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CE Chest Radiograph Evaluation and Interpretation

Debra Siela, RN, PhD, CCNS, ACNS-BC, CCRN, CNE, RRT

ABSTRACT

Chest imaging is an important tool in managing critically ill patients. Basic chest radiology is still used to quickly detect abnormalities in the chest. Critical care nurses are often the ones who first read the radiologist's report of chest radiograph results and provide their interpretation to a physician. Oftentimes, chest radiographs are obtained routinely on a daily basis for every critical care patient, with the goal of effective clinical management. Critical care nurses can confirm cardiopulmonary assessment findings by also evaluating their patient's chest radiographs and reviewing the radiologist's report. By learning some basic skills in interpreting and evaluating chest radiographs, nurses can recognize and localize gross pathologic changes visible on a chest radiograph. This article provides basic chest radiograph interpretation information that allows readers to review relevant anatomy and physiology, summarize normal and abnormal findings on chest radiographs, and describe radiographic findings in common pulmonary and cardiac disorders.
Keywords: APN, chest imaging, chest radiograph, chest radiograph evaluation, chest radiograph interpretation, critical care nurses

Chest imaging is an important tool in managing critically ill patients. Basic chest radiology is still used to quickly detect abnormalities in the chest. Critical care nurses are often the ones who first read the radiologist's report of chest radiograph results and provide their interpretation to a physician. Oftentimes, chest radiographs are obtained routinely on a daily basis for every critical care patient with the goal of effective clinical management.

Debate exists about the efficacy of daily or routine chest radiology for critically ill patients. It has been suggested that daily or routine serial chest radiographs are not needed. Some research supports obtaining daily routine chest radiographs for critically ill patients to be able to identify even subtle changes.[1–3] Mettler[4] supports daily chest radiographs in critically ill patients. Mettler reports that in daily chest radiographs, 60% do not disclose either new major or minor findings, 20% have new minor findings, and 20% have new major findings that are clinically unsuspected and are seen only on the radiograph.[4]

Other research supports discontinuing daily routine chest radiographs for critically ill patients because subtle changes may not be clinically significant and because of the use of resources and cost.[5–7] Some consensus exists for routine but not daily chest radiographs depending on the nature of the acute illness.[8] Many of these studies[1,3,9] suggest using clinical assessment to guide the need to obtain confirmatory chest radiographs whether or not they are daily or routine.

Critical care nurses can confirm cardiopulmonary assessment findings by also interpreting

Debra Siela is Assistant Professor, Ball State University School of Nursing, and ICU Clinical Nurse Specialist, Ball Memorial Hospital, 2000 University Ave, Muncie, IN 47306 (dsiela@bsu.edu).

their patient's chest radiographs and reviewing the radiologist's report. This process can aid in planning appropriate nursing care. This article provides information on basic chest radiology interpretation that will allow the reader to review relevant anatomy and physiology, summarize normal findings on chest radiographs, and describe radiographic findings in common pulmonary and cardiac disorders.

Basic Chest Radiography

X-rays are very short wavelengths of electromagnetic radiation that penetrate matter.[4,10–12] A traditional radiograph is created when x-rays penetrate a structure and produce images on a piece of photographic film usually contained in a cassette. However, in most hospitals and medical centers, the traditional x-ray film has been replaced with digital images. Special detectors are used to replace the film in the cassettes and convert the x-ray energy into digital signals to construct a digital radiograph. The digital images are stored on and distributed on a picture archiving and communications system (PACS).[4,10–13] A PACS allows viewing access far from the radiology department at any computer workstation at any time. Digital radiographs can be manipulated to alter contrast and brightness or magnify images to see any abnormality. Every sample radiograph included in this document is from a digital format.

Each radiograph has a continuum of shades from black to white in its images due to the way the body structures or tissues absorb the x-ray beam.[4,10–13] X-rays penetrate body tissues that have minimal tissue density, such as air or air-filled structures, and produce black or dark areas on the radiograph; these areas are referred to as *radiolucent*. Areas or body tissues that cannot be penetrated by x-rays are *radiopaque* and appear light or white on the radiograph. Thus, each body tissue or structure has different radiodensity.

The 4 basic roentgen densities or radiodensities[4,10–13] are

- gas (air), which appears black or radiolucent; examples are gas or air in trachea, bronchi, or stomach;
- fat, which appears gray or less radiolucent than air; an example is lipid tissue around muscle;
- water (soft tissue), which appears white with slight radiopacity; examples are the heart, blood vessels, muscle, and diaphragm; and
- bone (or metal), which appears all white or completely radiopaque; examples are bones, calcium deposits, prostheses, and contrast media.

Figure 1 provides a review all of the chest tissues and structures basic radiodensities in a chest radiograph, and Figure 2 identifies each chest structure or tissue on a frontal chest radiograph (note that all figures appear at the end of the article).

If 2 structures of equal density are adjacent to each other, the border of neither structure can be detected. This phenomenon, the silhouette sign, is used to identify normal chest structures and diagnose and localize lung diseases.[4,10–13] The silhouette sign may be used to distinguish anterior from posterior structures on a chest radiograph.[4,10–13] For example, a silhouette sign would be expected in an area of consolidation in the left upper lobe of the lung because this lobe borders the left sides of the atrium and the mediastinum. Because both the area of consolidation and the heart are water densities, the left border of the atrium cannot be distinguished from the border of the left upper lobe of the lung (Figure 3).

Views of the Chest

Two of the most common radiographs are posteroanterior (PA) and anteroposterior (AP) or frontal views of the chest.[4,10–13] For PA views, the x-ray beam passes through the chest from the back to the front. For AP views, the beam passes through the chest from the front to the back. For acutely ill patients who cannot stand up for a PA view, AP views are obtained with a portable x-ray machine. Ketai and coworkers[13] report that more than half of all chest radiographs in hospitals are performed at the bedside. Many of the sample radiographs in this article are AP views.

The next most common view of the chest after the frontal view is the lateral view.[9] Lateral views of the chest enable detection of lesions behind the heart, near the mediastinum, or near the diaphragm.[10,12] The lateral view also allows for visualizing the tracheal air column, inferior vena cava, retrosternal space, posterior margin of the heart, and diaphragmatic contour.[10,11] A patient's frontal and lateral view radiographs allow the viewer to have a vision of the chest in 3 dimensions so that the viewer can more easily localize infiltrates and lesions.

In addition, if ventricle enlargement is suspected on the frontal chest image, a lateral view may help confirm the finding. Portable lateral view chest images are also used to evaluate presence of pleural effusions that cannot be seen on frontal views because they are small in size.[10] It takes 250 mL of fluid to blunt a lateral costophrenic sulcus on a frontal radiograph, but only 75 mL to blunt the posterior costophrenic sulcus on a lateral radiograph.[10] Sometimes, a lateral decubitus view radiograph (patient lying either on one side or on the other) is obtained to evaluate possible pleural effusions and their fluid levels.[13] Figure 4 shows lateral view chest structures and tissues.

Technical Factors of Viewing Chest Radiographs

It is necessary to consider whether each of the following factors are adequate or appropriate to accurately assess and evaluate normal and abnormal chest radiograph findings.[10–13]

Penetration

X-rays must adequately penetrate body structures to visualize the structures. For example, one should be able to faintly see the thoracic spine through the heart shadow.[10] If you cannot, the chest radiograph is underpenetrated or too light. In this situation, the left hemidiaphragm may not be visible because the left lung base can appear opaque, which may hide or mimic true disease in the left lower lung field.[10] A lateral view will be necessary to confirm any abnormalities in the left lower lung field. In addition, the pulmonary markings may appear more prominent then they really are and may be interpreted as interstitial pulmonary edema or pulmonary fibrosis.[10] Again, a lateral view will be necessary to confirm the interstitial findings.

If the chest radiograph is overpenetrated or too dark, the lung markings may appear to be absent or decreased.[10] It is then possible to make the judgment that the patient has emphysema or pneumothorax when in reality this pathophysiology does not exist.[10] One could also miss a pulmonary nodule when the chest radiograph is overpenetrated.[10]

Inspiration

A full-inspiration chest radiograph can be reproduced from one time to the next to eliminate the possibility of artifacts that may confuse the viewer to think that disease is present.[10] If one can count 10 posterior ribs above the diaphragm, it is an excellent inspiratory film.[10] When less than 10 ribs can be counted above the diaphragm, it is either poor inspiratory effort or a sign of low lung volume. Low lung volume from a poor inspiration effort can crowd and compress the lung markings, producing the impression that a lower lobe pneumonia is present.

Rotation

In AP radiographs, patient malposition or rotation may appear to indicate abnormalities in cardiac, vascular, or mediastinal contours when, in fact, they may not exist.[11,13] The position of the clavicles help identify a patient's rotation.[11,13] If one clavicle appears to be shorter in length than the other, then one side of the chest may be rotated close to or away from the detector cassette, producing what appear to be abnormalities.[11,13] Figure 9 shows a difference in clavicle length. Comparing the length of the clavicles in addition to comparing the symmetry of the distance between the spinal pedicles and clavicle heads as a method of identifying chest rotation is an important part of the radiograph examination. If one identifies asymmetry in the distance between the pedicles (outer edge of spinal vertebra) and the clavicle heads, the chest is likely rotated.[10,13] Figure 9 shows an example of asymmetry between the spinal pedicles and the clavicle heads due to chest rotation.

Magnification

Anteroposterior views obtained with a portable machine have some disadvantages. Structures in the anterior part of the chest are magnified on AP views, so structures such as the heart are not as distinct as on PA views and may even be distorted.[4,10–13] The heart and the mediastinum appear about 15% wider than on the PA view.[13] This phenomenon occurs on an AP view mainly because of the shorter distance between the x-ray tube and the patient than occurs in a PA view.[4,10,11]

Angulation

Posteroanterior views are sharper and more distinct with less chest rotation and have consistent clavicle placement because they are always obtained with the patient upright and 2 m (6 ft) away from the source of the x-rays and at a 90° angle to the beam, whereas angles less than 90° are often used for AP radiographs because of inability of critically ill patients to

be positioned at 90°.[12] Thus, the x-ray beam ends up entering the chest with the patient's head and chest tilted backward.[10] This creates what is called an apical lordotic view of the chest.[10] In a chest radiograph that is an apical lordotic view, the clavicles project at or above the posterior first ribs and make the clavicles appear to be straight.[10] The apical lordotic view can create the illusion of cardiomegaly when, in fact, it does not exist.[10]

Systematic Method of Viewing

A systematic method should be used to examine chest radiographs.[4,10–12,14,15] Radiographs may be examined from side to side, from top to bottom, or structure by structure. Following is the suggested order for structure-by-structure examination of the frontal view (PA or AP) chest radiograph, which can all be viewed in Figure 2.

1. Soft tissues
2. Trachea
3. Bony thorax/ribs
4. Intercostal spaces
5. Diaphragm
6. Structures below diaphragm
7. Pleural surfaces
8. Mediastinum
9. Hila
10. Lung fields
11. Support catheters, tubes, wires, and lines

Following is the suggested order for viewing lateral view structures, which all can be seen in Figure 4.

1. Bones
2. Mediastinum
3. Hilum
4. Heart
5. Diaphragm/pleura
6. Lung fields
7. Support catheters, tubes, wires, and lines

Viewing of Normal Chest Structures

Soft Tissues

The soft tissues of the chest consist mainly of fat and some water densities.[4] The tissues should appear symmetric when compared side to side. Breast tissue is an example of soft tissue. Sometimes, breast tissue shadows obscure the lower lung tissue (Figure 5).[4] Subcutaneous emphysema results from air in the subcutaneous layer of the skin or soft tissues.[10] Subcutaneous emphysema commonly occurs from penetrating chest injury or a chest tube.[10] Figure 6 shows subcutaneous emphysema.

Trachea

The trachea appears as a column of radiolucent tissues or gas density midway between the clavicles or over the spine. The carina is normally positioned approximately at the level of the sixth posterior rib or T4 (Figures 3 and 7).[11–13] When an endotracheal (ET) tube is placed correctly, the tip of the tube is approximately 3 to 5 cm (approximately 2 in.) above the carina. Figure 8 shows a chest radiograph with an ET tube down in the right main stem bronchus.

Tracheal deviation is present when the trachea is positioned to the right or to the left of the midline. A common cause of apparent tracheal deviation is chest rotation (Figure 9). True tracheal deviation may be caused by the presence of a tumor, mediastinal shift, pneumothorax, or major atelectasis. Figure 25 shows the tracheal deviation due to atelectasis.

Bony Thorax

The humeri, scapulae, clavicles, spine, and ribs should be identifiable as bone densities. On a radiograph obtained during inspiration, 9 to 10 posterior ribs to the lateral top of the diaphragm should be visible (Figure 2). Each rib should be followed along its course to assess for any notching or deformities; symmetry of rib structures should be assessed bilaterally. Radiographs of patients with chest trauma should be checked for evidence of rib fractures.

Intercostal Spaces

Each intercostal space is numbered according to the rib above it. The width of the intercostal spaces is determined by measuring the degree of the costovertebral angle relative to the posterior ribs. The normal angle is about 45°; with widened intercostal spaces, the angle may double to more than 90°.[11–13] Widened intercostal spaces occur in conditions such as chronic obstructive pulmonary disease, pneumothorax, and pleural effusion that increase lung volume.[11–13] Figures 9 and 10 show an example of widened intercostal spaces. Narrowed intercostal spaces, such as that occurs in atelectasis and interstitial fibrosis, are associated with conditions that decrease lung volume.[11–13] Figures 22 and 30 show an example of narrowed intercostal spaces.

Diaphragm

The diaphragm has a water density, and each hemidiaphragm is dome-shaped. The right hemidiaphragm is normally higher in the chest than is the left hemidiaphragm because of the liver (Figure 2).[11–13] Diaphragmatic elevation is evident when fewer than 9 to 10 ribs are visible and can be caused by abdominal distension, phrenic nerve compression, or lung collapse (atelectasis; Figures 22, 24, and 28).[11–13]

Diaphragmatic depression is present when 11 to 12 ribs are visible.[11–13] Depression or flattening of the diaphragm is associated with hyperinflation of the lung or thorax as in chronic obstructive pulmonary disease and pneumothorax.[11–13] See examples of diaphragm depression (flattening in chronic obstructive pulmonary disease, Figure 9, and also in a pneumothorax, Figure 10).

