Acute Respiratory Distress Syndrome

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After completing this article, the reader should be able to:

- Describe the pathophysiological changes that occur in acute respiratory distress syndrome (ARDS).
- List some causes of ARDS, distinguishing between direct and indirect causes.
- Identify risk factors for ARDS.
- Discuss signs and symptoms associated with the syndrome.
- Summarize diagnostic criteria and differential diagnoses for ARDS.
- Explain the advantages and disadvantages of chest radiography, computed tomography, and ultrasound imaging in ARDS.
- Discuss treatment strategies for ARDS.
- Name common complications in patients who have ARDS.
- Describe continuing health concerns for ARDS survivors.

Acutely, respiratory distress syndrome (ARDS) is a life-threatening condition with multiple causes and a high mortality rate. Approximately 150,000 cases are reported in the United States annually, making ARDS a public health concern. Management of the condition is complex because of its severity, and medical imaging is essential for both the diagnosis and management of ARDS.

This article introduces common signs, symptoms, risk factors, and causes of ARDS. Diagnostic criteria, histopathology, treatment strategies, and prognostic information are also discussed. The article explains the value of medical imaging studies of ARDS, especially radiography, computed tomography, and ultrasonography.

Acute respiratory distress syndrome (ARDS) is a life-threatening condition and a leading cause of mortality in hospital intensive care units (ICUs). Approximately 150,000 ARDS cases are diagnosed in the United States each year.

Among patients admitted to an ICU, 7.1% develop ARDS. The syndrome is characterized by the rapid onset of severe dyspnea and hypoxemia and can be caused by a variety of illnesses and traumatic injuries. ARDS develops as a result of an inflammatory process that occurs at the alveolar-capillary interface in the lungs, the space in which the blood in the capillaries is separated from the gas present in the alveoli. This produces pulmonary infiltrates that lead to acute respiratory failure and in some cases death.

It is beneficial for radiologic technologists to understand ARDS because medical imaging plays an important role in its diagnosis and management. Abnormalities visible on chest radiographs and computed tomography (CT) scans reflect the histopathological changes that occur in ARDS.

According to the recently adopted Berlin definition of ARDS, bilateral opacities consistent with pulmonary edema must be present on a chest radiograph, but detection on CT also can fulfill the requirement for diagnosing the syndrome. Both chest radiography and chest CT are useful for diagnosis and for determining a patient’s prognosis and progress, as well as detecting complications. Although not standard practice, recent research has determined that CT also can be helpful for directing ventilation. Other imaging modalities can play a role in ARDS as well, particularly
ultrasonography. Chest ultrasonography is an easy method of evaluating for pleural fluid or pneumothorax without using ionizing radiation. Medical imaging also can be a part of follow-up care and research; therefore, radiologic technologists should be aware of the various problems that can occur in patients who survive ARDS.

Pathophysiology

Normal, healthy lungs are designed to efficiently carry out the body’s pulmonary physiologic functions. The main function of the respiratory system is to oxygenate blood and eliminate carbon dioxide. The lungs’ normal physiologic functions include:

- Delivering inhaled oxygen to lung alveoli.
- Diffusing gases (oxygen and carbon dioxide) between the alveolar capillary membrane and alveolar lumen.
- Optimizing gas exchange by matching alveolar ventilation with pulmonary capillary blood flow.
- Maintaining a continuous flow of fluid through the lung alveoli, alveolar ducts, and bronchioles without inducing lung edema or alveolar consolidation.

ARDS is the consequence of lung injury and subsequent inflammation. Pathologically, it is characterized by widespread alveolar damage. It develops from injury to the alveoli, the primary site of gas exchange (see Figure 1). Direct pulmonary or indirect systemic inflammation leads to the activation of proinflammatory processes that create substances that damage and disrupt the barriers between capillaries and air spaces. ARDS occurs as a consequence of this inflammatory process at the alveolar-capillary interface in the lung. Fluid, protein, and cellular debris flood the air spaces and interstitium, disrupting pulmonary surfactant. Surfactant is a substance that lines the alveoli and reduces surface tension, allowing for easier expansion and stretching, which is known as pulmonary compliance.

Disruption of surfactant adds to surface tension, increasing the work required to breathe. It also makes alveolar overexpansion and collapse of the alveolar sacs more likely, which is referred to as atelectasis. Surfactant dysfunction can encourage the development of ARDS because of the resultant alveolar instability. In ARDS, the quantity and quality of surfactant are altered. Alveolar collapse, ventilation-perfusion mismatch, shunting, and pulmonary hypertension consequently take place. Airspace collapse more commonly occurs in dependent lung zones (ie, the lowest part of the lung in relation to gravity). Box 1 explains how ARDS occurs step by step.

The typical histopathological features of ARDS are known as diffuse alveolar damage. There are 3 stages or phases of ARDS, which pathophysiologically mirror the radiographic changes that can be seen (see Figure 2). Various terms are used to describe the phases of ARDS, and some authors cite 4 stages rather than 3. In addition, the number of days associated with each phase varies according to different sources (see Table). However, the literature is consistent regarding the typical pathologic and radiographic patterns that evolve.

Epidemiology

Although there are more than 50 recognized lung injuries associated with the development of ARDS, some are more likely to progress to ARDS than others. Most cases occur as a result of a small number of causes (see Box 2). Sepsis, bacterial pneumonia, multiple trauma, and aspiration pneumonia account for more than 70% of adult cases. Patients who have sepsis (infection in the blood) are at very high risk; approximately 35% of patients with sepsis develop ARDS. Sepsis is
### How Acute Respiratory Distress Syndrome (ARDS) Occurs

- The tissues lining the alveoli and the pulmonary capillaries are injured, either directly or indirectly.
- The injured tissues release molecules that cause inflammation. White blood cells collect at the site, and swelling occurs. The tissues become more permeable to fluid and proteins. The hydrostatic pressure gradient between the alveoli and capillaries is reversed.
- Proteins and fluid move from the capillaries into alveoli, causing impaired gas exchange in the affected alveoli. The alveoli then collapse (a condition known as atelectasis), and gas exchange becomes impossible.
- The fluid that accumulates in the interstitial spaces, alveolar spaces, and small airways causes the lungs to stiffen, preventing air from moving into the lungs.
- As alveoli fill with fluid or collapse, the capillaries surrounding the alveoli can no longer absorb oxygen. The body responds by shunting blood away from these alveoli.
- As fluid builds up in the alveoli, the patient develops low blood oxygen (hypoxemia) and increasing respiratory distress. Pulmonary edema progresses and inflammation leads to fibrosis, further disrupting gas exchange.
- Tachypnea from respiratory distress causes carbon dioxide levels to decrease, resulting in alkalosis (abnormal blood pH). The body attempts to compensate through metabolic acidosis. Unless gas exchange is restored and the process reversed, acidosis will worsen until all organ systems fail.

