Acute respiratory distress syndrome (ARDS)

The right clinical information, right where it's needed
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## Disclaimer
Most common symptoms and signs are dyspnea and hypoxemia, which progress to acute respiratory failure.

Common causes are pneumonia, sepsis, aspiration, and severe trauma.

Mortality is between 30% and 50%.

Low tidal volume, plateau-pressure-limited mechanical ventilation is the primary treatment that has been shown to reduce mortality. In severe acute respiratory distress syndrome (ARDS), neuromuscular blockade and prone positioning may improve clinical outcomes.

Complications include pneumothorax, ventilator-associated pneumonia, multiple organ failure, and pulmonary fibrosis with prolonged respiratory failure.

This topic covers ARDS in patients over the age of 12 years.
**Definition**

Acute respiratory distress syndrome (ARDS) is a noncardiogenic pulmonary edema and diffuse lung inflammation syndrome that often complicates critical illness. The diagnosis of ARDS is based on fulfilling three criteria:

- Acute onset (within 1 week)
- Bilateral opacities on chest x-ray
- PaO₂/FiO₂ (arterial to inspired oxygen) ratio of ≤300 on positive end-expiratory pressure (PEEP) or continuous positive airway pressure (CPAP) ≥5 cm H₂O.[1]

If no risk factors for ARDS are present, then acute pulmonary edema as a result of heart failure should be ruled out.

**Epidemiology**

Overall, 10% to 15% of patients admitted to the intensive care unit meet the criteria for ARDS,[2] [3] with an increased incidence among mechanically ventilated patients.[2]

The incidence of ARDS is estimated at 64 cases in 100,000 people, or 190,000 cases per year in the US. This incidence rate is 2 to 40 times greater than previous estimates, which probably does not represent a rising incidence but rather a historical underestimation.[4] The incidence of ARDS may be higher in the US than in Europe and other developed countries,[5] although evidence suggests that rates in the US may be declining.[6]

Critical illness, cigarette smoking, and alcohol use are predisposing factors for ARDS.[7] [8] Sex, ethnicity, and race have not been associated with the incidence of ARDS.

The mortality of ARDS is approximately 30% to 50%,[3] [4] although mortality in large clinical trials seems to be steadily decreasing.[9] The distinction between mild (PaO₂/FiO₂ 200-300), moderate (PaO₂/FiO₂ 100-200), and severe (PaO₂/FiO₂ ≤100) ARDS has been associated with clinical outcomes.[1] Ongoing research suggests there are at least two discrete ARDS subphenotypes, although the clinical implications of this are under investigation.[10] [11] [12]

**Etiology**

Many different conditions can lead to ARDS, although sepsis is the most common cause, usually with a pulmonary origin (e.g., pneumonia).[4] Other conditions associated with ARDS include aspiration, inhalation injury (including e-cigarette or vaping product-associated lung injury [EVALI]), acute pancreatitis, trauma, burns, pulmonary contusion, transfusion-related lung injury, cardiopulmonary bypass, fat embolism, disseminated intravascular coagulation, and drug overdose.[13]

ARDS is a common feature of infection with severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), the virus responsible for pandemic coronavirus disease in 2019-2020 (COVID-19). Early reports indicate that older age, neutrophilia, and organ and coagulation dysfunction are risk factors associated with the development of ARDS, and progression from ARDS to death, in patients with COVID-19 pneumonia.[14]
Pathophysiology

The pathophysiology of ARDS is complex and incompletely understood.[15] Early in the development of ARDS, the primary pathologic finding is diffuse alveolar damage, although this is not seen uniformly in all patients. The diffuse alveolar damage leads to injury to the alveolar-capillary membrane, made up of type I and type II alveolar pneumocytes and capillary endothelial cells. The alveolar air spaces are subsequently flooded with proteinaceous edema fluid, inflammatory cells (neutrophils and activated alveolar macrophages), and inflammatory mediators, including pro-inflammatory cytokines, lipid mediators, and oxidants. Epithelial injury may be severe, with necrosis and sloughing of the type I cells exposing the basement membrane. Fibrin deposition occurs along the denuded basement membrane, resulting in the hyaline membranes that are characteristic of diffuse alveolar damage. Injury to type II cells and alveolar flooding contribute to surfactant dysfunction.

Mechanical ventilation with high pressures and high volumes may further injure the lung, contributing to the pro-inflammatory cytokine cascade.

The early phase of ARDS manifests clinically as acute hypoxemic respiratory failure with an increased alveolar-arterial oxygen gradient and poorly compliant lungs. Concomitant multiple organ failure may occur, particularly if the underlying cause of ARDS is sepsis. Right ventricular dysfunction is also common and is associated with worse outcomes.