Structures Below the Diaphragm

Almost all of the structures below the diaphragm are primarily water densities. The exception is the gastric air bubble, which may be visible in many frontal radiographs (Figure 2).[11–13]

Pleural Surfaces

The pleurae normally appear as thin, hairlike lines along the lateral edges of the chest and along the diaphragm. When the pleural line deviates medially and appears in the lung fields, a pneumothorax may be present.[11–13] When a pneumothorax is present, the area outside the line to the lateral edge of the chest (eg, the pleural space) will appear more radiolucent or completely black.[11–13] The costophrenic sulcus (angle) may also be displaced inferiorly and increase the lucency at the same time in a large pneumothorax. This is termed the *deep sulcus sign*.[10] The lung itself will appear more radiopaque or more dense (Figure 10). Mediastinal shift toward the affected lung may also occur in a large pneumothorax. In contrast, in tension pneumothorax, the mediastinum shifts away from the affected lung, and the diaphragm commonly is depressed on the affected side.[12] See reinflated left lung with chest tubes in Figure 11.

The costophrenic sulcus/angle (Figure 2) should appear as deep sharp points like a V on a frontal view. If the costophrenic sulcus are not distinct deep points on the lateral sides of the chest and appear blunted, a pleural effusion may be the cause.[11–13] If a horizontal fluid level or meniscus can be visualized in the area of the costophrenic sulcus, a pleural effusion most likely has occurred.[11–13] It takes approximately 250 mL of fluid to blunt the costophrenic sulcus on a frontal view radiograph.[10] Right-side pleural effusions occur frequently in heart failure. Figures 8, 12, and 32 show pleural effusions.

Mediastinum

The mediastinum includes the heart, major blood vessels, the trachea, and the right and left main bronchi. The heart and blood vessels are water densities, whereas the trachea and bronchi are air densities. The right atrium forms the right border of the heart. The right ventricle cannot be detected directly on a chest radiograph because this structure is located in the center of the heart shadow. The superior vena cava is visualized above the ascending aorta shadow in the right chest.[12] An indentation can be located down in the mid-hilar area that marks the junction of the right atrium and superior vena cava (Figure 2). This is the area where the tips of central venous catheters should be located and where a pulmonary artery catheter should enter the right side of the heart.[10]

The left atrium and the left ventricle form the left border of the heart. The aortic arch is visible as the aortic knob on the frontal view. The descending aorta is about 3 cm above and to the left of the carina (Figure 3).[11–13] The main pulmonary artery is the next shadow contour below the aortic knob on a frontal view radiograph.[10] Sometimes, the left descending pulmonary artery can be seen as far down as the next contour shadow following the main left pulmonary artery (Figure 2).

The cardiothoracic ratio can be determined to assess the overall size of the heart.[11–13] This ratio is determined by measuring the horizontal width of the heart and dividing that width by the widest interval of the thorax. The normal cardiothoracic ratio is 1:2 or less (Figure 13).[11–13] A cardiothoracic ratio greater than 1:2 is suggestive of cardiac enlargement.[11–13]

Cardiac enlargement can be determined more precisely by comparing the findings on serial radiographs.[13] An increase in the diameter of the heart of 1 cm or greater is considered cardiac enlargement.[13]

If cardiac enlargement appears to be present, right ventricular enlargement must be distinguished from left ventricular enlargement. Frontal view radiographs show the following during left and right ventricular enlargement:

- The left lower border of the heart is moved laterally and becomes rounded in left ventricular enlargement (Figure 14).[12,13]
- The right atrium protrudes into the right side of the chest and becomes more convex in right ventricular enlargement (Figure 15).[12,13]

Left and right cardiac enlargement is likely in the lateral view when

- the left heart border moves inferoposteriorly beyond the inferior cava toward the spine in left ventricular enlargement (Figure 16).[12,13]
- the right ventricle shows enlargement by moving anteriorly and superiorly with filling of the anterior retrosternal clear space. Normally, on the lateral view, the lower one third of the right ventricle contacts the sternum (Figure 4). Whereas, in right ventricle enlargement, the right ventricle contacts the lower one half of the sternum.[12,13] See Figure 17.

The mediastinum can also be widened for many reasons, including focal masses or fluid infiltration. Focal masses usually cause sharp and convex mediastinal widening.[12] Mediastinal infiltrations come from hemorrhage or infection and produce widening that appears generalized and diffuse.[12] See Figure 18, which shows a wide mediastinum for a new postoperative cardiac surgery patient. Figure 19 shows a thoracic aortic aneurysm, which may be suspected because of the widened thoracic aorta, which also widens the mediastinum.

Hila

The hila consist of the pulmonary arteries and veins and appear blotchy because of various areas of radiopacity associated with different sizes and thickness of blood vessels. The heart shadow obscures the left hilar area and makes the left hilum appear smaller and higher than the right hilum.[13] The left hilum is positioned slightly higher than the right hilum in 97% of all individuals.[12] Bronchovascular markings refer to blood vessels and bronchi that branch out from the hila to the periphery of the lung fields (Figure 2).[12] As the markings extend out into the lung fields and gradually taper off in the periphery, the structures consist of mainly pulmonary blood vessels and no bronchi.

The pattern of pulmonary venous hypertension is seen in patients with elevated pulmonary venous pressure, which is usually caused by left ventricular failure and increased hydrostatic pressure.[16] You will note increased prominence and thickening of the upper lobe blood vessels in the hila, decreased prominence of the lower lobe blood vessels, and haziness of the hilar vessels, which is the process of cephalization.[10,11] Figure 20 shows pulmonary venous hypertension.

Pulmonary artery hypertension is caused by conditions that decrease the flow of blood through the pulmonary capillary bed such as emphysema, pulmonary emboli, and vasoconstrictive states.[16] The hilar trunks are enormously dilated in response to the constricted arterial bed.[11] See Figure 21 to view pulmonary arterial hypertension.

Hilar elevation is usually present in collapse of the upper lobes of the lung, whereas hilar depression occurs in collapse of the lower lobes of the lung.[13] Collapse of the right middle lobe does not cause hilar displacement.[13]

Lung Fields

The lung fields consist mainly of air and very little tissue or blood.[11–13] As a result, lung fields should be visualized as an air/gas density or as a completely radiolucent area. Abnormalities of the lung fields on chest radiographs include interstitial patterns and alveolar consolidation patterns. See further descriptions of interstitial and alveolar consolidation patterns under radiographic signs of cardiac and pulmonary disease.

Determining the locations of the various lobes of the lungs is useful for diagnosing and localizing pulmonary disease.[11–15] The right middle lobe is located anteriorly, adjacent to the right side of the heart. The left lingular lobe is in direct contact with the left border of the heart.[12] The upper lobes and the right middle lobes of the lung are primarily anterior thoracic structures, whereas the lower lobes are primarily posterior structures. Knowing the different locations of the lung lobes makes it possible to use the silhouette sign. See Figure 3 for a left upper lobe lung consolidation that borders the left heart. See Figure 21 for a right lower lobe pneumonia where the right-sided heart border is distinct. See Figure 28 for a left lower lobe infiltrates with distinct left-sided heart border. See Figure 30 for an example of complete consolidation where the heart cannot be identified.

Fissures are separations or spaces between the lung lobes and appear as narrow white lines on a chest radiograph.[11–13] Fissures are normally the thickness of a sharpened pencil line.[10] In frontal and lateral views, the area

where the minor fissure separates the right middle lobe from the right upper lobe may be visible. The minor fissure is located almost in the middle of the right lung fields, where it appears as a horizontal line on the frontal radiograph (Figure 22).[12] One of the direct signs of lung collapse in the right upper lobe on a frontal radiograph is elevation of the minor fissure (Figure 23).[11–13] In collapse of the right middle lobe, downward displacement of the minor fissure may be evident.

The major or oblique fissures separate the upper lobes of the lung from the lower lobes. The major fissures cannot be seen on a frontal view but are visible on a lateral view.[13] See Figure 4 to view the major fissure.

Support Catheters, Tubes, Wires, and Lines

Critically ill patients commonly have support catheters, tubes, wires, and lines. This invasive support equipment includes, for example, ET tubes, tracheostomy tubes, chest tubes, nasogastric or feeding tubes, central line catheters, peripherally inserted central catheter lines, pulmonary artery catheters, and pacemaker wires. Invasive support equipment is often evaluated as to its position in the chest or abdomen on a chest radiograph. See Table 1 for a list of common catheters, tubes, wires, and lines that are inserted in the chest with their proper location in a chest radiograph.

Nurses should be aware of the correct positions of this support equipment in the chest and abdomen and on a radiograph. The radiographs in Figures 24 and 25 contain various support equipment in both correct and incorrect positions. Several other examples of chest radiographs also show support equipment in both correct and incorrect positions (Figures 3, 6, 8, 16, 18, 27, 32, and 35). However, identifying that a catheter, tube, wire, or line is in a correct position in the chest or abdomen will also require confirmation by a radiologist and the nurse should rely on his or her evaluation.

Alveolar and Capillary Anatomy and Physiology

An understanding of alveolar and capillary anatomy and physiology is essential for interpreting radiographic indications of pulmonary disease of the air spaces and interstitium. Alveoli consist of 2 different types of cells. Most alveoli are lined with type I cells, which are flat, squamous cells. The corners of alveoli contain type II cells, which produce surfactant. Alveolar macrophages are phagocytic cells that clear particles from these air spaces. Openings between alveoli (pores of Kohn) permit movement of gases between adjacent alveoli.[16,17]

Many structures and substances, such as fluid, connective tissues, leukocytes, and macrophages, are located in the walls between alveoli.[16,17] Capillaries are interposed between alveoli in a network. The contact of alveolar surfaces with capillary surfaces forms the alveolar-capillary membrane, the structure through which gas exchange occurs.

The alveolar-capillary membrane has a thin side and a thick side.[16,17] The thin side bulges into the alveolus and is the primary site for gas exchange. The fusion of alveolar and capillary basement membranes creates the thin side. The thick side includes the alveolar and capillary basement membrane separated by the alveolar interstitial space. The alveolar interstitial space is made of connective tissue such as elastic fibers, collagen fibrils, and fibroblasts. The thick side of the alveolar-capillary membrane does not conduct gas exchange as easily as the thin side does; rather, it promotes fluid exchange in the lung.[16,17]

Excess fluid in an alveolus is drained via the alveolar interstitial space into the nearby lymphatic system and/or into the connective tissue fibers.[16,17] The connective tissues of the alveolar interstitial space form the support system for the alveolar and pulmonary capillaries. Excess fluid in the alveolar interstitial space fluid is drained via the connective tissue fibers into the potential peribronchial and perivascular interstitial space.[16,17] The peribronchial and perivascular interstitial spaces surround the bronchioles, bronchi, pulmonary arterioles, and pulmonary arteries interspersed between alveoli in a network. The contact of alveolar surfaces with capillary surfaces forms the alveolar-capillary membrane through which gas exchange occurs.

Radiographic Signs of Pulmonary Disease

Most pulmonary diseases are associated with increased density of the lung fields on chest radiographs.[12] Increased density involves changes in the interstitium, the air spaces, or both.

Interstitial Pulmonary Disease

In interstitial pulmonary disease, the volume of the alveolar interstitium increases with no

Table 1: Support Catheters, Tubes, Wires, and Lines in Chest Radiographs

Support Equipment	Proper Position	Additional Information
ET tube	3 to 5 cm from the carina	ET tube tends to slide into the right main bronchus because of a shallower angle
	Half the distance between the medial ends of the clavicle and carina	ET tube can descend 2 cm during neck flexion
	Carina is over T5 to T7	ET tube can ascend 2 cm during neck extension
TT	TT tip should be halfway between the stoma in which the tube was inserted and the carina	Incorrect positioning of a TT could penetrate the trachea and cause pneumothorax and subcutaneous emphysema
	TT tip should be at about T3	
CVC	Reach the medial end of clavicle before descending	Common malpositioning of a CVC includes placement in the right atrium and internal jugular vein
	If CVC tip descends beyond the indentation that marks the junction of the right atrium and superior vena cava, it is in the right atrium	Pneumothorax occurs in 5% of all CVC insertions[10]
	Descend lateral to spine	Should obtain chest radiograph postinsertion
	CVC tip should be in the superior vena cava and not into the right atrium	
PICC	PICC tip should be within the superior vena cava, which should be above the indentation that marks the junction of the right atrium and superior vena cava	PICC may migrate over time, so monitoring its position is important
	May be difficult to visualize	
Pulmonary artery catheter (Swan Ganz Catheter)	Tip should be in the proximal left or right pulmonary artery	Pulmonary artery catheter tip should not be located in a peripheral or distal artery because of the increased risk of infarction
	Tip should be about 2 cm from the hilum	
Double lumen catheters: "Quinton" hemodialysis catheters	Insertion into right jugular vein most commonly	
	Temporary catheter tips should be in superior vena cava	
	Permanent catheter tips should be in the right atrium	
Pleural drainage tubes: Chest tubes and thoracotomy tubes	For pleural effusion, tubes should be placed posteriorly and inferiorly	If side hole extends outside chest wall, an air leak may occur causing subcutaneous emphysema
	For pneumothorax, tube tips should be placed anteriorly and superiorly	Rapid lung reexpansion can cause reexpansion pulmonary edema
	Ideal position is anterosuperior for evacuating a pneumothorax	
	Ideal position is posteroinferior for evacuating a pleural effusion	

(continues)

Table 1: Support Catheters, Tubes, Wires, and Lines in Chest Radiographs (*Continued*)

Support Equipment	Proper Position	Additional Information
Pacemakers	Implanted subcutaneously in left anterior chest wall	Pneumothoraces occur during insertion
	Tip of one lead is almost always located in the apex of the right ventricle	Fracture of leads may occur
	Two lead pacemakers have 1 lead in the right atrium and 1 in the right ventricle	
	Three lead pacemakers have 1 lead in the right atrium, 1 lead in the right ventricle, and 1 lead in the coronary sinus	
	All leads should have gentle curves	
AICDs	AICDs can be differentiated from pacemakers by the wider and more opaque segment of at least 1 of the electrodes	Visible complications can include lead breakage and dislodgement
	One lead is placed in the superior vena cava or brachiocephalic vein	
	If has another lead, it is placed in the apex of right ventricle	
	Bends in leads should be smooth curves, not sharp kinks	
Intra-aortic counter pulsation balloon pump (IABP)	Placed in descending thoracic aorta	If catheter is too proximal, balloon may occlude great vessels leading to stroke
	Tip should lie distal to origin of the left subclavian artery	If balloon is too distal, device has decreased effectiveness
	Metallic marker may point slightly toward the right in the region of the aortic arch	
	When inflated, the balloon may appear as an air-containing sausage in the thoracic aorta	
NG tube	Tip and all side holes should extend about 10 cm into the stomach beyond the EG junction to prevent aspiration from the administration of the feeding into esophagus	NG tubes are the most commonly malpositioned of all tubes; always check with a radiograph to confirm the location
	The EG junction is usually located at the junction of the left hemidiaphragm and the left side of the thoracic spine (left cardiophrenic angle)	When malpositioned, NG tubes are frequently coiled in the esophagus
		If inserted in the trachea, the NG tube can extend into a bronchus and to the periphery of the lung
Dobbhoff feeding tubes	Tip of feeding tube should be in duodenum so as to reduce the risk of aspiration	Placement in the stomach is common
	Tip is recognizable by a weighted metallic end	Placement in the trachea can occur
		Confirmatory radiograph is needed after placement before beginning feedings

Abbreviations: AICD, automatic implantable cardiac defibrillators; CVC, central venous catheter; EG, esophagogastric; ET tube, endotracheal tube; NG tube, nasogastric tubes; PICC, peripherally inserted central catheter; TT, tracheostomy tube.

change in the alveolar air volume. The alveolar interstitial space increases in size because of fluid, fibrosis, inflammation, or abnormal tissue growth.[12] Chronic diffuse interstitial lung disease is usually caused by pulmonary fibrosis.[12] In chronic interstitial pulmonary disease, the markings are sharp and the branching is irregular.[12] See Figure 26 to view an interstitial pulmonary pattern.