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**Figure 2.** The progression of ARDS displayed on chest radiographs, beginning with initial hospital admission to the emergency department. The patient developed ARDS that was triggered by H1N1 pneumonia infection. A. This initial chest radiograph performed at the time of the patient’s admission demonstrates bilateral lower lobe consolidation. B. A second chest radiograph obtained 21 hours later demonstrates greater consolidation of bilateral basilar infiltrates. C. This chest radiograph taken 72 hours after hospital admission demonstrates “white out,” bilateral air space opacification. Bilateral lung infiltrates and pleural effusions were noted. Images courtesy of Community Health Network, Community Hospital South, Indianapolis, IN.

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the most common cause of indirect lung injury. Sepsis-related ARDS is typically severe and, compared with nonsepsis-related ARDS, has a higher risk of mortality. The most common cause of direct lung injury in adults is pneumonia.

When ARDS is diagnosed with no obvious cause, reviewing the patient’s medications and recent diagnostic tests, procedures, and treatments might suggest an unrecognized cause, such as the use of a radiographic contrast agent. Contrast agents are a rare indirect cause of ARDS. When no cause can be determined, bronchoscopy or lung biopsy might reveal useful information.

Certain variables are associated with an increased risk of developing ARDS. Any critically ill person with a history of chronic alcohol abuse has a significantly
greater risk. A history of alcoholism also is associated with higher mortality in patients with ARDS. Other predisposing factors include advanced age, significant medical comorbidities, malignancy, renal disease, and a genetic predisposition to severe inflammation. To some degree, every patient in a hospital ICU is at risk for developing ARDS. Mechanically ventilated patients, in particular, are at an increased risk. Studies have shown that patients with diabetes are less likely to develop ARDS. Patients who have diabetes might have protection against ARDS because of hyperglycemia. Hyperglycemia causes impaired neutrophil function; neutrophils are thought to play a major role in the development of ARDS.

Illness and injury that do not have an obvious direct effect on the lungs can trigger ARDS, but the exact reason for this is not known. A most likely explanation is that the body produces harmful substances in response to an indirect lung injury and that these substances overwhelm the system and affect the lungs. It is not known why ARDS develops in some patients with direct or indirect acute lung injury but not in others; it might be explained by the balance between proinflammatory and anti-inflammatory factors in individuals.

ARDS is a specific type of respiratory failure and a general term describing inadequate gas exchange that prohibits a person from breathing on his or her own. Not everyone who has respiratory failure will develop ARDS.

### Signs and Symptoms

Symptoms of ARDS usually occur within 24 to 48 hours of the cause. They can develop more quickly when due to an infection or after aspiration of gastric contents. Characteristic dyspnea develops soon after the triggering lung injury. Patients develop hypoxemia and typically have tachypnea, shortness of breath, and hypoxia. ARDS patients might show signs and symptoms of hypotension, cyanosis, severe wheezing, fatigue, or excessive sweating. Cough and fever can be present, particularly if ARDS is caused by pneumonia. ARDS is characterized by refractory hypoxemia, which is insufficient oxygenation of arterial blood despite oxygen delivery. A patient who has acute hypoxemia might appear restless or anxious; he or she also might appear confused or have an altered level of consciousness. In ARDS, dyspnea becomes progressively severe, coinciding with increasing alveolar flooding and decreasing pulmonary compliance. An obvious alveolar infiltrate on a chest radiograph indicates alveolar flooding. When initial symptoms begin, hypoxemia can occur even before any changes are visible on a chest radiograph.

### Diagnostic Criteria

Since ARDS was first described in 1967, definitions of the syndrome have varied. In August 1967, Ashbaugh and Petty reported a case in a British journal in which they first described the syndrome. They described 12 patients ranging in age from 11 to 48 years who presented with similar symptoms. Despite different underlying causes, all of these patients had dyspnea, cyanosis resistant to supplemental oxygen, and bilateral chest infiltrates on chest radiographs. Most developed rapid respiratory distress within 48 to 72 hours of the precipitating cause, and many had symptoms that were preceded by severe trauma or viral infection. Physicians

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**Table**

<table>
<thead>
<tr>
<th>Phases of ARDS</th>
<th>Phase</th>
<th>Alternate Terms</th>
<th>No. of Days Since Symptom Onset</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute</td>
<td>Exudative, early inflammatory</td>
<td>0–3</td>
<td></td>
</tr>
<tr>
<td>Subacute</td>
<td>Proliferative, intermediate</td>
<td>4–10</td>
<td></td>
</tr>
<tr>
<td>Chronic</td>
<td>Fibrosing alveolitis, late</td>
<td>&gt; 10</td>
<td></td>
</tr>
</tbody>
</table>

**Box 2**

Direct and Indirect Causes of ARDS

**Direct lung injury:**
- Pneumonia or other lung infection
- Aspiration
- Near-drowning
- Inhalation injury

**Indirect lung injury:**
- Sepsis
- Severe trauma
- Drug overdose
- Acute pancreatitis

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realized that the symptoms occurring in adults were similar to the respiratory distress known to occur in newborns. Initially, the syndrome was termed adult respiratory distress syndrome to distinguish it from the respiratory distress syndrome of newborns. It was later recognized that the syndrome also can occur in children, and it was renamed acute respiratory distress syndrome.

In 1994 the American-European Consensus Conference established a definition of required criteria that was accepted worldwide. The conference definition for ARDS requires:2

- Acute onset.
- Bilateral infiltrates visible on a chest radiograph.
- Noncardiac causes.
- No left atrial hypertension.
- Pulmonary artery occlusion pressure of 18 mm Hg or less.
- Severe hypoxemia.
- A partial pressure arterial oxygen over fraction of inspired oxygen (PaO2/FiO2) value of 200 mm Hg or less, regardless of the positive end-expiratory pressure (PEEP) level. (A value between 200 to 300 mm Hg meets criteria for a diagnosis of acute lung injury.)

In 2012, an expert panel published a revised ARDS definition that includes a severity classification system. The Berlin definition for ARDS categorizes ARDS as mild, moderate, or severe. A major change from the 1994 American-European Consensus Conference definition is that the term acute lung injury has been abandoned. Patients who previously met the criteria for acute lung injury now have “mild ARDS.” The requirement that bilateral opacities consistent with pulmonary edema be present on an imaging examination was retained; however, the new definition recognizes bilateral opacity findings on either a chest radiograph or a CT scan. In general, the required clinical criteria are based on timing, radiography or CT chest imaging, and the origin of the pulmonary edema.