After the acute onset of alveolar flooding and inflammation, some patients have rapid resolution and return to normal lung histology and function. Pulmonary edema fluid is cleared by active transport of sodium and chloride across the alveolar epithelium. In other patients, this early exudative inflammatory phase progresses to a fibroproliferative phase. During this later phase, the lung develops organized fibrous tissue and collagen deposition, which leads to irreversible and sometimes catastrophic lung fibrosis.[13] This phase is characterized by continued respiratory failure, high minute ventilation, and poorly compliant lungs.
Case history

Case history #1

A 60-year-old man presents with acute onset of shortness of breath, fever, and cough. A chest x-ray shows a right lower lobe infiltrate, and sputum has gram-positive diplococci. He is given intravenous antibiotics but his respiratory status declines over 24 hours. He becomes hypotensive and is transferred to the intensive care unit. He is intubated for hypoxemia and requires vasopressors for septic shock despite adequate volume resuscitation. He requires high levels of inspired oxygen (FiO₂) and positive end-expiratory pressure (PEEP) on the ventilator to keep his oxygen saturation >90%. Repeat chest x-ray shows bilateral alveolar infiltrates, and his partial pressure of oxygen, arterial (PaO₂)/FiO₂ ratio is 109.

Step-by-step diagnostic approach

Because the diagnosis of ARDS is based on clinical criteria rather than a pathologic diagnosis, ARDS should be considered in all critically ill patients. As many as 40% of patients who meet the criteria for ARDS are never diagnosed with the condition.[35] [36] If patients develop new bilateral infiltrates on chest x-ray, they may have or may be developing ARDS. The importance of evaluating patients for the development of ARDS stems primarily from the survival benefit gained by ventilating with a low tidal volume, plateau-pressure-limited ventilator strategy.

History

The history should be directed at determining whether there is an underlying condition associated with ARDS, such as sepsis, pneumonia, aspiration of gastric contents, pancreatitis, blood transfusions, severe trauma, or e-cigarette use/vaping. The underlying cause can be an important determinant of outcome; patients with ARDS due to sepsis generally have the highest mortality. Specific treatments directed at the underlying cause are warranted, with particular attention to source identification and treatment in the context of sepsis. Symptoms that suggest ARDS include the acute onset of shortness of breath and hypoxemia leading to acute respiratory failure, and cough with expectoration of frothy pulmonary edema. The history should also collect information that might suggest an alternate diagnosis of an ARDS mimic, such as pulmonary edema secondary to heart failure, diffuse alveolar hemorrhage due to pulmonary vasculitis, collagen vascular disease, or acute eosinophilic pneumonia.[37]

Examination

Physical examination findings that support the diagnosis of ARDS are acute hypoxic respiratory failure requiring high levels of oxygen and/or positive end-expiratory pressure (PEEP) to maintain an oxygen saturation >90%. Both peak inspiratory pressure and end-inspiratory plateau pressure are also increased. Lung examination may reveal basilar or diffuse rales.[38] Particular attention should be put on identifying the source of infection if sepsis is suspected to be the underlying cause of ARDS.

Investigation

Key tests include arterial blood gas analysis for calculation of the partial pressure of oxygen, arterial (PaO₂)/inspired oxygen ratio. In screening for ARDS, the oxygen saturation to inspired oxygen fraction (SpO₂/FiO₂) can also be used as long as the SpO₂ is less than 97% (below the plateau on the oxyhemoglobin dissociation curve). An SpO₂/FiO₂ ratio of 315 has been shown to correlate with PaO₂/
Acute respiratory distress syndrome (ARDS)

Diagnosis

FiO₂ of 300.[39] Use of the SpO₂/FiO₂ ratio to diagnose ARDS identifies patients with similar clinical outcomes to patients diagnosed using the PaO₂/FiO₂ ratio.[40]

A chest x-ray should be performed to look for bilateral infiltrates that are consistent with pulmonary edema and not fully explained by atelectasis or pulmonary effusions. Brain natriuretic peptide (BNP) levels should be considered if heart failure is a potential cause in patients with bilateral infiltrates on radiography. BNP levels <100 picograms/mL make heart failure unlikely, whereas BNP levels >500 picograms/mL make it likely. An echocardiogram should be ordered if heart failure is still a possible diagnosis after BNP levels are available, particularly if there are no risk factors for ARDS present. If the BNP and echocardiogram are inconclusive, insertion of a pulmonary artery catheter (to estimate left ventricular end-diastolic pressure) may be helpful to differentiate heart failure from ARDS. However, routine insertion of a pulmonary artery catheter in all patients is not indicated.[41]

Blood, sputum, and urine cultures should be performed to investigate for the presence of sepsis. Viral testing should be considered in the appropriate clinical setting (e.g., influenza, SARS-CoV-2). Bronchoalveolar lavage (BAL) or endotracheal aspiration for Gram stain and cultures are also recommended in patients with ARDS due to suspected pneumonia and those without a defined predisposing condition.[42] However, bronchoscopy should be avoided in patients with suspected SARS-CoV-2 (COVID-19)-related ARDS due to high risk of provider exposure during aerosolizing procedures.[43] BAL can also be helpful for identifying other causes of acute respiratory failure with bilateral radiographic infiltrates that mimic ARDS, such as diffuse alveolar hemorrhage or acute eosinophilic pneumonia.