Acute diffuse interstitial lung disease is usually due to cardiogenic or noncardiogenic pulmonary edema such as that associated with acute respiratory distress syndrome (ARDS), viral/mycoplasmal pneumonia, or heart failure.[12] In ARDS, interstitial edema is caused by direct injury of pulmonary endothelial cells by inflammatory mediators. Gas exchange is impaired by thickening of the thin side of the alveolar-capillary membrane, which increases the distance that oxygen and carbon dioxide diffuse between the alveoli and the capillaries. In acute interstitial pulmonary disease, the markings are hazy with normal branching of the vascular bed and bronchi.[12]

In interstitial lung disease with normal aeration of the alveoli, chest radiographs typically show increased vascular markings due to more prominent blood vessels.[11–13] In addition, lines may be visible in the upper or lower lobes because of thickening of fissure lines. Depending on the type of interstitial lung disease, the markings are as follows[11–13]:

- reticular or linear, which appear as small lines or sometimes as a mesh such as in interstitial pulmonary edema and pulmonary fibrosis;
- nodular, which appear as small round dense opacities; examples include bronchogenic cancer and lung metastases; and
- reticulonodular, which are a combination of reticular and nodular markings and is the most common type, which includes sarcoidosis.

Air Space Disease (Alveoli)

Air space or alveolar disease involves reduction of air because of alveolar consolidation, atelectasis, or both. In alveolar consolidation, also termed infiltrates, tissue or fluid replaces all of the air in the alveoli and the lung tissue appears opacified or a water density.[12] An alveolar pattern occurs when many alveoli are filled with fluid or tissue creating a water density or fluffy appearance. However, in an alveolar pattern, not all alveoli in 1 lung area are filled with fluid or tissue, so the infiltrates appear as "patchy" water densities.

Signs of alveolar consolidation on a chest radiograph include the silhouette sign and the air bronchogram sign.[11–13] An air bronchogram sign is the radiographic shadow (a radiolucent area) of an air-filled bronchus running through an airless area of lung, which appears opacified (Figure 27).[11–13] Figure 3 illustrates a silhouette sign with no clear left-sided heart border caused by left upper lobe pneumonia and consolidation.

Decreased lung air volume in alveolar consolidation decreases respiratory gas exchange of oxygen.[16] Common causes of consolidation are bacterial pneumonias, cardiogenic pulmonary edema, and noncardiogenic edema such as ARDS.[11–13] In cardiogenic pulmonary edema, increased hydrostatic pressure in the blood forces fluid out into the interstitium and alveoli, which can cause alveolar consolidation.[10] In ARDS or noncardiogenic edema, direct injury to the alveoli increases permeability of the alveolar-capillary membrane, which over the next 48 hours allows fluid to fill the alveoli and can cause complete lung consolidation.[10,16] This complete consolidation can be visualized as a complete whiteout on chest radiographs for ARDS or cardiogenic pulmonary edema (Figures 30 and 35).[10–13]

Atelectasis is caused by the following 4 pathophysiologic conditions[10,13]:

1. Air is absorbed from the alveoli (resorptive), usually because of lack of ventilation from an obstruction above the alveoli such as a mucus plug, tumor, foreign body, or occluding inflammation or edema.
2. Alveoli are compressed because of increased intrathoracic pressure (relaxation or passive), such as the opposite lung compressed from a tension pneumothorax.
3. Alveoli collapse from fibrosis or scarring, which is termed cicatrization.
4. Alveoli collapse from loss of surfactant (adhesive) like in ARDS.

Hypoventilation can also cause alveoli to lose air volume and become atelectic. Left lower lobe atelectasis is very common following coronary artery bypass surgery.[13] One should expect to view signs of left lower lobe atelectasis on these patients' postoperative

radiographs. In all instances of atelectasis, diminished tissue oxygenation occurs because of lack of oxygen in the alveoli.

Direct signs of atelectasis on a chest radiograph include displaced fissures, crowded bronchovascular markings, and shifted position of a marker structure such as a scar, nodule, or granuloma.[12] Indirect signs of atelectasis are structural shifts in the positions of the hila, diaphragm, and mediastinum and increased density or radiopacity of the lung tissue.[12,13] Figure 23 shows both direct and indirect signs of atelectasis.

When atelectasis is localized, oblique or horizontal lines may appear. These radiograph changes are called discoid, subsegmental, or plate-like atelectasis (Figure 31).[13] Keep in mind that to confirm atelectasis in a lower lobe, a lateral view radiograph may be necessary.[13] Two other airspace deformities that are seen on chest radiographs are blebs and bullae. Blebs and bullae are thin-walled air-filled spaces commonly located in the upper lobes and are radiolucent.[10] These develop because of alveolar destruction associated with emphysema. Blebs are relatively small air cavities of size less than 1 cm.[4] A bulla is greater than 1 cm and often much larger.[4] See Figure 9 for examples of bullae.

Air Space Disease and Noncardiogenic Pulmonary Edema (ARDS)

Patients with ARDS can have a combination of air space disease with both alveolar consolidation and atelectasis.[13] Diffuse and patchy alveolar infiltrates occur beginning in the periphery about 12 hours after the insult to the alveolar-capillary membrane (Figure 28).[10] Chest radiographs show a primary alveolar pattern with additional signs of atelectasis caused by reduced lung volume from loss of surfactant within 24 to 48 hours postinsult (Figure 29).[10,11] Air bronchograms are commonly seen during this stage.[10] Complete alveolar consolidation may also occur (Figure 30).

In addition, most likely both indirect and direct lung injury coexist in ARDS.[10] However, the consolidation pattern of direct injury usually predominates over the interstitial pattern attributable to indirect injury.[13] When a patient survives ARDS, a chronic interstitial pattern due to pulmonary fibrosis often becomes apparent on chest radiographs obtained 1 week or more after the acute stage or diagnosis of ARDS.[10,11] The chest radiograph of a post-ARDS patient may look similar to that of Figure 26.

Radiographic Signs of Cardiac Disease

Radiographic signs of cardiac disease are similar but also differ from those of pulmonary disease. Following are descriptions of radiographic signs of cardiac disease.

Heart Failure and Cardiogenic Pulmonary Edema

In addition to left ventricular enlargement, cephalization or vascular redistribution is an indication of left ventricular failure.[11–13] In a patient standing upright, the pulmonary blood vessels are larger in the lower lobes of the lung than in the upper lobes. If the blood vessels in the upper lobes are larger than the blood vessels in the lower lobes, the condition is termed cephalization or vascular redistribution.[12] Cephalization occurs because of increasing left ventricular pressure. When pulmonary capillary wedge pressure (PCWP) becomes elevated or left atrial pressure is greater between 10 and 15, cephalization occurs.[10,11] In addition, if hydrostatic pressure increases to greater than 10, fluid leaks into the interstitium.[10] See Figures 20 and 32 to view cephalization.

In addition, as PCWP continues to increase to 19 to 20 mm Hg, interstitial edema begins (Figure 33).[10] Kerley lines are a radiographic sign with interstitial pulmonary edema. They are thin linear pulmonary opacities caused by fluid or cellular infiltration into the interstitium of the lungs. There are 3 types of Kerley lines with Kerley B being the most common. Kerley B lines are due to thickening of interlobular septa caused by increased tissue or fluid, as in interstitial pulmonary edema.[10,13] Kerley B lines are seen as horizontal lines no more than 2 cm long that appear in the lung periphery near the costophrenic angles and lateral wall (Figure 33). Kerley A lines are caused by distension of channels that connect the peripheral and central lymphatics of the lungs. Kerley A lines appear as oblique lines approximately 2 to 6 cm long that course diagonally toward the hila from the periphery in the upper lobes.[10,11] One does not see Kerley A lines without Kerley B lines (Figure 33).[10] Kerley C lines supposedly represent thickening of lymphatics that appear as short fine lines throughout the lungs with a reticular appearance. Some radiologists claim that Kerley C lines do not actually exist.[10] It is also suggested that what is thought to be Kerley C lines are a combination of Kerley A and Kerley B lines.[10]

Peribronchial cuffing may also be seen in interstitial pulmonary edema. Normal bronchial walls appear as pencil-point thin when viewed end-on.[13] Interstitial edema widens the bronchial walls and makes their margins appear indistinct, which is termed peribronchial cuffing.[13] Some radiologists describe peribronchial cuffing as appearing as small doughnut densities on the radiograph.[10] See Figures 12, 14, 30, 33, and 34 for examples of peribronchial cuffing.

In addition, a butterfly or batwing pattern may become apparent on chest radiographs during interstitial pulmonary edema due to heart failure (Figure 34).[10,11] The butterfly pattern is due to the fluid engorgement of the interstitium adjacent to the hilar blood vessels.[13] Lower lung zones are usually much more affected than upper lung zones.[10]

Once edema due to heart failure involves the entire alveolar interstitium as a result of increasing PCWP and increased left ventricular pressure, the alveoli start to become edematous.[13] At this point, the PCWP is usually greater than 25 mm Hg.[10,11] At this time, an alveolar consolidation pattern becomes evident on chest radiographs (Figure 35).[10,11] When resolution of this alveolar edema begins, it clears from the periphery and moves centrally.[10] However, not all patients with high PCWP have evidence of heart failure on chest radiographs. Pleural effusions are also common in moderate to severe heart failure as indicated by a shallow costophrenic sulcus. The 3 classic stages of cardiogenic pulmonary edema are cephalization (Figure 32), interstitial edema (Figures 33 and 34), and alveolar edema (Figure 35).

However, radiographic findings often differ between acute heart failure and chronic heart failure.[13] For example, interstitial edema and Kerley B lines do not seem to occur until venous pressures are higher in chronic heart failure than in acute heart failure.[13] Radiographs of chronic heart failure are more likely to show cephalization (vascular redistribution) than interstitial edema, whereas in acute heart failure, one is more likely to view interstitial edema.[13]

Radiographic Sign Differences of Cardiogenic and Noncardiogenic Pulmonary Edema

Radiographic signs differ between cardiogenic and noncardiogenic pulmonary edema. Kerley B lines and peribronchial cuffing are seen in radiographs of cardiogenic pulmonary edema but not in ARDS radiographs.[10] The distribution of pulmonary edema tends to be even in cardiogenic pulmonary edema but is distributed more to the periphery in ARDS.[10] Air bronchograms are seen commonly in ARDS radiographs, whereas they are not commonly seen in cardiogenic pulmonary edema radiographs.[10] Pleural effusions are much more likely to be seen on cardiogenic pulmonary edema radiographs than on ARDS radiographs.[10]

Implications for Nurses

Signs and symptoms that patients have during acute illness may be indicative of many different types of pathophysiologic or disease processes. Critical care nurses can use chest radiographs as an additional bedside assessment tool to assist in determining pathophysiologic abnormalities and to confirm other assessment findings. By learning some basic skills in interpreting chest radiographs, nurses can recognize and localize gross pathological changes visible on a chest radiograph.

Nurses are the care providers who are consistently present at the bedside and have up-to-date information on patients' clinical status. For example, changes in densities in the lung fields on a patient's chest radiograph along with auscultation of crackles, pulse oximeter oxygen saturation of 88%, and a PCWP pressure of 25 mm Hg may prompt changes in fluid management and supplemental oxygen for the patient. Using a stethoscope, oxygenation status, hemodynamic information, and chest radiographs together for cardiopulmonary assessments enables nurses to recognize additional important clues concerning a patient's current clinical status. Nurses analyze these findings and develop a plan of care for their patients. For example, a nurse may have determined that a primary goal is to improve lung function and oxygenation. Thus, their plan of care may include interventions related to therapeutic body position such as head-of-bed elevation, good lung down, use of lateral rotation or kinetic therapy, and determination of an optimal body position for chest physiotherapy. By incorporating the chest radiograph as an additional bedside assessment tool, critical care nurses and advanced practice nurses can more completely monitor patients' clinical status and be able to plan and prioritize nursing interventions.

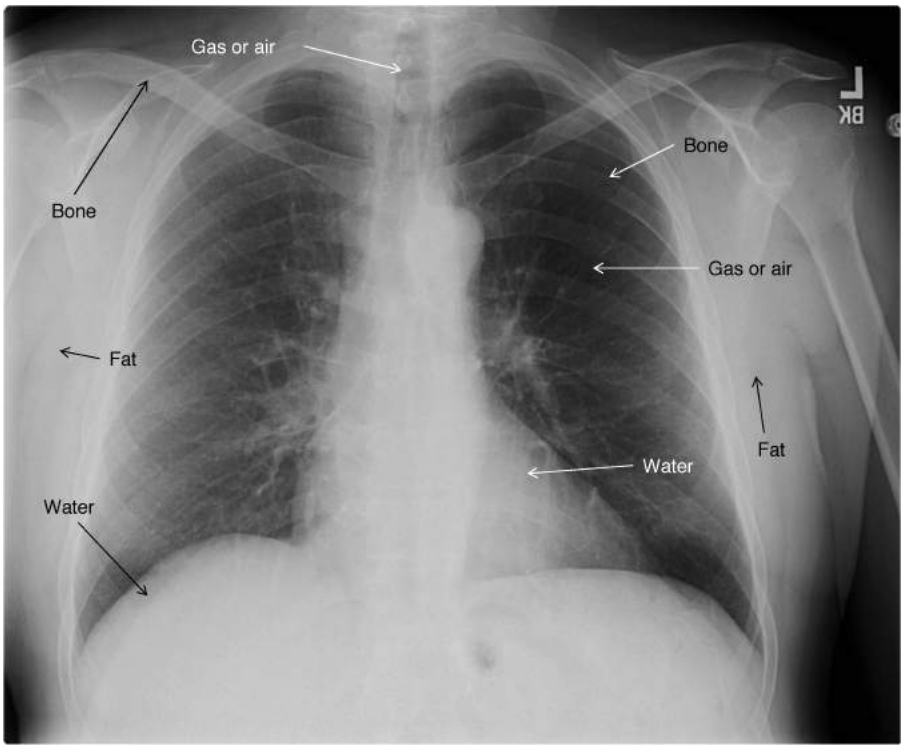

Figure 1: Chest radiodensities.