According to the Berlin definition, ARDS can be diagnosed after cardiogenic pulmonary edema has been ruled out. Other causes of acute respiratory failure and bilateral infiltrates also must be excluded. The Berlin definition requires all of the following criteria be met for an ARDS diagnosis:12,23:

- Respiratory symptoms must have begun within 1 week of a known clinical insult, or the patient must have new or worsening symptoms during the past week.
- Bilateral opacities consistent with pulmonary edema must be present on a chest radiograph or CT scan. These opacities must not be fully explained by pleural effusions, lobar collapse, lung collapse, or pulmonary nodules.
- The patient’s respiratory failure must not be fully explained by cardiac failure or fluid overload.
- A moderate to severe impairment of oxygenation must be present. The severity of the hypoxemia defines the severity of the ARDS.

Radiologic technologists should be aware that ARDS has been called by a variety of other terms, such as:

- Adult hyaline membrane disease.
- Postperfusion lung.
- Pump lung.
- Shock lung.
- Ventilator-associated lung injury.
- Adult respiratory insufficiency syndrome.

The terms primary graft dysfunction, primary graft failure, or transplant lung have been used to describe ARDS occurring immediately after lung transplantation. Lung shock was used during the Vietnam War when soldiers developed ARDS from the shock of their injuries.

Differential Diagnoses
Congestive Heart Failure

ARDS can be confused with other illnesses that have similar symptoms, especially congestive heart failure (CHF). It is essential to the diagnosis of ARDS that a patient’s pulmonary edema is not cardiac related.22 ARDS causes pulmonary edema that is not explained by heart failure. As in ARDS, CHF causes fluid to build up in the lungs, but there is no injury to the lungs in CHF (see Figure 3). Unlike the American-European Consensus Conference definition, the Berlin definition of ARDS dismisses the need to exclude heart failure because patients with CHF can simultaneously have ARDS. The new Berlin criterion states that respiratory failure cannot be fully explained by cardiac failure (or fluid overload).12,23 CHF and ARDS are manifestations
of unique diseases that demand different management strategies, so it is imperative to quickly differentiate the 2 forms of pulmonary edema.⁴

**Acute Lung Injury**

Acute lung injury, sometimes referred to as ALI, is associated with ARDS. Acute lung injury and ARDS represent a spectrum of lung injury, with ARDS being reserved for more severe gas exchange abnormalities.⁵ ARDS is considered the most severe form of acute lung injury.⁴ Acute lung injury that is severe enough to cause acute hypoxemic respiratory failure is commonly referred to as ARDS.⁶ ARDS and acute lung injury are on the same spectrum of clinical features, pathophysiology, and radiographic appearances. In this sense, all ARDS patients have acute lung injury, but all acute lung injury patients do not have ARDS. The definition of both ARDS and acute lung injury requires the presence of bilateral infiltrates on chest radiographs.¹¹ According to the Berlin definition, patients who previously met the criteria for acute lung injury would be diagnosed with mild ARDS because the diagnosis of acute lung injury is no longer used.¹⁵,¹³

**ARDS in Pediatric Patients**

ARDS can occur in children as well as adults.¹⁹ Most pediatric cases of ARDS appear in children younger than 4 years of age who have an underlying medical disorder such as prematurity or cardiac disease.¹⁴ Definitions of ARDS for children and adolescents are basically identical to those for adults. However, there are essential differences between pediatric patients and adults that can affect management strategies. Younger children might be at greater risk for ventilator-induced lung injury because their lungs are still developing.

In children, the leading cause of ARDS is pneumonia, and the highest mortality rates from ARDS are associated with near-drowning. Pediatric patients with ARDS represent a small percentage of pediatric ICU patients. Mortality rates in pediatric ARDS are similar between children and adults. Approximately 500 to 2000 children die from ARDS annually in the United States.²³ Mortality among children with ARDS has decreased significantly since the 1980s. Mortality of pediatric patients was between 65% to 80% in the 1980s and has declined to a current rate of about 20%.⁶

**Imaging ARDS**

Chest radiography and CT are important for the diagnosis and management of ARDS. Radiographic evidence is one of the diagnostic criteria for ARDS and can provide ongoing insight into the patient’s clinical state. CT now is a recognized, effective method of confirming the diagnosis but is more readily used for identifying complications.¹¹ Ultrasonography and positron emission tomography (PET) are both well-recognized imaging tools in ARDS. Currently no particular indication exists for magnetic resonance (MR) imaging with ARDS.²¹

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**Figure 3.** A chest radiograph consistent with congestive heart failure. Characteristic cardiomegaly, vascular congestion, and bilateral pleural effusions are shown. Image courtesy of Community Health Network, Community Hospital South, Indianapolis, IN.
Chest Radiography

The chest radiograph can serve several purposes in the diagnosis and management of ARDS. Interpretation of a chest radiograph supports the physician’s diagnosis, confirms tube position and line placement, helps to monitor disease progression, and detects complications in ARDS. Obtaining daily chest radiographs for ARDS patients is common and allows health care providers to monitor the patient’s progress. Chest radiography is widely available, relatively inexpensive, and aids in promptly detecting complications such as pneumothorax or hospital-acquired pneumonia. Prompt detection of complications is important in critically ill patients, and a change recognized on a serial radiograph might be the first or only clue. Common indications for a chest radiograph include:

- Diagnose and confirm ARDS.
- Detect a possible complication not accompanied by clinical signs or symptoms.
- Confirm complications suspected clinically.
- Monitor a patient’s condition.

Significant lung changes occur as ARDS progresses, and the changes can be visible on a chest radiograph. In ARDS, the radiographic abnormalities follow a predictable sequence, mirroring the histopathological changes occurring in the patient. The radiographic appearance of ARDS correlates with the syndrome’s histopathological stage.

Acute Phase

A radiographic latent period during the first 24 hours is typical following the initial insult that causes ARDS. Immediate chest radiographs frequently appear normal. For example, if a patient is suspected of aspirating (e.g., water in near-drowning cases or gastric contents from vomiting), evidence might not appear on a chest radiograph during the first 24 hours following the event. The exception to this rule are cases involving direct lung injury, such as pneumonia. Pneumonia would be evident as consolidation on a chest radiograph, reflecting the primary disease process. An initial normal chest radiograph reflects that histopathologically there is little alveolar edema.