The best diagnostic test is an open lung biopsy. This is not routinely performed in critically ill patients because of the high risk of morbidity and mortality but it can be helpful in the setting of continued diagnostic uncertainty.[44] [45]

Serum lipase and amylase tests should be requested in patients with suspected acute pancreatitis. Both tests have similar sensitivity and specificity, but lipase levels remain elevated for longer (up to 14 days after symptom onset vs. 5 days for amylase).[46]

Computed tomography (CT) scanning of the thorax is not routinely required to diagnose or manage ARDS. It is more sensitive than a plain chest x-ray and may be helpful in some cases for diagnosing pneumonia or underlying lung disease.[47] CT scanning has shown that ARDS affects the lung parenchyma heterogeneously, with dependent portions of the lung being the most affected.[38] However, routine chest CT scanning in ARDS to assess the heterogeneity of infiltrates is not currently indicated.

[VIDEO: Radial artery puncture animated demonstration ]

[VIDEO: Femoral artery puncture animated demonstration ]

Risk factors

Strong
sepsis
• Sepsis is the most common underlying cause of ARDS, usually having a pulmonary origin.[4] The incidence of ARDS in patients with sepsis is between 6% and 7%,[16] [17] but is significantly higher in patients with septic shock.[7] Systemic activation of inflammation and coagulation is thought to lead to indirect injury to the alveolar-capillary membrane.

aspiration
• Aspiration of gastric contents is a common cause of ARDS. About one third of hospitalized patients with a witnessed aspiration event develop ARDS.[18] [19] Aspiration is thought to cause direct injury to the alveolar epithelium and alveolar-capillary membrane.

pneumonia
• Pneumonia from any source (bacterial, viral, fungal, parasitic) is a common cause of ARDS.[20] Direct injury by the pathogen and the inflammatory response to the pathogen are thought to be the responsible mechanisms.

severe trauma
• About 7% to 10% of patients with severe trauma develop ARDS.[21] Potential mechanisms include indirect injury from early hemorrhagic shock or later onset of multiple organ failure. Pulmonary contusions increase the risk of ARDS, as do long bone fractures, aspiration, and multiple transfusions of blood products.

blood transfusions
• Multiple transfusions of blood products are associated with ARDS.
• Transfusion-related acute lung injury (TRALI) can also develop with transfusion of as little as 1 unit of any plasma-containing blood product. Proposed mechanisms of TRALI include recipient neutrophil activation by donor-antibody recognition of recipient neutrophil epitopes or by biologically active lipids released from stored red blood cells.

lung transplantation
• ARDS, also known as primary graft dysfunction, occurs in 10% to 25% of patients after lung transplantation.[22] The mechanism is thought to be due to ischemia-reperfusion injury.
• Risk factors for ARDS (primary graft dysfunction) after lung transplantation include donor smoking, higher FiO₂ in the allograft at reperfusion, use of cardiopulmonary bypass, recipient body mass index, and pulmonary arterial hypertension in the donor or recipient.

pancreatitis
• Although not well studied, ARDS probably occurs in 10% to 20% of patients with severe acute pancreatitis.[23] In one study, treatment of patients with acute pancreatitis with octreotide reduced the incidence of ARDS.[24]

history of alcohol misuse
• Alcohol misuse is associated with an increased incidence of ARDS in adults.[7] [8]
• The mechanism is thought to be due to depletion of endogenous antioxidants.
Acute respiratory distress syndrome (ARDS)

**burns and smoke inhalation**
- ARDS is common after burns and smoke inhalation, with an incidence of 40% among mechanically ventilated patients with burns in one study.\[25\]

**drowning**
- ARDS is common after significant drowning episodes (grades 3 to 6).\[26\] \[27\] These patients usually recover much faster than those with other causes of ARDS.\[28\]

**e-cigarette and vaping product use**
- Emerging in the US in the summer of 2019, an outbreak of e-cigarette and vaping product-associated lung injury (EVALI) was reported among mostly young adults with a history of vaping, presenting with a clinical syndrome identical to ARDS.\[29\]
- Many cases seem to occur in patients vaping tetrahydrocannabinol (THC) products that contain vitamin E acetate.\[30\]

**Weak**

**drug overdose**
- Overdose of many common drugs (e.g., salicylates, tricyclic antidepressants, opioids, cocaine, phenothiazines) can cause ARDS, although loss of consciousness with aspiration of gastric contents may also contribute in this setting.\[31\]

**cigarette smoking**
- Smoking has been associated with an increased risk of ARDS in the setting of severe trauma,\[32\] nonpulmonary sepsis,\[33\] transfusion, and after lung transplantation.\[34\]