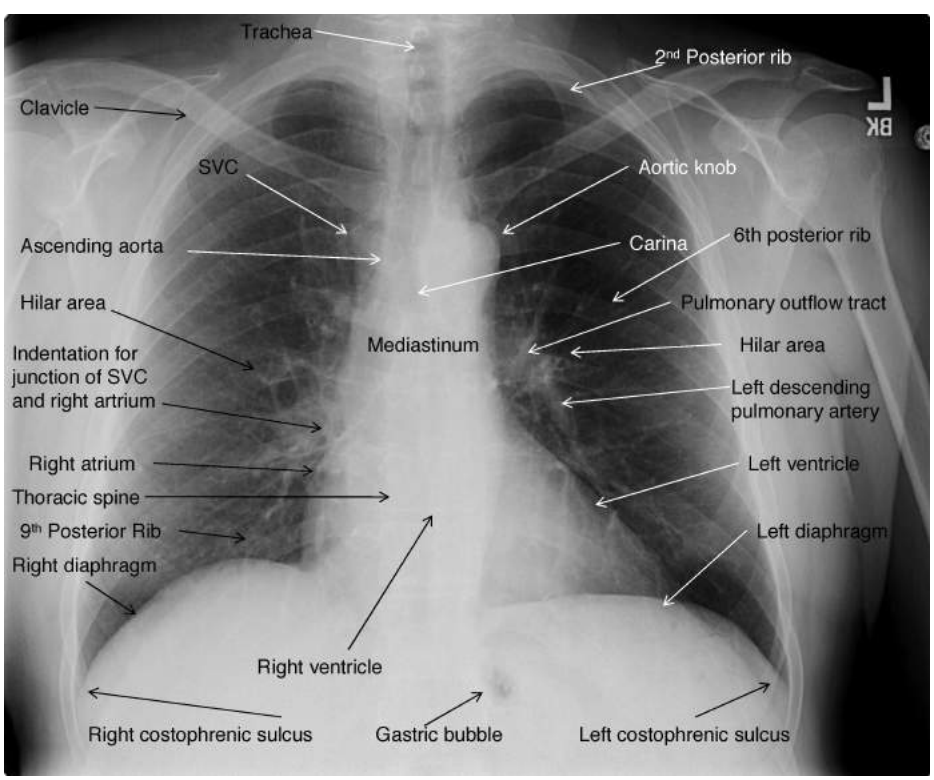

Figure 2: Chest structures, frontal view.

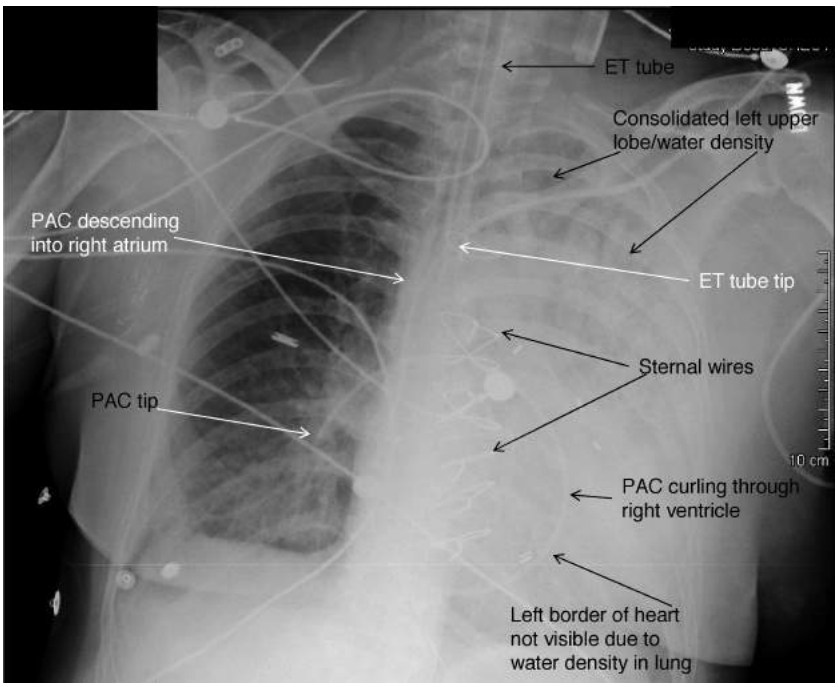

Figure 3: Silhouette sign, left upper lobe pneumonia, PAC, ETT, and sternal wires. Abbreviations: ETT, endotracheal tube; PAC, pulmonary artery catheter.

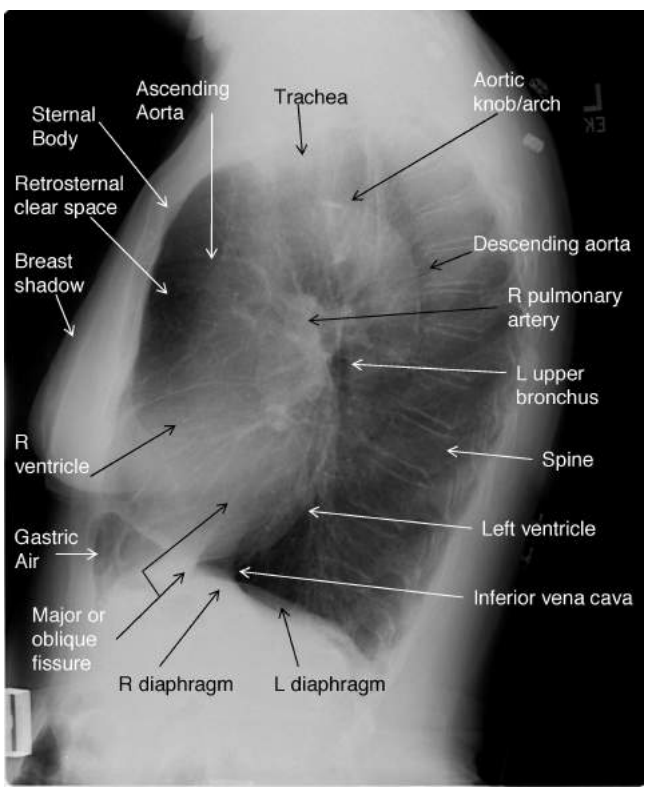

Figure 4: Chest structures left lateral view.

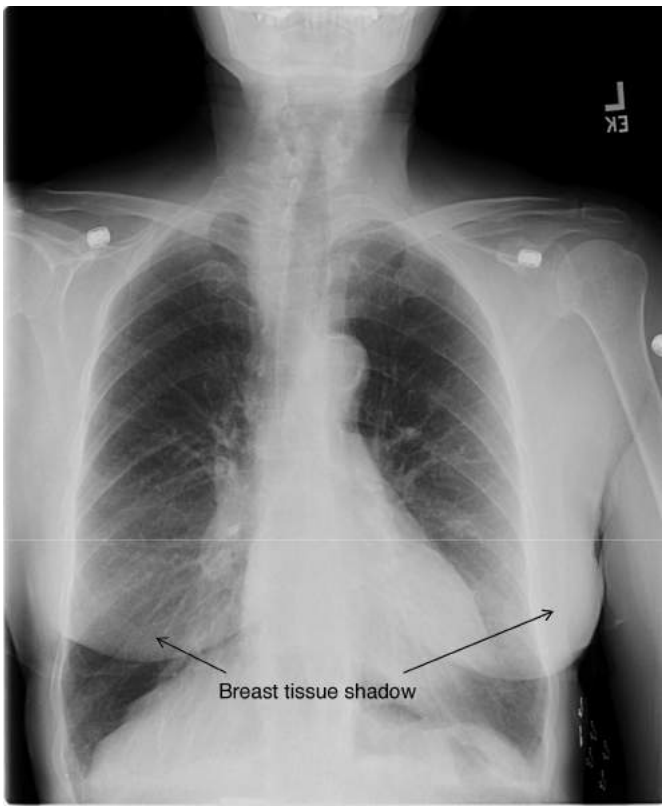

Figure 5: Breast tissue.

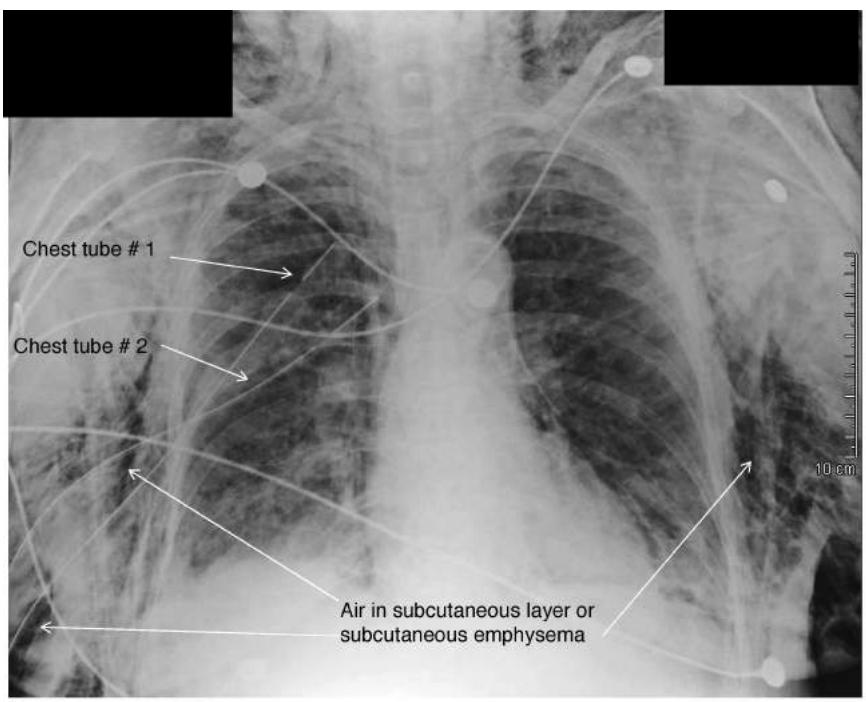

Figure 6: Subcutaneous emphysema and 2 chest tubes.

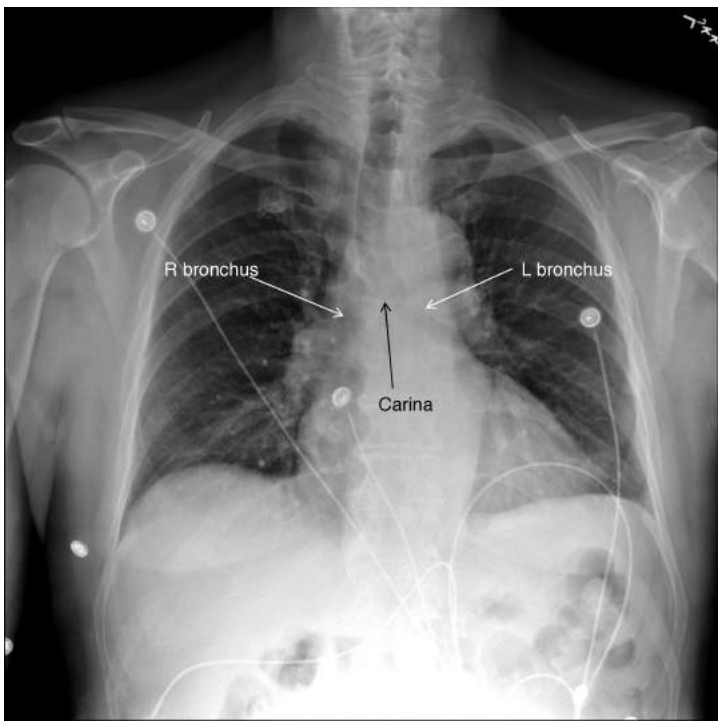

Figure 7: Carina and left and right bronchus.

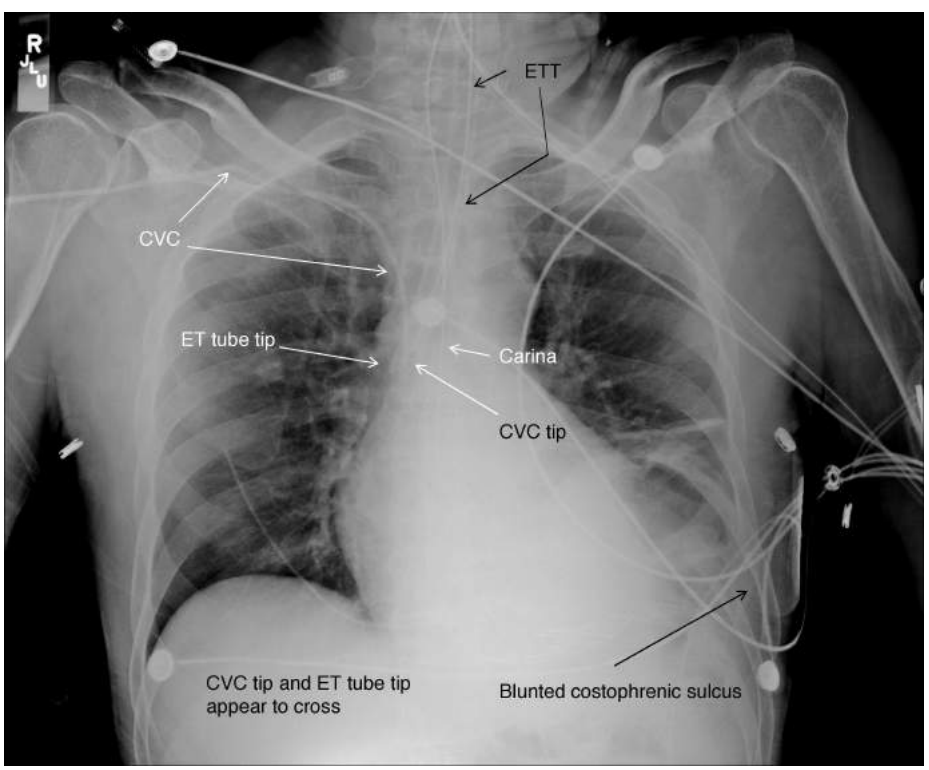

Figure 8: Endotracheal tube in right main stem bronchus, CVC, left pleural effusion.
Abbreviations: ETT, endotracheal tube; CVC, central venous catheter.