After the initial 24 hours, rapid deterioration is common. In the following 24 to 72 hours, pulmonary edema begins developing. Progressive bilateral infiltrates develop without visible cardiomegaly. Histopathologically, there is leakage of fluid into the lung and inflammatory alveolar infiltrates. In this initial phase, the patient begins to experience hypoxemia and reduced pulmonary compliance. Bilateral interstitial infiltrates usually are seen within 24 hours; they often are more prominent in the lung bases and periphery of the lungs. Infiltrates can be patchy or diffuse, fluffy or dense, and pleural effusions might occur. Air space and interstitial opacities on the chest radiograph in ARDS are predominately bilateral and symmetrical.

Ultrasonography also is a sensitive method for detecting pleural effusions, but chest radiography is more commonly used. However, patient positioning affects the appearance of pleural fluid on a chest radiograph. If the patient is supine, effusion is distributed along the posterior chest and appears as a generalized haze that might obscure infiltrates. When the patient is in the more optimal upright position, pleural effusions appear as a curved outline of fluid in the posterior costophrenic angle.

As ARDS evolves, widespread ground-glass opacification becomes apparent. During the next 36 hours frank consolidation becomes apparent on chest radiographs, coinciding with the release of more inflammatory fluid into lung spaces. Diffuse bilateral pulmonary infiltrates on a chest radiograph reveal “white out”; complete opacification is seen in severe, advanced cases. More extensive opacities (3 or 4 quadrants on a chest radiograph) have been associated with the severe ARDS category.

Intermediate Phase

Typically, the radiographic changes of ARDS usually plateau after the initial catastrophic burst. After the rapid development of radiographic changes that occur in the acute phase, a generally stable period typically is apparent on radiographic images. Knowledge of the radiographically stable phase has practical value; any significant change in serial radiographic appearances can be the harbinger of nosocomial pneumonia. For example, the development of new focal areas of air space opacification would be a significant change of concern. New air space opacities likely indicate
hospital-acquired infection or ventilator-induced injury. Most patients who do not survive ARDS die during this phase, despite aggressive treatment.

**Late Phase**

In the final phase or late phase, the radiographic abnormalities begin to resolve with variable speed and duration. Although the lungs might revert to normal, coarse reticular opacities and cysts are likely a consequence of lung repair and barotrauma. There are characteristic radiologic changes during this phase that correspond to the coinciding histopathologic changes. Patchy areas of increased lucency appear within days to weeks. Radiologic improvement follows clinical resolution of ARDS. The length of the recovery period depends on various factors, including comorbidities.

In most patients, ARDS substantially resolves after the acute phase. For others, the syndrome progresses to a fibroing alveolitis. Fibroing alveolitis usually becomes clinically apparent after 7 to 10 days. Radiographically, linear opacities develop during evolving fibrosis. Histologically, pulmonary edema and neutrophilic inflammation are less prominent. A severe fibroproliferative process fills the air spaces with granulation tissue that contains an extracellular matrix rich in collagen and fibrin, as well as new blood vessels and proliferating mesenchymal cells (skeletal stem cells). This late fibroproliferative phase can last weeks, and sometimes small gains in pulmonary function are offset by new problems such as hospital-acquired infections, organ failures, or barotrauma.

The main radiographic differential diagnosis is cardiogenic pulmonary edema. It is possible for ARDS and cardiogenic pulmonary edema to exist simultaneously in a patient. Discriminating between the 2 using chest radiography alone is often difficult. ARDS-related edema and edema secondary to cardiac failure can be difficult to distinguish on radiographs; however, there are several characteristic differences. Cardiogenic pulmonary edema is characterized by visible cardiomegaly and a widened vascular pedicle on a chest radiograph. Cardiomegaly is associated with cardiac failure and is not a typical finding in ARDS. Cardiogenic pulmonary edema generally is characterized by diffuse central edema.

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**Figure 4.** A. Normal, clear chest computed tomography (CT) image. B. Chest CT image of a patient with severe ARDS demonstrating bilateral pleural effusions and bilateral airspace disease. Images courtesy of Community Health Network, Community Hospital South, Indianapolis, IN.
infiltrates. Diffuse bilateral infiltrates typical of ARDS usually are more peripherally than centrally located. Furthermore, focal infiltrates are more typical of lobar pneumonia, atelectasis, or lung contusion.\(^1\)

**Computed Tomography**

Chest CT imaging also can play an important role in ARDS.\(^5\) Cross-sectional imaging with CT can be of more assistance than chest radiography under certain circumstances. Because of the characteristic widespread cloudy appearance of ARDS (indicative of diffuse opacification), a chest radiograph can be of limited assistance in identifying complications. Compared with chest radiography, CT provides better contrast resolution and anatomical visualization. In a deteriorating ARDS patient, CT can detect ventilation-associated complications and foci of infection that might not be apparent on a chest radiograph.\(^1\) In addition, chest CT can be helpful in differentiating between atelectasis and consolidation.\(^2\) However, the benefits of chest CT imaging must outweigh the risks associated with transporting a patient to the radiology department\(^1\) and the radiation exposure associated with CT.\(^6\)

CT use for evaluating ARDS is widespread, both for clinical and research purposes. It is helpful in confirming, problem solving, classifying, and prognosticating ARDS.\(^10\) CT imaging has been used extensively as an investigative tool to better define the nature of the infiltrates in patients who have ARDS.\(^5,17\) CT also has been a helpful research tool, leading to better understanding of the ARDS disease process.\(^17\) Furthermore, CT can assist in determining ventilation strategies and prognosis.\(^7\) Some indications for a chest CT scan include\(^7\):

- Problem solving when a patient has a complex appearance seen on a chest radiograph.
- Better quantification of the extent of disease than is possible with chest radiography.
- Determining the etiology when it is unknown.
- Imaging in clinical trials for new ARDS drug therapies.
- Determining the extent of lung damage when assessing outcomes in ARDS survivors.
- Diagnosing ARDS (when adhering to the Berlin diagnostic criteria).
- Identifying complications.
- Consideration of a patient for prone therapy.\(^10\)

It is not uncommon for patients who present with severe acute hypoxemia to initially have no major evidence of infiltration on a chest radiograph. Despite a normal chest radiograph, CT almost always shows evidence of “ground glass” infiltration that signifies microcollapse or edema.\(^27\) Ground-glass opacities on CT appear as hazy areas of increased attenuation amid visible, unobscured bronchial and vascular margins. In contrast, when there is consolidation, bronchovascular margins are obscured.\(^28\)

Ground-glass opacification is generally the most extensive CT pattern during the acute stages of ARDS. This sign likely denotes filling of the interstitium and alveoli with inflammatory fluid.\(^10\) Ground-glass opacification on CT is a nonspecific sign that reflects an overall reduction in the air content of the affected lung. Other common findings present on chest CT scans of ARDS patients include diffuse consolidation with air bronchograms, bullae, pleural effusions, and possibly pneumomediastina and pneumothoraces.\(^3\) Another observed CT feature in acute ARDS is bronchial dilation within areas of ground-glass opacification.\(^21\)