**History & examination factors**

**Key diagnostic factors**

**low oxygen saturation (common)**
- Low despite supplemental oxygen.

**acute respiratory failure (common)**
- Progressively worsening respiratory failure in the setting of critical illness.

**Other diagnostic factors**

**critically ill patient (common)**
- Patients developing ARDS are critically ill, often with multisystem organ failure.

**dyspnea (common)**
- Dyspnea is the most common presenting symptom.

**increased respiratory rate (common)**
- Respiratory rate >20 breaths per minute.
pulmonary crepitations (common)
  • Pulmonary crepitations on auscultation are common and typically diffuse.[26]

low lung compliance (common)
  • Measured by tidal volume/(plateau pressure minus positive end-expiratory pressure).

fever, cough, pleuritic chest pain (common)
  • These symptoms are often present, particularly if the underlying cause of ARDS is pneumonia.

frothy sputum (uncommon)
  • Presence of cough productive of frothy sputum, or frank pulmonary edema that may be blood-tinged.
## Diagnostic tests

### 1st test to order

<table>
<thead>
<tr>
<th>Test</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>chest x-ray</td>
<td>bilateral infiltrates</td>
</tr>
<tr>
<td>• New onset of bilateral opacities that is not fully explained by effusions, lobar/lung collapse, or nodules is part of the clinical diagnostic criteria for ARDS.[1] Therefore, chest x-ray is 100% sensitive.</td>
<td></td>
</tr>
<tr>
<td>• Specificity is poor because other conditions may cause bilateral pulmonary infiltrates, including cardiogenic pulmonary edema and diffuse alveolar hemorrhage. [Fig-1]</td>
<td></td>
</tr>
<tr>
<td>arterial blood gases</td>
<td>low partial oxygen pressure</td>
</tr>
<tr>
<td>• A PaO₂/FiO₂ (inspired oxygen) ratio of ≤300 on positive end-expiratory pressure (PEEP) or continuous positive airway pressure (CPAP) ≥5 cm H₂O is part of the diagnostic criteria for ARDS.[1]</td>
<td></td>
</tr>
<tr>
<td>• It is 100% sensitive, but specificity is poor because many other conditions can cause hypoxemia.</td>
<td></td>
</tr>
<tr>
<td>sputum culture</td>
<td>positive if underlying infection</td>
</tr>
<tr>
<td>• Sputum cultures are recommended to test for any possible underlying infection (as sepsis is the most common cause of ARDS).</td>
<td></td>
</tr>
<tr>
<td>blood culture</td>
<td>positive if underlying infection</td>
</tr>
<tr>
<td>• Blood cultures are recommended to test for any possible underlying infection (as sepsis is the most common cause of ARDS).</td>
<td></td>
</tr>
<tr>
<td>urine culture</td>
<td>positive if underlying infection</td>
</tr>
<tr>
<td>• A urine culture is recommended to test for any possible underlying infection (as sepsis is the most common cause of ARDS).</td>
<td></td>
</tr>
<tr>
<td>amylase and lipase</td>
<td>amylase and/or lipase 3 times the upper limit of the normal range in cases of acute pancreatitis</td>
</tr>
<tr>
<td>• Serum amylase and lipase, in conjunction with clinical assessment, can be used to help establish whether the patient has acute pancreatitis, a common cause of ARDS.[48] Both tests have similar sensitivity and specificity but lipase levels remain elevated for longer (up to 14 days after symptom onset vs. 5 days for amylase).[46] Its prolonged elevation creates a wider diagnostic window than amylase.</td>
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</table>
### Other tests to consider