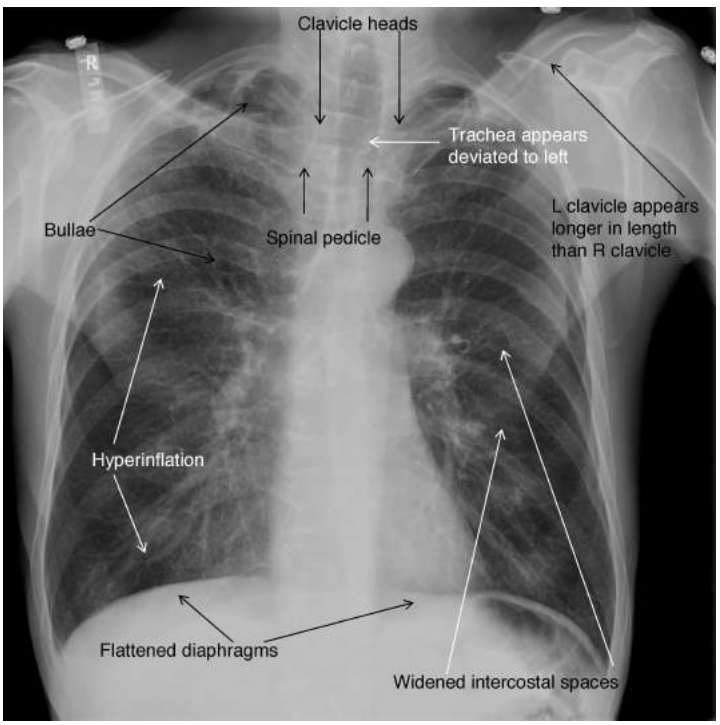

Figure 9: Chronic obstructive pulmonary disease, flattened diaphragms, hyperinflation, widened intercostal spaces, apical bullae, and chest rotation.

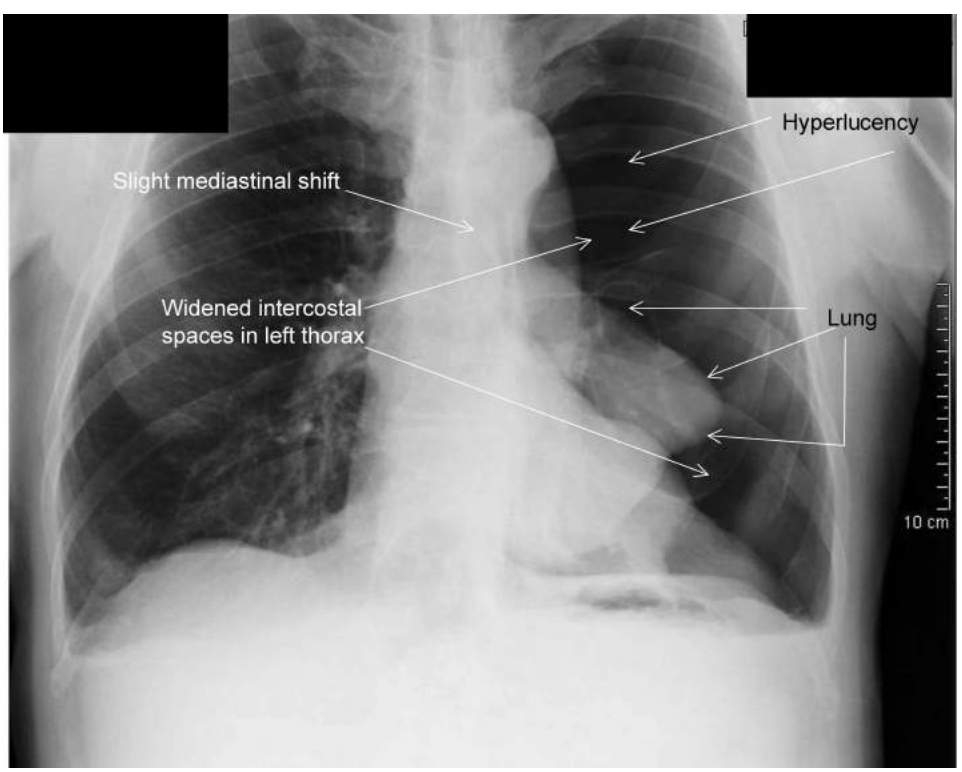

Figure 10: Left pneumothorax, hyperlucency, and widened intercostal spaces.

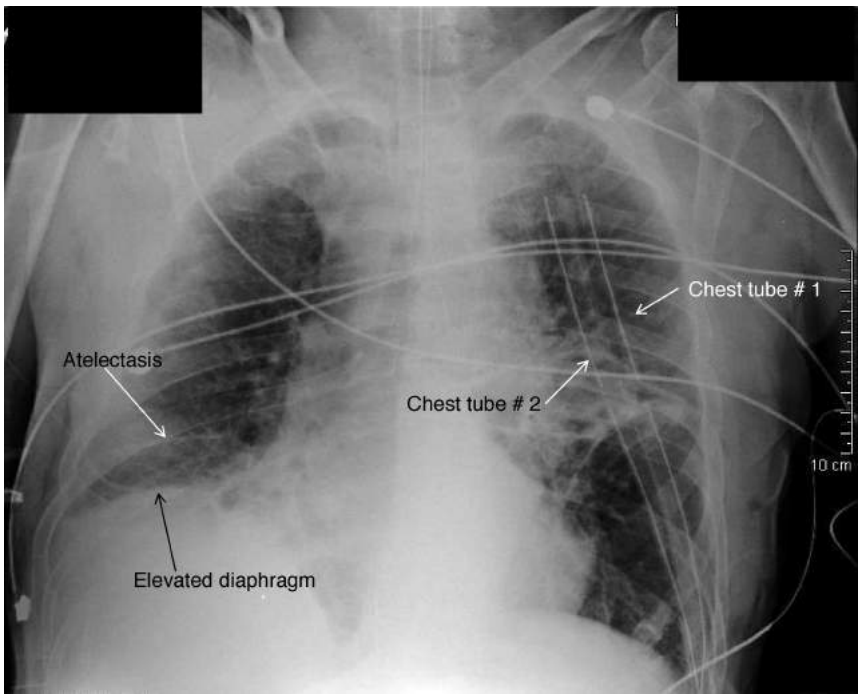

Figure 11: Chest tubes in left thorax with reinflated left lung and right lower lobe atelectasis.

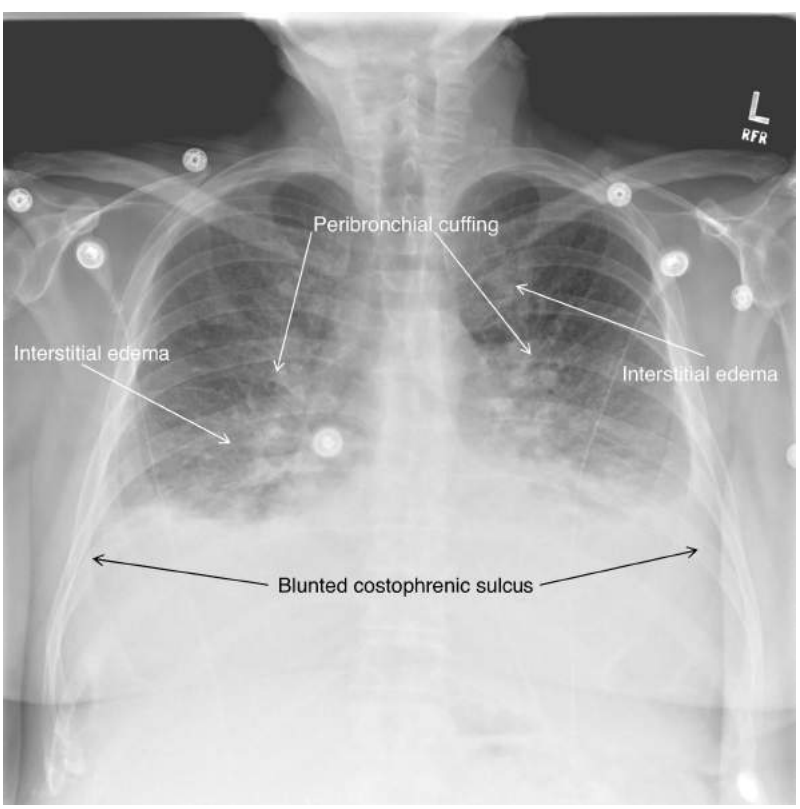

Figure 12: Bilateral pleural effusions with peribronchial cuffing and interstitial edema.

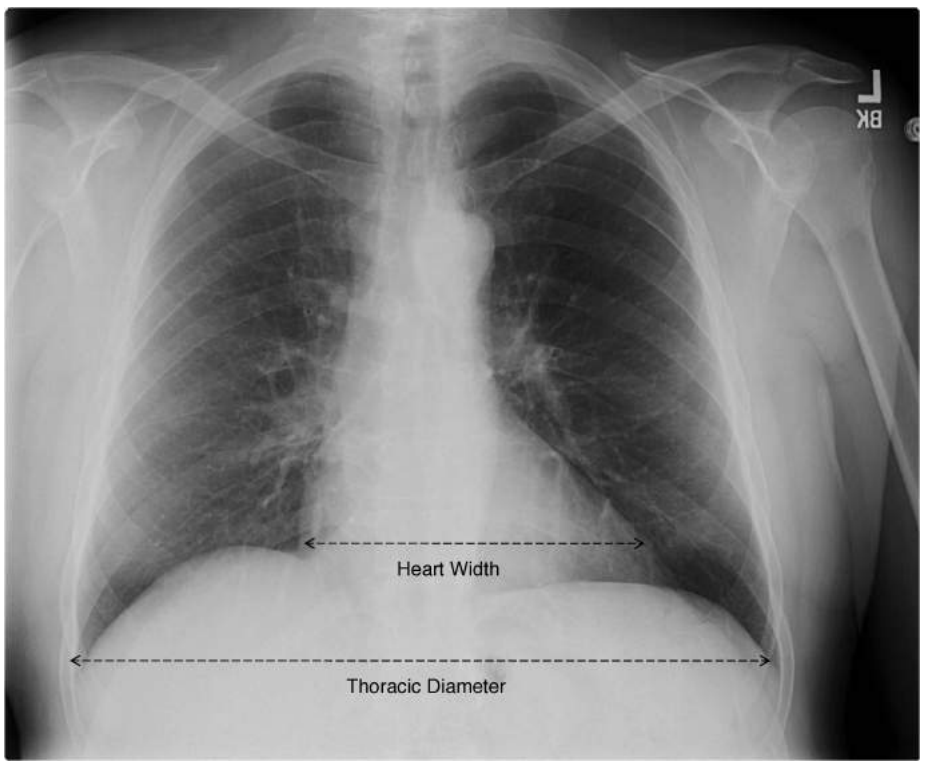

Figure 13: Measurements for cardiothoracic ratio.

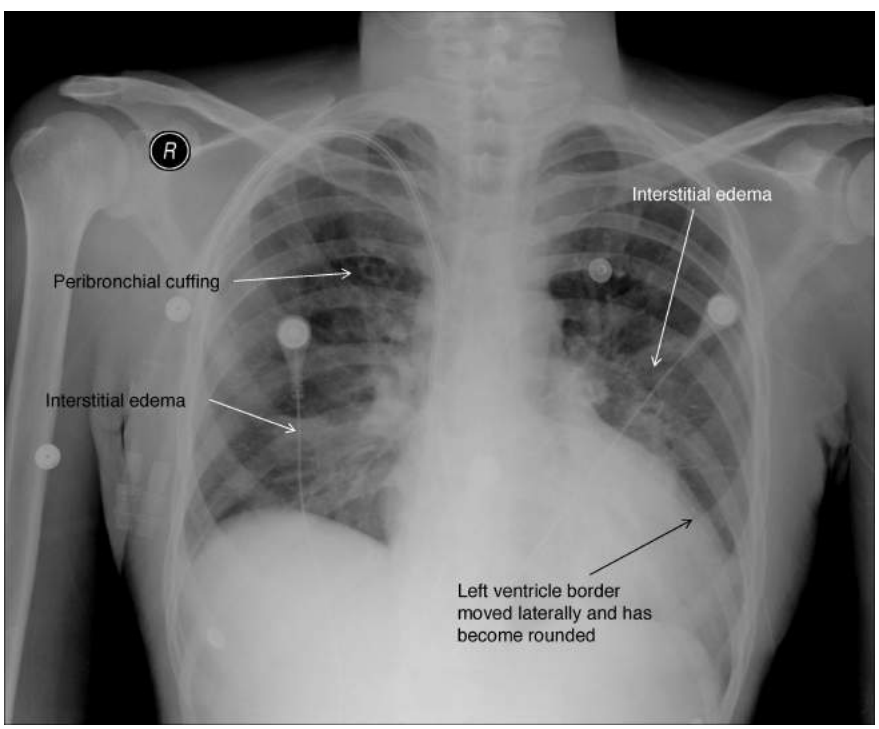

Figure 14: Left ventricle enlargement, interstitial edema, beginning butterfly pattern of cardiogenic pulmonary edema, and peribronchial cuffing.

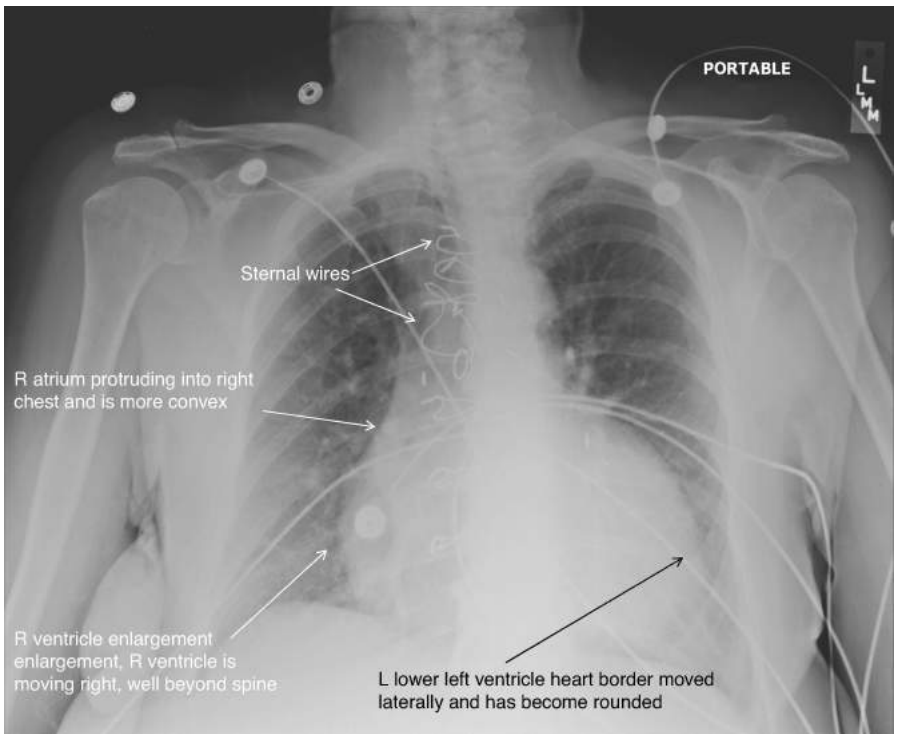

Figure 15: Left and right ventricle enlargement.