CT has shown that consolidation is not as diffuse in ARDS as chest radiography findings suggest.\(^21\) CT scans of the lungs show both normal-appearing areas and consolidated injured areas.\(^19\) While distribution of infiltrates visible on CT appears surprisingly patchy, areas of alveolar filling and consolidation occur predominantly in dependent lung zones,\(^4\) the lowest part of the lung in relation to gravity. CT has confirmed that consolidation in ARDS is typically in the gravity-dependent areas of the lungs.\(^21\) Therefore, CT has led to the understanding that in ARDS, air-space collapse occurs more in dependent lung zones.\(^15\) The classical CT appearance of acute-phase ARDS is opacification demonstrating an anterior-posterior density gradient within the lung, with dense consolidation in the most dependent regions, merging into a background of widespread ground-glass attenuation and then normal or hyperexpanded lung in the nondependent regions (see Figure 4).

CT can be of assistance when the cause of a patient’s ARDS is not known. The identification of nondependent opacities on CT in the acute stage might provide
a clue as to whether ARDS was secondary to direct or indirect lung injury. This is significant information because responses to mechanical ventilation differ between pulmonary (direct) and extrapulmonary (indirect) ARDS patients. Lung compliance is significantly lower in patients with pulmonary ARDS, which might be worsened by increasing PEEP. Conversely, increasing PEEP can have a positive effect when ARDS is due to an indirect injury. On CT imaging, ARDS resulting from a direct lung injury tends to show an asymmetrical mix of both consolidation and ground-glass opacification. When the cause is an indirect lung injury, predominately symmetric ground-glass opacification is seen.

**Ultrasoundography**

Chest ultrasonography can play a role in assessing ARDS. There are many benefits to using ultrasonography to evaluate chest diseases in critically ill patients. Some advantages are that it uses no radiation, can be performed bedside, is relatively inexpensive, and is noninvasive. It is a useful diagnostic tool for evaluating pleural effusions, which are abnormal amounts of pleural fluid (see Figure 5). Ultrasonography is more accurate than chest radiography in detecting pleural effusions, with a sensitivity rate of almost 100%. It also is highly sensitive in distinguishing between solid lung lesions and pleural fluid.

There are 2 types of pleural effusions: exudative and transudative. Generally, exudative pleural fluid is from lung/pleura inflammation. Transudative effusions develop without any damage to the pleural space. Distinguishing between the 2 types of effusions can be useful in treatment and diagnosis. Ultrasonography can assist in determining which type of pleural effusion a patient has. Pleural effusions are sometimes seen in ARDS; however, among the many causes of pleural effusions, CHF is the most common cause. CHF is associated with the transudative type of pleural effusions.

Ultrasonography also is helpful during invasive procedures, such as pleural draining. When used as a guidance tool during thoracentesis, ultrasonography reduces the risk of pneumothorax from about 18% to 3%. Ultrasonography also can be useful in detecting pneumothorax following the procedure because it uses no ionizing radiation, which is a particular advantage for pregnant and pediatric patients. In addition, it can be performed rapidly and at the patient’s bedside.

Lung ultrasonography can be beneficial in helping to diagnose ARDS, particularly during the acute phase. While chest radiography can yield inaccurate results when trying to distinguish between cardiogenic pulmonary edema and ARDS-related pulmonary edema, distinguishing between the 2 is very important. CT is considered accurate in making this distinction, but it exposes patients to high amounts of ionizing radiation, is costly, and can involve difficulty in transporting patients to the CT scanner. Lung ultrasonography, however, is helpful in distinguishing between the 2 types of edema and can be performed easily on a patient who has mechanical ventilation.

Some common ultrasonography findings in patients with ARDS include septal lines, also called Kerley B lines, which are short, faint linear shadows that occur from septal thickening. They measure 1 cm to 3 cm in length and typically are seen along the lower lateral lung margins. Other findings might include spared areas of normal lung parenchyma, consolidated areas, pleural line abnormalities, and pleural effusions. ARDS can be classified as focal or diffuse based on the distribution of aeration loss as detected by ultrasonography. Differentiating between the 2 can be important because mechanical ventilation strategies might be altered accordingly for optimal results.

Figure 5. Sonogram demonstrating collected pleural fluid. Image courtesy of Community Health Network, Community Hospital South, Indianapolis, IN.
**Positron Emission Tomography**

Fluorodeoxyglucose PET imaging of ARDS has been beneficial in experimental research, leading to a better overall understanding of ARDS. Clinically, it might be useful when evaluating therapeutic interventions and as an investigational tool in ARDS patients. In patients who have ARDS, fluorodeoxyglucose PET can be used to monitor lung neutrophils, which are the essential cells in the pathophysiologic mechanisms of acute lung injury. Lung inflammation is a key feature of acute lung injury. Neutrophils become highly activated in response to inflammatory stimuli and are influential in the severity of lung injury during ARDS. Fluorodeoxyglucose-PET imaging in ARDS noninvasively assesses lung neutrophil infiltration and activation. Fluorodeoxyglucose-PET scans have shown that actual lung inflammation can be more substantial than suspected. These types of images show inflammation in areas of the lung that might not appear on radiographs.

**Treatment**

ARDS mortality rates are declining thanks to advances in critical care. The main goals in treating ARDS patients are to raise their blood oxygen levels and maintain adequate gas exchange; another goal is to eliminate the underlying cause of ARDS. Recent ARDS treatment strategies have proved effective in improving patient outcomes. For example, strategies that aim to control the systemic inflammatory response that can lead to lung and other organ injuries have improved. Administering a neuromuscular blockade with initial mechanical ventilation, turning patients to a prone position, and extracorporeal membrane oxygenation (ECMO) are some potentially beneficial strategies.

Lung-protective ventilation is the most essential component to better ARDS outcomes. Normal ventilation is cyclical and occurs in 2 phases: inspiration and expiration. The volume of gas moving in or out of the respiratory tract measured during inspiration or expiration is called the tidal volume. Alveolar hyperinflation during ventilation is known to induce lung tissue injury known as volume trauma. An effective lung-protective ventilation strategy to avoid volume trauma involves the use of lower tidal volumes and limited pressure. The low-tidal-volume strategy is critical for reducing mortality in ARDS.