<table>
<thead>
<tr>
<th>Test</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>brain natriuretic peptide (BNP)</strong></td>
<td></td>
</tr>
<tr>
<td>• BNP levels &lt;100 picograms/mL make heart failure unlikely and thus ARDS more likely.</td>
<td>BNP levels &lt;100 picograms/mL</td>
</tr>
<tr>
<td>• BNP levels &gt;500 picograms/mL make heart failure likely and thus ARDS less likely.</td>
<td></td>
</tr>
<tr>
<td>• BNP levels between 100 and 500 picograms/mL are indeterminate.</td>
<td></td>
</tr>
<tr>
<td>• BNP levels may be difficult to interpret in patients with acute or chronic kidney failure. However, BNP levels should be &lt;200 picograms/mL in patients without heart failure with an estimated glomerular filtration rate &lt;60 mL/minute.</td>
<td></td>
</tr>
<tr>
<td><em>echocardiogram</em></td>
<td>usually normal</td>
</tr>
<tr>
<td>• Abnormal left ventricular systolic or diastolic function suggests cardiogenic pulmonary edema rather than ARDS.</td>
<td></td>
</tr>
<tr>
<td>• Some patients may have both ARDS and cardiac dysfunction.</td>
<td></td>
</tr>
<tr>
<td><strong>pulmonary artery catheterization</strong></td>
<td>pulmonary artery occlusion pressure (PAOP) ≤18 mmHg</td>
</tr>
<tr>
<td>• PAOP ≤18 mmHg suggests ARDS.</td>
<td></td>
</tr>
<tr>
<td>• Pulmonary artery catheterization should not be used routinely to manage patients with ARDS.</td>
<td></td>
</tr>
<tr>
<td>• Can be used to determine whether pulmonary edema is cardiogenic if the diagnosis is still in doubt after measuring BNP levels and carrying out echocardiography.</td>
<td></td>
</tr>
<tr>
<td>• Some patients can have an increased left ventricular end-diastolic pressure superimposed on ARDS. For this reason, PAOP measurements are no longer included in the definition of ARDS.[1]</td>
<td></td>
</tr>
<tr>
<td>• In the ARDS Network FACTT trial, approximately 20% of patients had an initial PAOP &gt;18 mmHg, although elevations &gt;24 mmHg were unusual.[41]</td>
<td></td>
</tr>
<tr>
<td><strong>bronchoalveolar lavage or endotracheal aspirate</strong></td>
<td>Identification of infectious pathogens; characteristic findings of alternative diagnoses</td>
</tr>
<tr>
<td>• Recommended in some patients with suspected pneumonia and patients without a defined predisposing condition, to exclude a noninfectious parenchymal lung disease.</td>
<td>may be helpful in identifying pulmonary causes of ARDS such as pneumonia</td>
</tr>
<tr>
<td>• Avoid in patients with suspected COVID-19-related ARDS.[43]</td>
<td></td>
</tr>
<tr>
<td><strong>CT scan of the thorax</strong></td>
<td></td>
</tr>
<tr>
<td>• CT scanning of the thorax is not routinely required to diagnose or manage ARDS. A CT scan provides more information than a plain chest x-ray and may be helpful in some cases for diagnosing pneumonia or another underlying lung disease.</td>
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</tbody>
</table>
### Differential diagnosis

<table>
<thead>
<tr>
<th>Condition</th>
<th>Differentiating signs / symptoms</th>
<th>Differentiating tests</th>
</tr>
</thead>
</table>
| **Acute exacerbation of congestive heart failure** | • A history of cardiac disease, acute myocardial ischemia or infarction, or a known low ejection fraction suggests cardiogenic pulmonary edema, as do an S3 and elevated neck veins on physical examination. | • Heart failure is suggested on chest x-ray by an enlarged cardiac silhouette, a vascular pedicle width >70 mm, central infiltrates, and Kerley B lines.  
• Brain natriuretic peptide levels >500 picograms/mL also suggest cardiogenic edema.  
• An echocardiogram and measurement of the pulmonary artery occlusion pressure (PAOP) may be needed if the history and physical and lab tests do not rule out cardiogenic pulmonary edema. |
| **Bilateral pneumonia**                        | • A history of fever and cough with or without sputum production.  
• Patients may have pleuritic chest discomfort. | • Severe pneumonia with bilateral infiltrates on chest x-ray meets the radiographic criteria for ARDS.  
If patients do not have severe hypoxemia with their pneumonia (PaO₂/FiO₂ ≤300 or SpO₂/FiO₂ ≤315), they do not have ARDS. |
| **Acute interstitial pneumonia**               | • Onset is usually subacute, over days to weeks.  
• Patients are previously healthy, with no related systemic illness.  
• Some authors have termed this disease idiopathic ARDS.[42] | • Meets all the clinical criteria for ARDS.  
• Best differentiated by history. |
| **Diffuse alveolar hemorrhage**                | • Associated with bleeding from the small vessels of the airways (capillaritis) and seen in many conditions, ranging from autoimmune to mitral valve diseases.  
• Almost always a reversible form of respiratory failure, once the underlying cause is known. | • A syndrome of hypoxia with infiltrates on chest x-ray.  
The hallmark is finding sequentially bloodier aliquots of fluid during serial bronchoalveolar lavage.  
• Serologic tests to look for autoimmune diseases may help differentiate it from ARDS.[42] |
| **Acute eosinophilic pneumonia**               | • Presents as a mild to severe pneumonia in previously healthy people. | • The hallmark of this disease is increased numbers of |
### Condition

<table>
<thead>
<tr>
<th>Condition</th>
<th>Differentiating signs / symptoms</th>
<th>Differentiating tests</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute respiratory distress syndrome (ARDS)</td>
<td>- Patients have an excellent response to intravenous corticosteroids.[49]</td>
<td>eosinophils (upward of 50%) on bronchoalveolar lavage.</td>
</tr>
<tr>
<td>Hypersensitivity pneumonitis</td>
<td>- A pneumonitis after inhalation of an organic antigen.</td>
<td>- No differentiating investigations.</td>
</tr>
<tr>
<td></td>
<td>- Patients present with infiltrates and a pneumonia-like syndrome that is clinically indistinguishable from ARDS if severe.</td>
<td></td>
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<tr>
<td></td>
<td>- Differentiated from ARDS by clinical history of an inhalational allergen, usually of avian origin.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Corticosteroids may be beneficial.[42]</td>
<td></td>
</tr>
<tr>
<td>Postobstructive pulmonary edema</td>
<td>- Acute pulmonary edema after removal of an upper airway obstruction, most commonly caused by laryngospasm.</td>
<td>- No differentiating investigations.</td>
</tr>
<tr>
<td></td>
<td>- Causes an acute respiratory failure often requiring mechanical ventilation with varying levels of positive end-expiratory pressure (PEEP).</td>
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<tr>
<td></td>
<td>- The keys to differentiation are the history of upper airway obstruction, postsurgical development, and the rapid resolution of symptoms.[50]</td>
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</tbody>
</table>