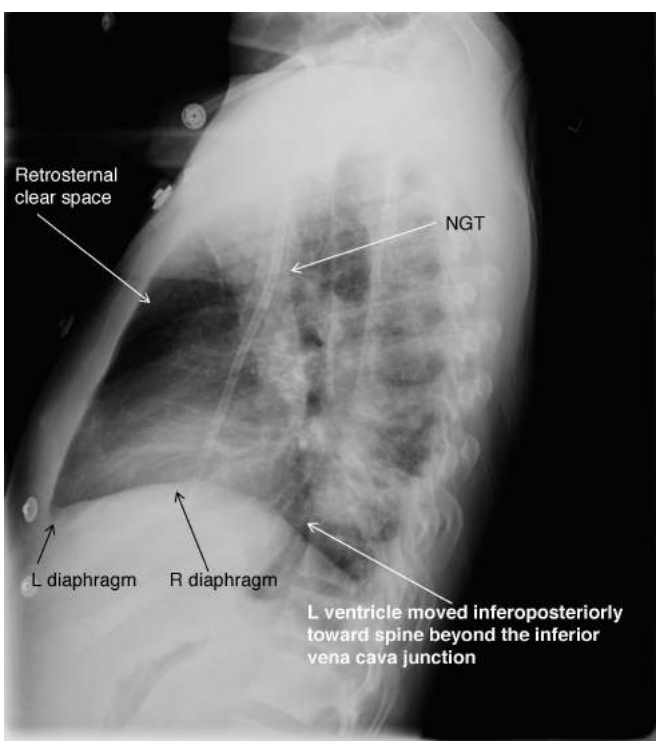

Figure 16: Left ventricle enlargement and NGT on left lateral view.
Abbreviation: NGT, nasogastric tube.

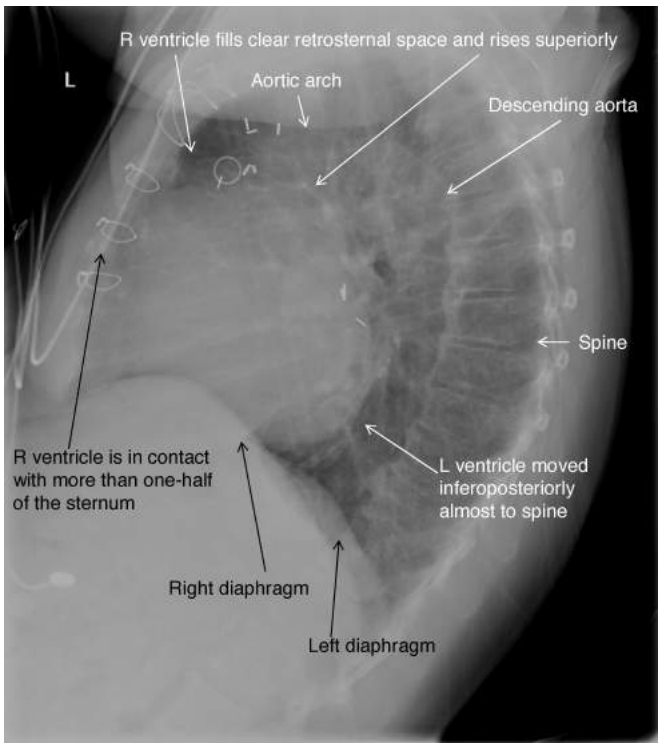

Figure 17: Left and right ventricle enlargement and sterna wires on left lateral view.

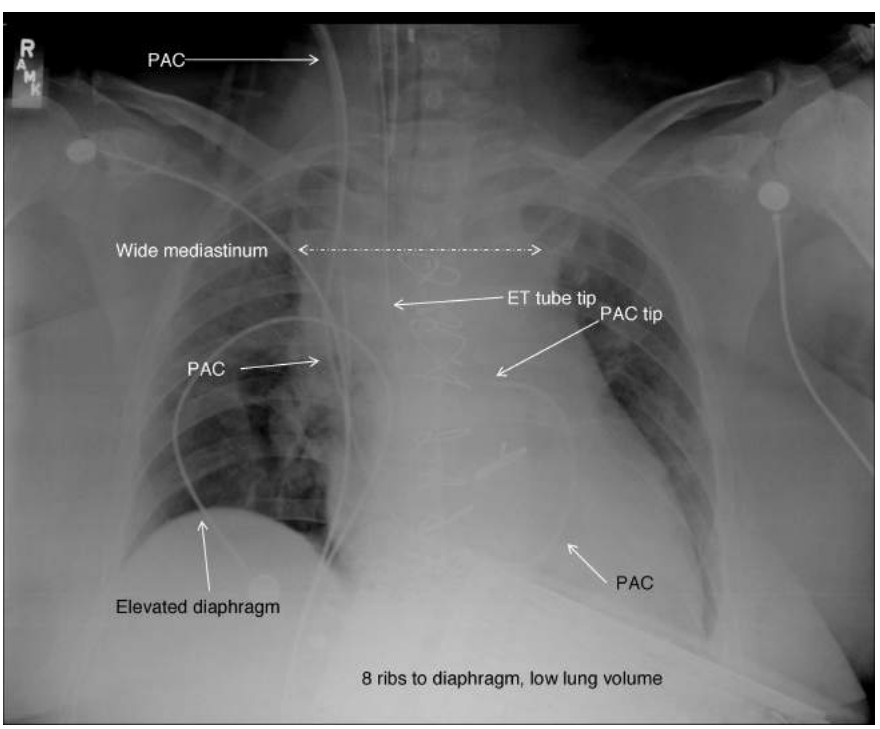

Figure 18: Diffuse generalized mediastinal widening in a postoperative cardiac surgery patient, PAC, ETT, and right atelectasis.
Abbreviations: ETT, endotracheal tube; PAC, pulmonary artery catheter.

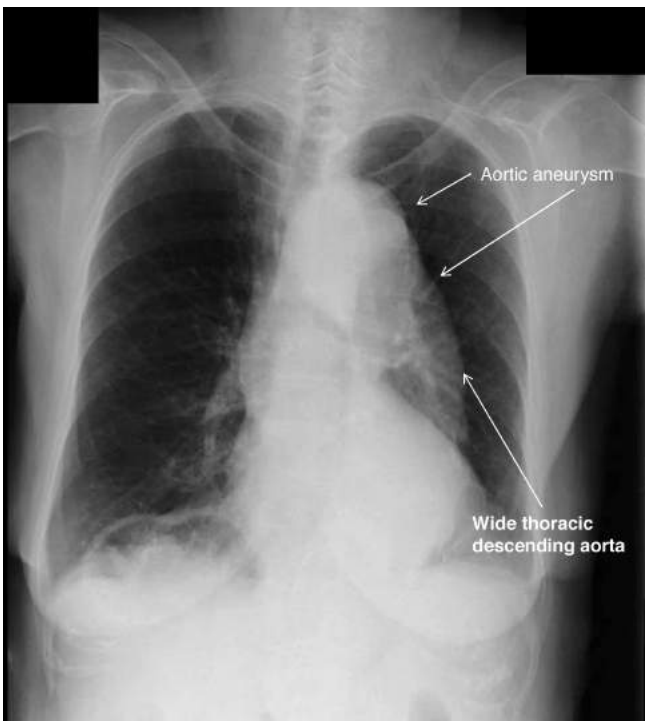

Figure 19: Widened descending aorta indicating thoracic aortic aneurysm.

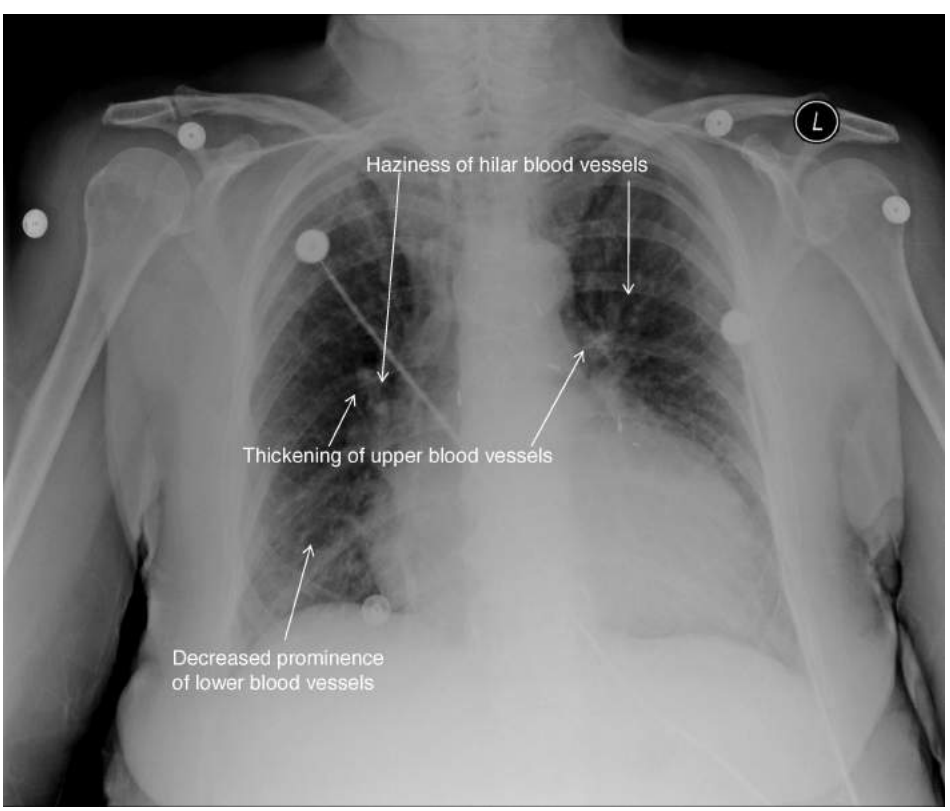

Figure 20: Pulmonary venous hypertension (cephalization) with biventricular enlargement.

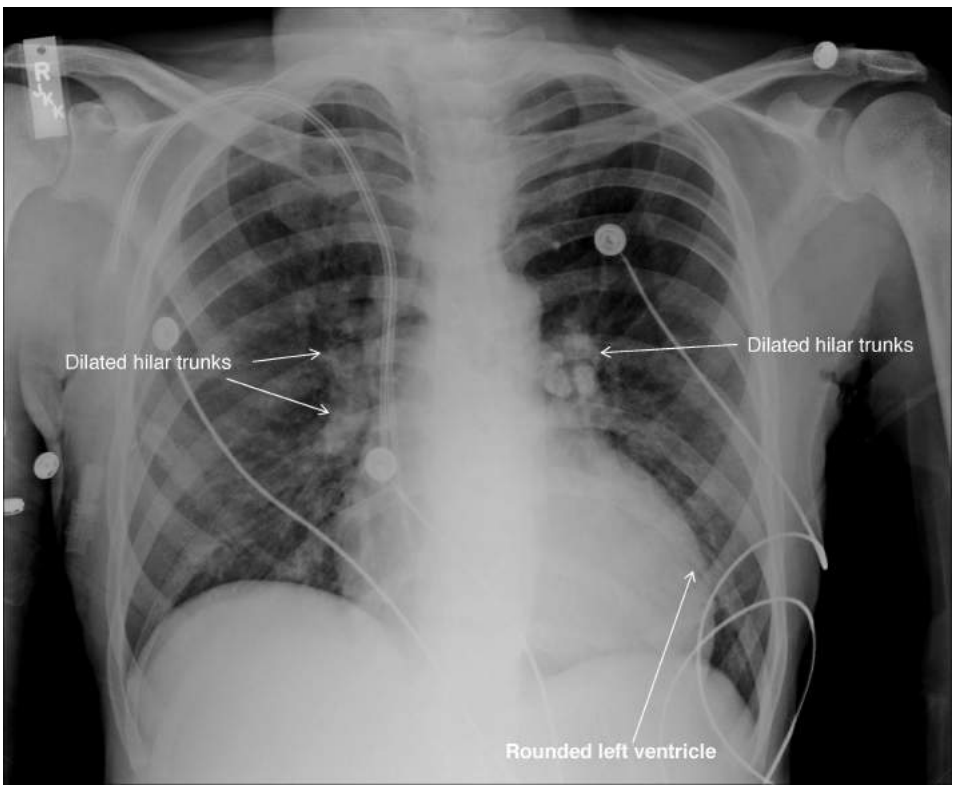

Figure 21: Pulmonary artery hypertension, dilated hilar trunks, and probable left ventricle enlargement.

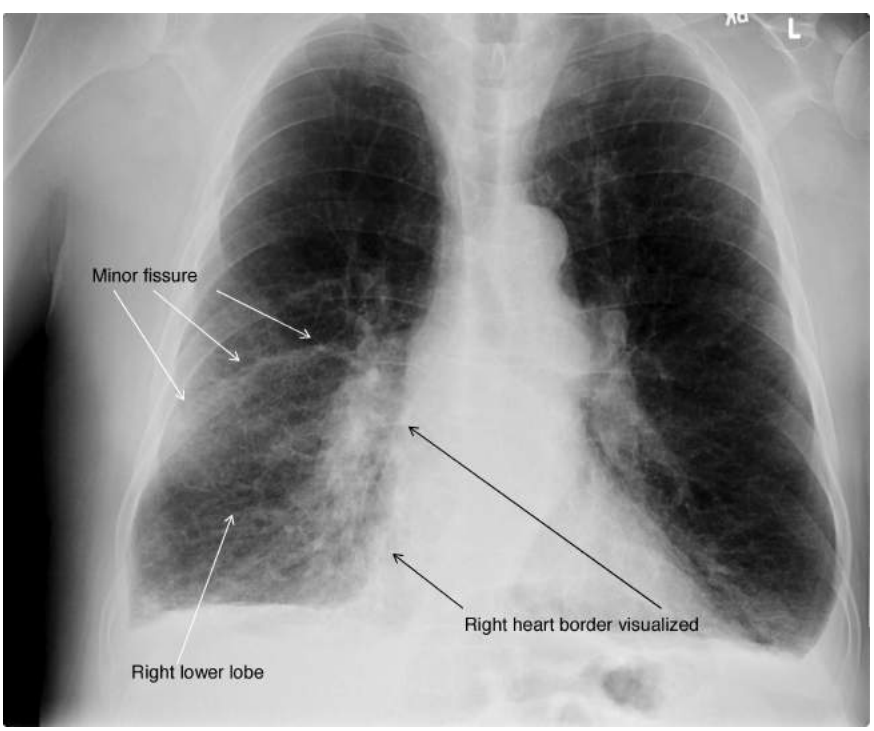

Figure 22: Right lower lobe pneumonia with minor fissure visualized.