ARDS is a clinical syndrome that has treatment requirements apart from its underlying cause. However, in addition to treating ARDS, it is imperative to identify the syndrome’s cause and treat it aggressively. For example, antibiotics are given if the cause of ARDS is known to be an infection. The overall care for ARDS patients requires recognizing and treating the underlying medical disorder, as well as providing adequate nutrition and minimizing procedures that can cause complications. Comprehensive care also includes measures to prevent venous thromboembolism, gastrointestinal bleeding, aspiration, excessive sedation, and central venous catheter infections. Managing fluid intake to maintain an adequate fluid balance is another important ARDS treatment strategy because too much fluid in the body can lead to fluid buildup in the lungs.

**Ventilation**

A hallmark of ARDS is profound hypoxemia despite increasing oxygen delivery, also known as refractory hypoxemia. An oxygen mask might be used, but most patients with ARDS are admitted to the ICU and placed on a mechanical ventilator. Mechanical ventilation decreases the work of breathing and improves oxygen transport. Studies of surviving patients have shown that the median duration a patient was dependent on a mechanical ventilator increased with the severity of the patient’s ARDS.

Mechanical ventilation can be life saving, but it also can be harmful. Evidence suggests that inappropriate mechanical ventilation techniques can actually cause or worsen ARDS. Ventilation at very high volumes and pressures can injure normal lungs, but an already injured lung might be more susceptible. In the injured lung, even tidal volumes that are well tolerated in the normal lung can lead to alveolar overdistension. Alveolar overdistension combined with cyclic opening and closing of alveoli is particularly likely to induce lung injury, and a proinflammatory cascade might be triggered.

A primary goal in treating ARDS is to give the patient enough oxygen to prevent organ failure. A
A well-established supportive breathing method is the use of PEEP. By controlling the pressure in the lungs, high PEEP can help increase lung functioning and reduce the risk of lung injury related to using a ventilator. Improved oxygenation can be obtained in many ARDS patients by increasing PEEP. This ventilation strategy was originally suggested by Ashbaugh et al in 1967. PEEP works by preventing the patient from completely exhaling. This recovers collapsed alveoli, which promotes gas exchange.

In ARDS, the lungs stiffen, decreasing their ability to make surfactant and consequently requiring higher levels of PEEP. ARDS patients typically experience problems maintaining adequate oxygenation, even when high amounts of oxygen are being delivered. The prolonged use of FiO₂ concentrations of 60% or more is more likely to cause oxygen toxicity and lung damage. For this reason, PEEP is an essential therapy for mechanically ventilated ARDS patients. Limiting peak inspiratory ventilator pressures as much as possible is important because high levels of peak positive pressure can cause barotrauma.

**Extracorporeal Membrane Oxygenation**

Despite appropriate treatment with mechanical ventilation, some ARDS patients exhibit profound refractory hypoxemia. Initial management in these patients is aimed at maintaining adequate oxygen. Increased sedation, and occasionally neuromuscular paralysis, can help maintain adequate oxygenation. Other methods of treatment are available for patients who do not respond to conventional treatment with low-tidal-volume mechanical ventilation. For patients who remain persistently hypoxic, an alternative pulmonary support method is ECMO. ECMO might be used in patients with severe respiratory failure such as ARDS who have severe refractory hypoxemia. ECMO provides gas exchange while bypassing the heart and lungs. Some blood is removed from the patient, oxygen is infused and carbon dioxide is removed, and then the blood is returned to the patient.

ECMO in combination with lung-protective ventilation has been used with some success with adults and children, particularly with ARDS caused by H1N1 influenza. Recent publications have reported survival rates of more than 70% in these patients. One study reported an overall survival rate with ECMO for pediatric patients with ARDS as 54%. ECMO therapy is reserved for the most severe cases of ARDS when patients don’t improve with traditional management because ECMO allows the lungs to rest and heal. In extreme cases of ARDS, ECMO might aid in recovery by enabling optimal lung-protective ventilation. However, ECMO is costly, labor intensive, and has many potential complications. Typically a “last-resort measure” for adult patients, the treatment is considered controversial.

**Positioning Therapy**

Ventilating patients with ARDS can be particularly challenging. Pulmonary edema causes lungs to become stiff and noncompliant, making ventilation more difficult. The fluid in the alveoli causes refractory hypoxemia. Positioning therapy, especially prone therapy, can improve lung recruitment and oxygenation. Prone positioning improves oxygenation in some cases. Recent studies conclude that ventilating in the prone position is effective for improving oxygenation and outcomes, particularly in severely hypoxemic ARDS patients. A recent study showed that early and prolonged prone positioning might improve survival.

Prone positioning typically involves positioning a patient on his or her abdomen for a portion of the day. The ventilated patient can be placed in the prone position by manually turning the patient or using a mechanically rotating bed. Hospitals can rent specialized beds for qualifying ARDS patients, such as a RotoProne bed (see Figure 6). There are many benefits to using mechanically rotating beds instead of relying on staff to physically turn the ventilated patient. For example, it takes multiple staff to physically turn a patient. Depending on the patient’s size, the task could require 4 to 8 people. A specialized rotating bed allows 1 or 2 people to make position changes.

Prone positioning improves oxygenation because it:
- Allows the patient’s dependent lung areas to expand, opening collapsed alveoli.
- Reduces the pressure on the lungs from overlying organs, reducing the effort required for breathing, allowing more energy for healing.
Promotes drainage, allowing more suction of secretions. 
Might help prevent further lung injury associated with use of a mechanical ventilator.

However, prone positioning also can cause significant facial edema, which can be upsetting for the patient’s family. 

Complications of ARDS

The complications that occur as a result of ARDS can be life threatening, and some are related to simply being in an ICU. Three severe complications that occur in ARDS patients are barotrauma, hospital-acquired pneumonia, and organ failure.

Ventilator-induced barotrauma is a well-recognized complication. Although it can occur in any mechanically ventilated patient, it is most frequently encountered in ARDS patients. Barotrauma describes the manifestations of extra-alveolar air that occur as a complication of positive pressure ventilation. Clinical signs vary, ranging from no obvious symptoms with subtle radiographic findings to respiratory distress or cardiac arrest due to a large tension pneumothorax. Pneumothorax describes the presence of abnormal air within the pleural space (see Figure 7). It can be life threatening, especially if it is large or under tension; immediate diagnosis and chest tube placement are essential. Excessive airway pressure during mechanical ventilation also can induce pneumomediastinum, pulmonary edema, and subcutaneous emphysema. Pneumothorax also can be caused by puncture during certain line placements.

A chest radiograph usually is sufficient for diagnosing a pneumothorax. Air leak syndromes are a common finding on chest radiographs of ARDS patients. If pneumothorax is suspected, an upright chest radiograph should be acquired. In a supine image, a pneumothorax is more easily missed because it becomes less visible. A decubitus chest radiograph also can be useful for detecting a pneumothorax. Two well-established signs of abnormal air collections include the deep sulcus sign and the double diaphragm sign.