### Diagnostic criteria

**Berlin modification of the American European Consensus Committee (AECC) definitions[1]**

In 2012, minor modifications to the AECC definitions of ARDS (termed the "Berlin Definition") were made. A diagnosis of ARDS can be made if the patient fulfills all of the following criteria:

- Acute onset (within 1 week of known clinical insult)
- Bilateral opacities on chest x-ray (not explained by effusions, collapse, or nodules)
- Respiratory failure not fully explained by heart failure or fluid overload (objective assessment such as echocardiogram recommended if no risk factor).

Severity of ARDS
• Mild: $\text{PaO}_2/\text{FiO}_2$ 200-300 with positive end-expiratory pressure (PEEP) or continuous positive airway pressure (CPAP) ≥5 cm H$_2$O
• Moderate: $\text{PaO}_2/\text{FiO}_2$ 100-200 with PEEP ≥5 cm H$_2$O
• Severe: $\text{PaO}_2/\text{FiO}_2$ ≤100 with PEEP ≥5 cm H$_2$O.
Step-by-step treatment approach

The goals of treatment in patients with ARDS are supportive care and a protective strategy of lung ventilation using low tidal volumes to limit end inspiratory plateau pressure.[51] If the suspected underlying cause of ARDS is infection, then the source should be identified and controlled, and antibiotics started immediately. Otherwise the immediate goals are supportive care and the prevention of complications.

The mortality of patients with ARDS is usually not due primarily to respiratory failure. Most patients die from the underlying cause of ARDS, secondary infections, other organ failures, underlying comorbidities, or the complications of prolonged hospitalization.

Oxygenation and ventilation

Although the original low tidal volume trial by the ARDS Network targeted an oxygen saturation between 88% and 95%, two subsequent clinical trials suggest that higher oxygenation targets may be associated with better clinical outcomes. A French randomized trial of oxygenation saturation target of 88% to 92% versus ≥96% in patients with ARDS was stopped early due to safety concerns, with numerically higher mortality in the low oxygen saturation target group compared with the higher saturation group at both day 28 and day 90.[52] An Australian and New Zealand trial of lower versus higher oxygenation targets in critically ill mechanically ventilated patients showed nonsignificant trends toward worse outcomes in the lower oxygen target group.[53]

Based on these findings, it seems prudent to target an oxygen saturation of ≥92%.

Occasionally patients can be managed with noninvasive ventilation,[54] but the failure rate is high and the majority will require endotracheal intubation and mechanical ventilation. Data regarding the use of high-flow oxygen via nasal cannula (HFNC) in patients with acute hypoxemic respiratory failure are inconsistent;[55] [56] the safety and efficacy of HFNC in patients with ARDS has not been studied prospectively. Ventilator-associated lung injury may be limited by the use of a low tidal volume, plateau-pressure-limited protective ventilatory strategy. This therapy has been shown to reduce mortality.[57] [58] [59]

A tidal volume of 4-8 mL/kg predicted body weight should be used to maintain an inspiratory plateau pressure <30 cm H₂O.[60] Predicted body weight for men is calculated as 50 + 0.91 × (height [cm] - 152.4), and for women is 45.5 + 0.91 × (height [cm] - 152.4).[57] If the plateau pressure is >30 cm H₂O, then tidal volume should be lowered to 5 mL/kg or as low as 4 mL/kg, if needed.

Use positive end-expiratory pressure (PEEP) titration tables

Positive end-expiratory pressure (PEEP) and FiO₂ should be titrated using established PEEP titration tables.[57] [61] The available data suggest that higher levels of PEEP are safe and may improve oxygenation in some patients.[60] [62] [63] Mortality is reduced in patients who respond with improved oxygenation.[64]

Individualized PEEP titration (rather than using a PEEP titration table), lung recruitment maneuvers in conjunction with higher PEEP levels, and PEEP titration based on radiographic classification of ARDS (as diffuse or focal) have all been evaluated in patients with ARDS.[65] [66] [67] [68] However, consistent clinical benefits have not been demonstrated with these approaches.

Managing respiratory acidosis
Acute respiratory distress syndrome (ARDS)  

Treatment

Respiratory acidosis, a common complication of low tidal volume ventilation, is treated by increasing the respiratory rate. Although it is not known what level of respiratory acidosis is harmful in patients with ARDS, permissive hypercapnia is often tolerated due to low tidal volume ventilation. However, severe hypercapnia is independently associated with higher intensive care unit (ICU) mortality.[69] Normocapnia often cannot be achieved (and should not be a goal).

