccular or fusiform aneurysm, with continued expansion and ultimately may even rupture. Ninety percent involve the aortic isthmus. These patients typically have fewer associated injuries at the time of the traumatic event. Typically, 35% have no other injuries, and 50% have only one.
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