The incidence of hospital-acquired pneumonia in patients with ARDS varies widely, from 15% to 60%. Chest radiography aids in detecting hospital-acquired pneumonia. Uncontrolled infection can lead to death in an ARDS patient; therefore, timely diagnosis and treatment is imperative. Clinical signs and symptoms of pneumonia include fever, an elevated white blood cell count, secretions consisting of pus, and lung infiltrates. These signs, however, often are already present in patients who have ARDS without pneumonia.

Although ARDS is often thought of as a primary pulmonary disorder, evidence suggests that ARDS is a systemic disorder. Multisystem organ dysfunction is a common complication in ARDS and can result from underlying causes of ARDS, such as sepsis, or it can occur independently. Patient survival ultimately depends on the successful support of the failing organs. Patients with ARDS often die from multigorgan dysfunction/failure due to systemic inflammation, not from the respiratory failure they have. ARDS patients also are at risk for developing sepsis during their hospital stay.

Prognosis

Mortality

Although mortality rate estimates vary, ARDS outcomes appear to be improving; a decline in mortality was cited in several recent studies. Improved outcomes are likely the result of better treatment strategies for sepsis and improvements in mechanical ventilation. Despite improvements in critical care, ARDS currently has a high mortality rate of approximately 40%. 

Figure 6. A mechanically rotating hospital bed used for prone therapy. RotoProne image reprinted with permission from ArjoHuntleigh.
In recent studies, mortality was shown to increase with disease stage. In ARDS cases that are severe, the mortality rate is more than 40%. Sepsis resulting in multiple organ failure is the leading cause of death in adult patients with ARDS. Twenty percent of all ARDS patients die of refractory hypoxemia; however, death is more often caused by multiorgan failure than respiratory dysfunction. A major study concluded that the risk of in-hospital mortality was highest in ARDS patients with sepsis (43%). Patients who have pneumonia-induced ARDS have a moderate risk of in-hospital death (46%), as do those who have ARDS caused by aspiration (37%). Risk of death from ARDS caused by multiple trauma is 11%. Overall, increased mortality and worsened prognosis in ARDS patients are linked to advanced age, sepsis, chronic liver disease, organ insufficiency, and organ dysfunction.

**Quality of Life in ARDS Survivors**

ARDS patients typically experience prolonged respiratory failure and are temporarily dependent on a ventilator for survival. They can have minimal long-term pulmonary consequences, but some develop severe lung fibrosis. In lung fibrosis, lung tissue becomes thick, stiff, and scarred, interfering with a person’s ability to breathe (see Figure 8). However, most survivors recover with normal or near-normal lung function. For most ARDS survivors, pulmonary function returns to close to normal within 6 to 12 months of recovery. Survivors of severe ARDS might be more likely to suffer from persistent pulmonary symptoms and neuromuscular weakness. One recent study concluded that a longer duration of ECMO is associated with reduced total lung capacity at follow-up. Pulmonary rehabilitation therapy might be needed during recovery, and can help strengthen the patient’s respiratory system and increase lung capacity. Considering the severe oxygenation problems and impaired respiratory system compliance characteristic of ARDS, it is remarkable that survivors often have near-normal pulmonary function tests 6 to 12 months after recovery.

Despite lung recovery, survivors face a reduced quality of life attributable to ARDS for at least 5 years. Specifically, they are at risk for impaired functional capacity (ie, exercise tolerance) and mental health problems. In a study of survivors at 5-year follow-up, researchers concluded that exercise limitation, physical and neuropsychological impairments, reduced physical capacity, and high health care costs were consequences of ARDS.

According to recent studies, survivors frequently have long-term functional disability, cognitive dysfunction, and psychosocial problems. In one study, ARDS survivors were compared with patients who survived a similarly severe illness. The ARDS survivors reported significant decreases in health-related quality of life, such as reduced physical function. However, patients who survive an episode of ARDS do not appear to have
a higher subsequent mortality rate than other ICU patients who were similarly ill.\textsuperscript{19}

Largely as a result of ICU-acquired weakness, most patients who survive an episode of ARDS sustain some degree of permanent disability and reduction in health-related quality of life.\textsuperscript{19} Six-minute walking distances were persistently low at 12 months, largely because of persistent muscle wasting and weakness rather than pulmonary function abnormalities.\textsuperscript{5,19} A recent study found that patients who had better functional capacity after ARDS\textsuperscript{19}:

- Did not receive systemic corticosteroid medication.
- Avoided hospital-acquired illness.
- Experienced rapid and successful resolution of their lung injury.

In addition, ARDS survivors have high rates of depression, anxiety, and posttraumatic stress disorder.\textsuperscript{5,7} Herridge reports that survivors have been found to have significant signs and symptoms of posttraumatic stress disorder.\textsuperscript{38} Another study by Herridge et al established that ARDS survivors had diminished cognitive (ie, intellectual) function. The authors concluded that severe hypoxemia during inpatient treatment was a key contributor to the cognitive dysfunction.\textsuperscript{40}

**Adverse Effects on the Brain**

ARDS can cause significant long-term brain-related problems, notably brain atrophy, lesions, and neurocognitive impairments. ARDS survivors who underwent CT brain imaging showed cognitive impairments, significant brain atrophy, and ventricular enlargement. Specifically, 53% of imaged patients had atrophy or lesions noted by the interpreting radiologist.\textsuperscript{41} Hypoxemia during ARDS is the probable cause for persistent cognitive impairments in survivors.\textsuperscript{40}

**Follow-up Imaging**

Radiographic findings during follow-up can be the result of ARDS or the result of ventilator-induced lung injury.\textsuperscript{21} On follow-up chest imaging, radiologic changes will have completely resolved in most cases; however, persistent changes are demonstrated in some patients. These changes often are attributed to fibrosis, although a small number of patients show pleural thickening or small cysts.\textsuperscript{19}

*Figure 8. Posteroanterior (A) and lateral (B) chest radiographs demonstrating fibrosis, or lung scarring that sometimes occurs in ARDS survivors. Image courtesy of Community Health Network, Community Hospital South, Indianapolis, IN.*
Bronchial dilatation is a frequent finding on CT scans of patients with ARDS, and airway dilatation seems to persist in most survivors. Airway dilatation is associated with ground-glass opacification on CT scans of survivors. Ground-glass opacification seen at follow-up is probably different from the opacification seen in acute patients. Because there are dilated airways in areas of ground-glass opacification, the likely explanation is that this finding in survivors represents fine intralobular fibrosis, which is below the resolution limits of thin-section CT.¹⁰