Clinical guidelines recommend an arterial pH of 7.30 to 7.45 is maintained, but studies suggest patients who undergo permissive hypercapnia can tolerate a blood pH as low as 7.15. Bicarbonate infusions may be administered when the pH falls below 7.15.

[VIDEO: Tracheal intubation: animated demonstration ]

[VIDEO: Bag-valve-mask ventilation: animated demonstration ]

Prone positioning

Prone positioning can improve oxygenation in patients with ARDS and has been shown to reduce mortality in patients with severe ARDS (PaO₂/FiO₂ <150).[60] [73] [74] [75] [76] [77]

One systematic review found that reduced mortality was contingent upon patients remaining prone for at least 12 hours daily.[78] Given the potential complications of prone positioning, including facial edema, pressure sores, and dislodgement of catheters and endotracheal tubes, prone positioning should only be considered in patients with severe ARDS (PaO₂/FiO₂ <150).

Conservative intravenous fluid management

The patient’s fluid balance should be maintained as slightly negative or neutral (providing the patient is not in shock). A central line is recommended to measure the central venous pressure (CVP), with regular assessments of fluid status. The goal is to keep the CVP <4 cm H₂O. The routine use of a pulmonary artery catheter (to measure pulmonary artery occlusion pressure) is not recommended as insertion is associated with more complications than a central line.[41]

A conservative fluid strategy reduced the duration of mechanical ventilation but had no effect on mortality in a large clinical trial in patients with ARDS who were not in shock.[79] Similar results were reported in a systematic review and meta-analysis of adults and children with ARDS, sepsis, or systemic inflammatory response syndrome.[80]

[VIDEO: Central venous catheter insertion: animated demonstration ]

Antimicrobials

In patients who have an infectious cause for ARDS (e.g., pneumonia or sepsis), the prompt initiation of antimicrobials is important.[90] [91]

Empiric antibiotics targeted at the suspected underlying infection should be used as soon as possible after obtaining appropriate cultures including blood, sputum, and urine cultures. Antivirals or antifungals may be appropriate in patients with suspected or confirmed viral or fungal infections. Once culture results are available, the antimicrobial regimen can be tailored for the identified organism. There is no evidence to support the use of antibiotics in patients who have ARDS without infection.
Supportive care

Standard supportive care of critically ill patients includes prevention of deep vein thrombosis, blood glucose control,[92] prophylaxis against stress-induced gastrointestinal bleeding,[93] hemodynamic support to maintain a mean arterial pressure >60 mmHg, and transfusion of packed red blood cells in patients with hemoglobin <7 g/dL. Nutrition should be provided enterally where possible.[94] In a large randomized trial of 1000 patients with ARDS, low-dose enteral feeding for the first 5 days of ARDS had similar clinical outcomes compared with full-calorie feeding.[95] Supplemental nutrition with omega-3 fatty acids and antioxidants is not recommended.[96]

Inhaled or intravenous beta-adrenergic agonists to promote alveolar fluid clearance and resolution of pulmonary edema are not recommended.[97] [98] Neither early nor late administration of corticosteroids has been shown to improve mortality in patients with ARDS, and their routine use is not recommended.[99] [100]

Refractory hypoxemia

In patients with refractory hypoxemia despite an FiO₂ of 1.0 and high levels of PEEP, rescue therapies for oxygenation should be considered.

Neuromuscular paralysis

• Neuromuscular paralysis improves ventilator-patient synchrony and often improves oxygenation.
• Intermittent doses of paralytics can be used as effectively as a continuous intravenous infusion. If a patient is on a continuous intravenous infusion of a paralytic, train-of-four monitoring should be used to monitor the muscle fiber twitch response to the drug.
• Although one randomized clinical trial showed a 28-day mortality benefit with use of neuromuscular paralysis with cisatracurium besylate for the first 48 hours in severe ARDS (PaO₂/FiO₂ <150), a subsequent study with a similar approach to early neuromuscular blockade in ARDS was stopped early for futility.[101] [102] Given these findings, neuromuscular blockade should be reserved for patients with severe ARDS and refractory hypoxemia despite low tidal volume ventilation and adequate sedation, particularly if there is still evidence of ventilator-patient dyssynchrony.