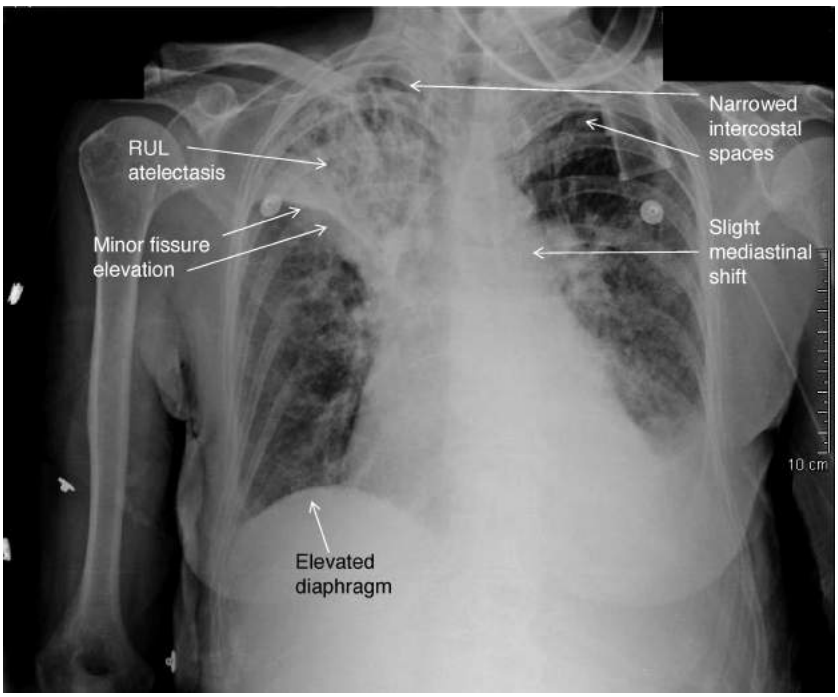

Figure 23: Right upper lobe atelectasis with elevation of right diaphragm and narrowed intercostals spaces.
Abbreviation: RUL, right upper lobe.

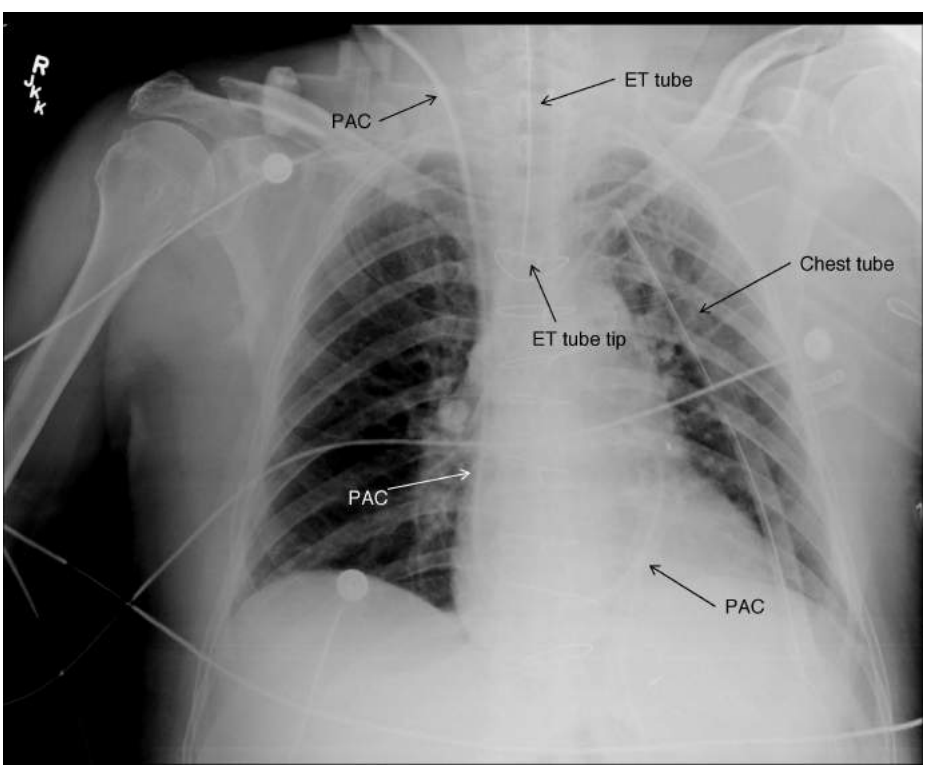

Figure 24: Pulmonary artery catheter, ETT, and left chest tube.
Abbreviations: ETT, endotracheal tube; PAC, pulmonary artery catheter.

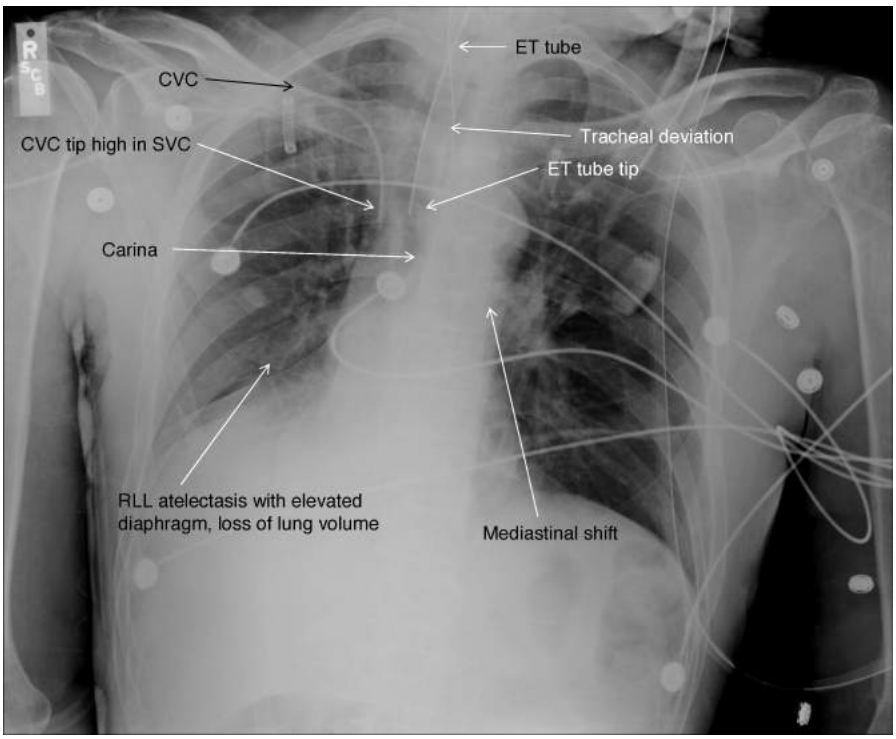

Figure 25: Central venous catheter, ETT, NGT, atelectasis in RLL, and mediastinal shift. Abbreviations: CVC, central venous catheter; ETT, endotracheal tube; NGT, nasogastric tube; RLL, right lower lobe.

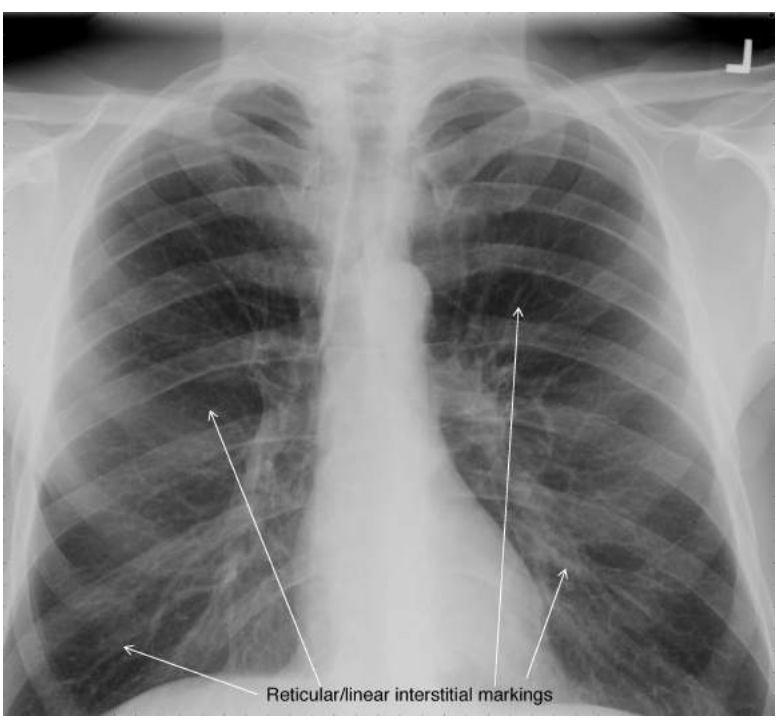

Figure 26: Interstitial pulmonary edema.

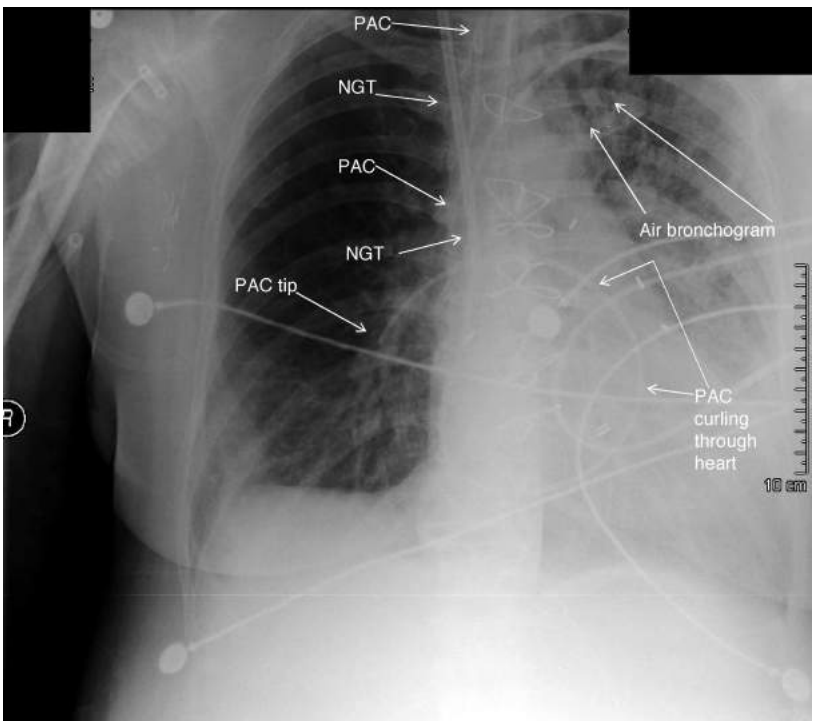

Figure 27: Air bronchogram sign, NGT, sternal wires, and jugular PAC. Abbreviations: NGT, nasogastric tube; PAC, pulmonary artery catheter.

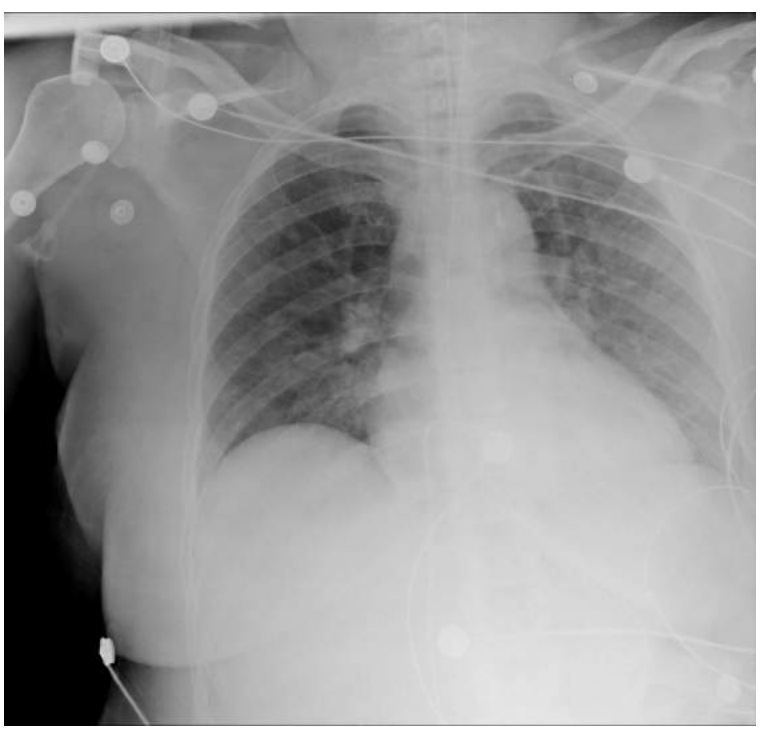

Figure 28: Beginning acute respiratory distress syndrome, soon after hospital admission, patchy infiltrates, alveolar filling in left lower lobe, bilateral atelectasis, and elevated diaphragms.

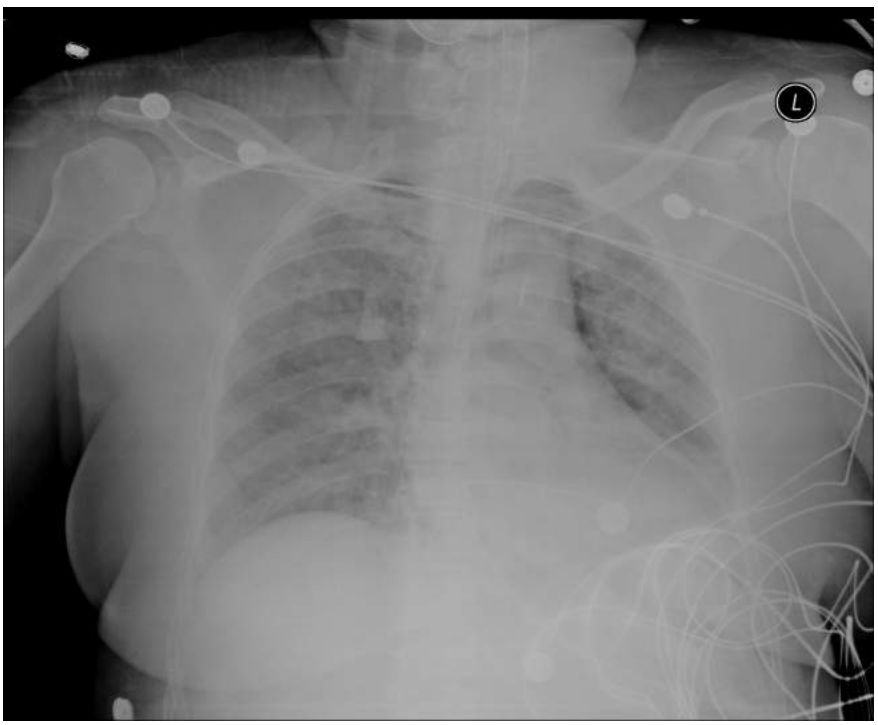

Figure 29: Acute respiratory distress syndrome progressing with increased alveolar filling and atelectasis, 10 hours after the radiograph in Figure 28.