CT abnormalities have been found during follow-up imaging of ARDS survivors. Higher levels of lung fibrosis are present on CT images in patients who spent the most time on mechanical ventilation and who had the highest levels of PEEP. According to studies, direct-injury ARDS survivors are more likely to have pulmonary fibrosis on follow-up CT.¹¹ A possible explanation for this is that patients with pulmonary ARDS are more prone to ventilator-induced lung injury. Fibrosis likely develops in these patients from barotrauma secondary to mechanical ventilation. Sheard et al reported that 76% of patients in their study had abnormalities on high-resolution CT at 6-month follow-up.¹¹ Areas of reticular and ground-glass opacification were the most prominent findings. They concluded that ground-glass opacities probably represent small areas of fibrosis.¹¹

Herridge suggested that radiographic findings in survivors seen on follow-up imaging might reflect ventilator-induced lung injury. CT findings observed at 3-year follow-up included reticular changes, areas of decreased attenuation, and ground-glass opacities.¹³

Conclusion

ARDS is a public health problem encountered frequently by radiologic technologists who care for critically ill patients.⁴ When providers recognize ARDS risk factors early and avoid known aggravating factors during a patient’s hospital stay, they can help decrease its incidence.²² Management of ARDS patients is complex because of the syndrome’s severity, and although many survivors recover with normal or near-normal pulmonary function, they can suffer from other ailments and incur major health costs as a result of this illness.¹ The cumulative cost of ARDS in terms of both human lives and medical resources is high.¹ Radiologic technologists play an important role in the diagnosis of ARDS, monitoring its treatment, and follow-up.

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References


Acute Respiratory Distress Syndrome

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Read the preceding Directed Reading and choose the answer that is most correct based on the article.

1. Which imaging modalities are useful for monitoring patients with acute respiratory distress syndrome (ARDS) and detecting possible complications such as pneumothorax?
   a. computed tomography (CT)
   b. radiography
   c. ultrasonography
   
   a. 1 and 2
   b. 1 and 3
   c. 2 and 3
   d. 1, 2, and 3

2. What is the primary site for gas exchange?
   a. bronchioles
   b. alveoli
   c. bronchi
   d. trachea

3. Hypoxemia develops when:
   a. fluid builds up around the patient’s heart.
   b. more fluid is needed intravenously.
   c. alveoli fill with fluid.
   d. the patient’s airways open up.

4. Patients who have ______ are at very high risk for developing ARDS.
   a. osteoarthritis
   b. hyperglycemia
   c. sepsis
   d. hyperthyroidism

5. A history of ______ is associated with a significantly higher mortality rate in ARDS patients.
   a. alcoholism
   b. radiation exposure
   c. pancreatitis
   d. psychological illness

continued on next page
6. Patients with ______ are less likely to develop ARDS.
   a. cancer
   b. H1N1 influenza
   c. renal disease
   d. diabetes

7. ARDS was first described in:
   a. 1936.
   b. 1967.
   c. 1994.
   d. 2012.

8. Which of the following terms is another name that has been used to refer to acute respiratory distress syndrome?
   a. adult respiratory distress syndrome
   b. severe acute respiratory syndrome
   c. acquired respiratory distress syndrome
   d. latent pulmonary distress syndrome

9. According to the Berlin definition, diagnostic criteria for ARDS require that symptoms begin within ______ of a known clinical insult.
   a. 48 hours
   b. 72 hours
   c. 1 week
   d. 2 weeks

10. The severity of ______ defines the severity of ARDS.
    a. blood loss
    b. hypoxemia
    c. injury
    d. inflammation

11. The symptoms of ARDS can be confused with other illnesses, in particular:
    a. severe acute respiratory syndrome.
    b. congestive heart failure.
    c. asthma exacerbation.
    d. exacerbation of chronic obstructive pulmonary disease.

12. In children, the main cause of ARDS is:
    a. near-drowning.
    b. pneumonia.
    c. trauma.
    d. influenza.

13. Since the 1980s the mortality rate among pediatric patients with ARDS has:
    a. increased slightly.
    b. remained constant.
    c. decreased slightly.
    d. decreased significantly.

14. On chest radiographs, interstitial opacities associated with ARDS are predominately:
    a. bilateral and symmetrical.
    b. unilateral and patchy.
    c. diffuse and central.
    d. focal and central.

15. A characteristic radiographic appearance of ARDS is:
    a. cardiomegaly.
    b. “white out.”
    c. ascites.
    d. presence of Kerley B lines.
16. Following the acute phase of ARDS, new air space opacities noted on a serial chest radiograph most likely indicate that:
   a. the patient’s condition will improve.
   b. the syndrome was caused by an indirect lung injury.
   c. organs are rapidly failing.
   d. the patient has developed a hospital-acquired infection or ventilator-induced injury.

17. Chest CT shows that areas of alveolar filling and consolidation occur predominately in:
   a. independent lung zones.
   b. nongravity lung zones.
   c. dependent lung zones.
   d. distal lung zones.

18. Which of the following is mentioned as a use for fluorodeoxyglucose positron emission tomography imaging in patients with ARDS?
   a. detecting pneumothorax
   b. guiding thoracentesis
   c. monitoring neutrophils
   d. distinguishing between transudative and exudative pleural effusions

19. Profound hypoxemia despite increasing oxygen delivery is known as:
   a. refractory hypoxemia.
   b. tension hypoxemia.
   c. hyperventilation.
   d. cerebral hypoxemia.

20. A primary goal of ARDS treatment is preventing:
   a. fluid retention.
   b. air leaks.
   c. congestive heart failure.
   d. organ failure.

21. In some patients, improved ventilation can be accomplished using supine positioning.
   a. true
   b. false

22. Which of the following severe complications occur in ARDS patients?
   1. barotrauma
   2. hospital-acquired pneumonia
   3. organ failure
   a. 1 and 2
   b. 1 and 3
   c. 2 and 3
   d. 1, 2, and 3

23. When compared to intensive care unit patients who survive similar illnesses, ARDS survivors have a significantly higher mortality rate.
   a. true
   b. false

24. ARDS survivors have been found to have significant signs and symptoms of:
   a. emphysema.
   b. posttraumatic stress disorder.
   c. bipolar disorder.
   d. chronic bronchitis.

25. According to the article, which of the following are seen on follow-up images of ARDS survivors?
   1. ground-glass opacifications
   2. bronchial dilatation
   3. lung fibrosis
   a. 1 and 2
   b. 1 and 3
   c. 2 and 3
   d. 1, 2, and 3