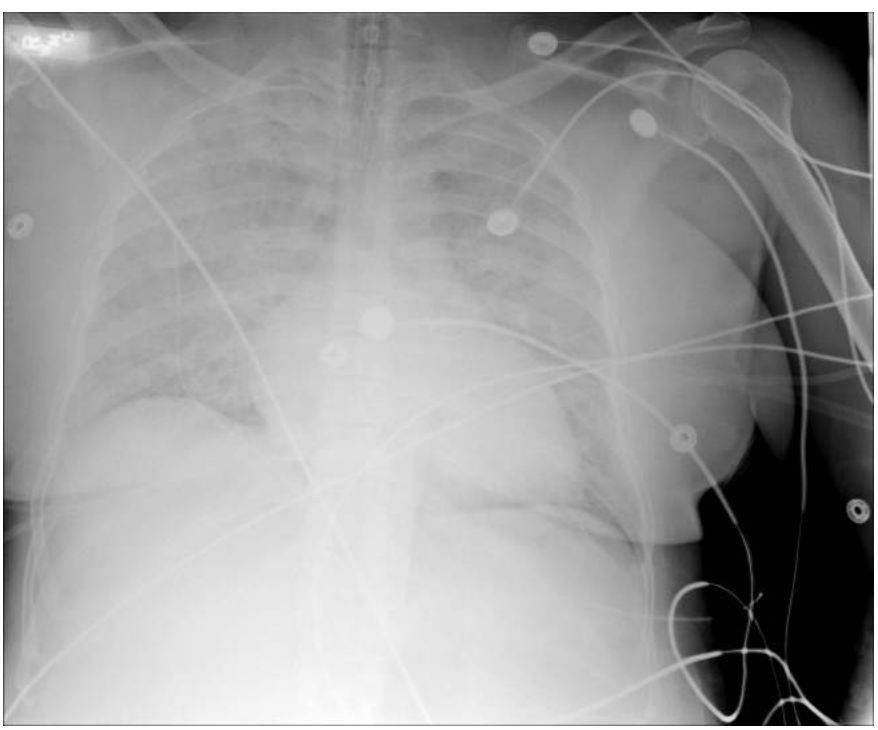

Figure 30: Acute respiratory distress syndrome with whiteout to almost complete alveolar consolidation, noncardiogenic pulmonary edema, atelectasis, 20 hours after the radiograph in Figure 29.

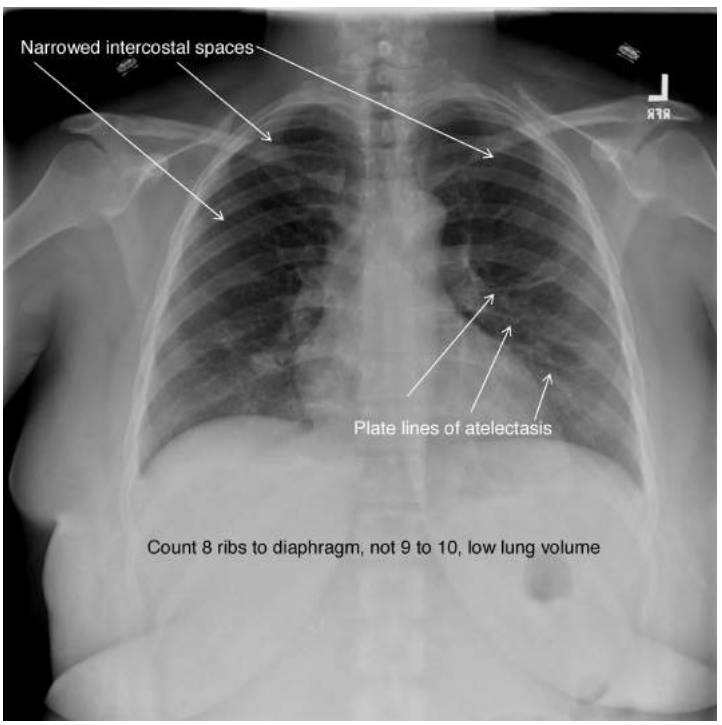

Figure 31: Plate-like atelectasis with diaphragm elevation with narrowed intercostal spaces.

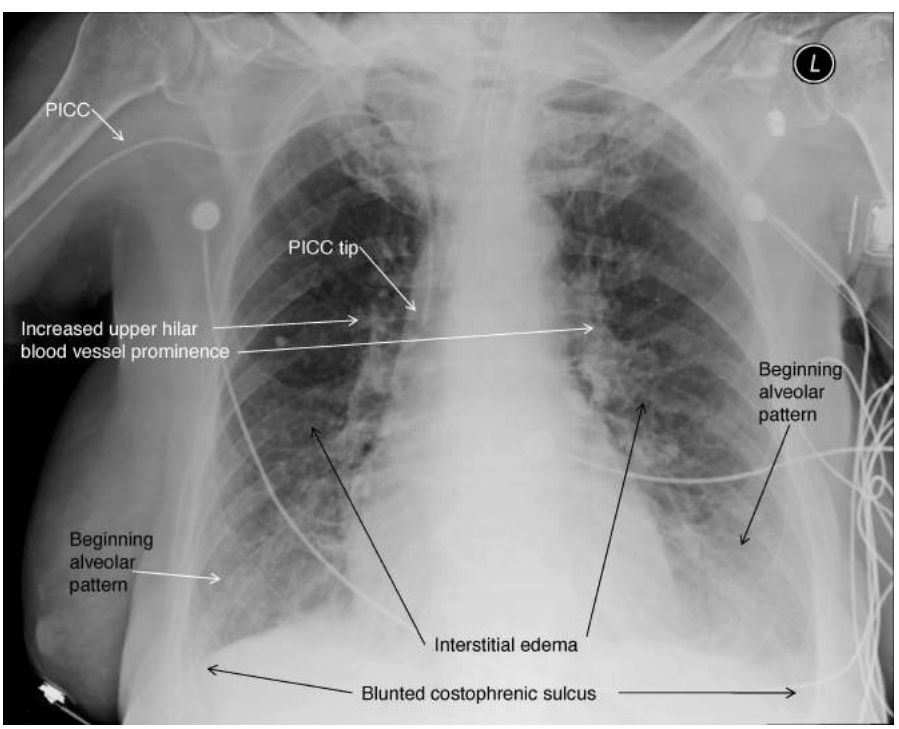

Figure 32: Cephalization, interstitial edema of cardiogenic pulmonary edema, beginning pleural effusions, and PICC.
Abbreviation: PICC, peripheral inserted central catheter.

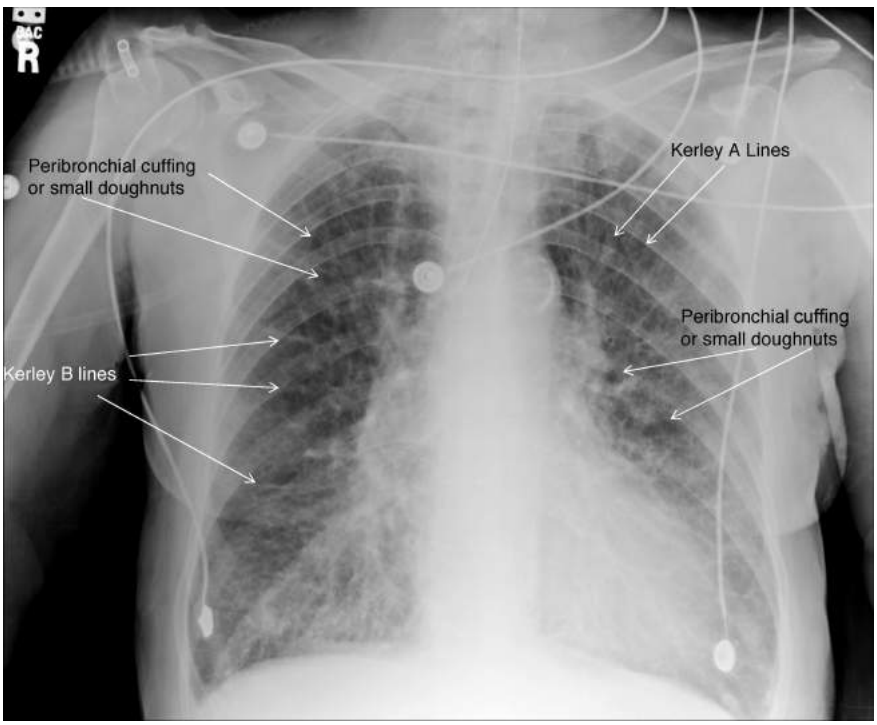

Figure 33: Kerley A and B lines, peribronchial cuffing, and interstitial pattern of cardiogenic pulmonary edema.

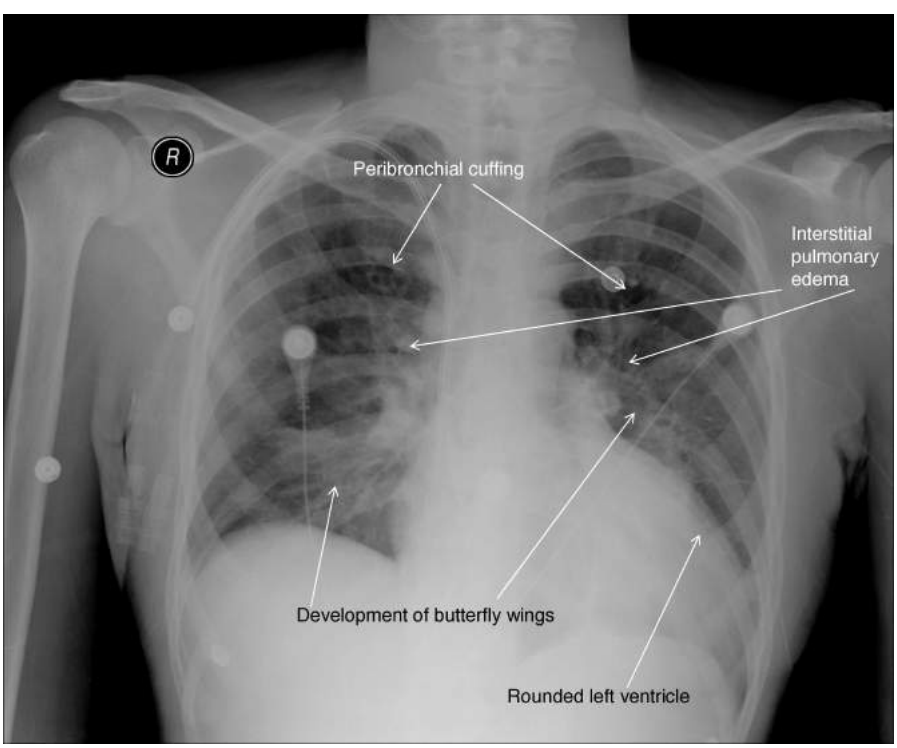

Figure 34: Beginning butterfly pattern of interstitial edema and peribronchial cuffing in cardiogenic pulmonary edema.

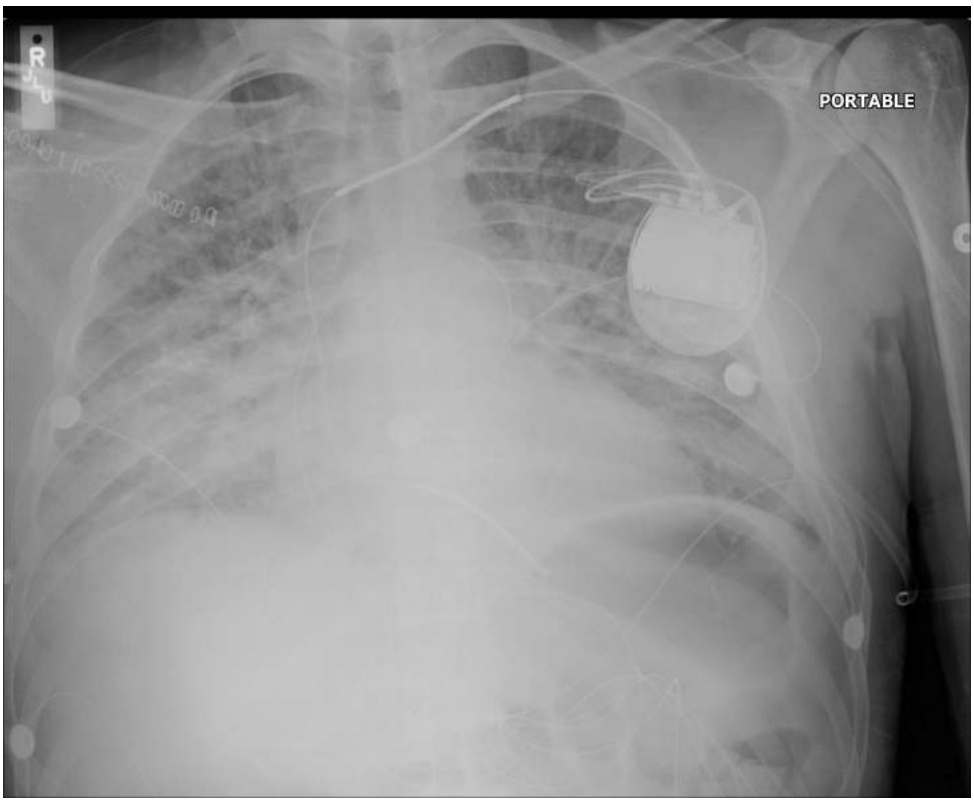

Figure 35: Alveolar edema in cardiogenic pulmonary edema; note the increased edema in bases versus upper lobes, left ventricle enlargement, and automatic implantable cardiac defibrillator.

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