Inhaled nitric oxide and inhaled prostacyclin

• Inhaled nitric oxide can improve oxygenation in patients with ARDS, but does not improve mortality and has been associated with acute kidney injury.[103] [104] [105] Thus, it should be used only as a rescue therapy for refractory hypoxemia.
• Inhaled prostacyclin is easier to administer than inhaled nitric oxide, and also has the potential to improve oxygenation in ARDS through better ventilation perfusion matching. However, there are currently no published large randomized controlled trials of inhaled prostacyclin; thus, it should be used cautiously and only as a rescue therapy.[106]

Extracorporeal membrane oxygenation

• Where available, extracorporeal membrane oxygenation (ECMO) should be considered (in conjunction with low tidal volume mechanical ventilation) in select patients with severe ARDS in whom standard therapies are failing (i.e., patients with profound refractory hypoxemia).[107]
• One multicenter trial showed that patients with severe ARDS randomized to transfer to a tertiary care center for consideration of ECMO (75% [n=68] of whom actually received ECMO) were more likely to survive to 6 months without disability than patients randomized to continued conventional
management (RR 0.69, 95% CI 0.05 to 0.97, P=0.03). One subsequent randomized multicenter trial (n=249) did not demonstrate significantly lower 60-day mortality in the ECMO treatment group compared with standard care (35% vs. 46%, respectively; P=0.09); however, a meta-analysis pooling data from both trials reported significantly lower 60-day mortality in the venovenous ECMO group compared with the control group (RR 0.73, 95% CI 0.58 to 0.92, P=0.008) despite a moderate risk of major bleeding in the ECMO group.

High-frequency oscillatory ventilation

- Routine use of high-frequency oscillatory ventilation (HFOV) in moderate-to-severe ARDS is not beneficial, and may be harmful.
- HFOV may have a role as a rescue therapy for patients with severe ARDS and refractory hypoxemia because the use of HFOV often improves oxygenation.

### Treatment details overview

Please note that formulations/routes and doses may differ between drug names and brands, drug formularies, or locations. Treatment recommendations are specific to patient groups: see disclaimer

<table>
<thead>
<tr>
<th>Acute</th>
<th>( summary )</th>
</tr>
</thead>
<tbody>
<tr>
<td>all patients</td>
<td>1st oxygen and ventilation</td>
</tr>
<tr>
<td></td>
<td>adjunct prone positioning</td>
</tr>
<tr>
<td></td>
<td>adjunct intravenous fluids</td>
</tr>
<tr>
<td></td>
<td>adjunct antimicrobials + identification and treatment of source of infection</td>
</tr>
<tr>
<td></td>
<td>adjunct supportive care</td>
</tr>
<tr>
<td></td>
<td>adjunct rescue therapies</td>
</tr>
</tbody>
</table>
Treatment options

Please note that formulations/routes and doses may differ between drug names and brands, drug formularies, or locations. Treatment recommendations are specific to patient groups: see disclaimer.
Acute respiratory distress syndrome (ARDS)

**Treatment**

1st oxygen and ventilation

- Although the original low tidal volume trial by the ARDS Network targeted an oxygen saturation between 88% and 95%, two subsequent clinical trials suggest that higher oxygenation targets may be associated with better clinical outcomes.\(^{[52]}\)\(^{[53]}\) Based on the findings of these studies, it seems prudent to target an oxygen saturation of ≥92%.

- Occasionally patients can be managed with noninvasive ventilation,\(^{[54]}\) but the failure rate is high and the majority will require endotracheal intubation. Data regarding the use of high-flow oxygen via nasal cannula (HFNC) in patients with acute hypoxic respiratory failure are inconsistent;\(^{[55]}\)\(^{[56]}\) the safety and efficacy of HFNC in patients with ARDS has not been studied prospectively.

- Ventilator-associated lung injury may be limited by the use of a low tidal volume, plateau-pressure-limited protective ventilatory strategy. This therapy has been shown to reduce mortality.\(^{[57]}\)\(^{[58]}\)\(^{[59]}\)

- A tidal volume of 4-8 mL/kg predicted body weight should be used to maintain an inspiratory plateau pressure <30 cm H₂O with an initial setting of 6 mL/kg.\(^{[60]}\) Predicted body weight for men is calculated as 50 + 0.91 × (height [cm] - 152.4), and for women is 45.5 + 0.91 × (height [cm] - 152.4).\(^{[57]}\) If the plateau pressure is >30 cm H₂O, then tidal volume should be lowered to 5 mL/kg or as low as 4 mL/kg, if needed.

- Positive end-expiratory pressure (PEEP) and FiO₂ should be titrated using established PEEP titration tables.\(^{[57]}\)\(^{[61]}\) The available data suggest that higher levels of PEEP are safe and may improve oxygenation in some patients.\(^{[60]}\)\(^{[62]}\)\(^{[63]}\) Mortality is reduced in patients who respond with improved oxygenation.\(^{[64]}\)

- Respiratory acidosis, a common complication of low tidal volume ventilation, is treated by increasing the respiratory rate. Although it is not known what level of respiratory acidosis is harmful in patients with ARDS, permissive hypercapnia is often tolerated due to low tidal volume ventilation. However, severe hypercapnia is independently associated with higher intensive care unit mortality.\(^{[69]}\) Normocapnia often cannot be achieved (and should not be a goal).
Acute respiratory distress syndrome (ARDS)

**TREATMENT**

Clinical guidelines recommend an arterial pH of 7.30 to 7.45 is maintained, but studies suggest patients who undergo permissive hypercapnia can tolerate a blood pH as low as 7.15. Bicarbonate infusions may be administered when the pH falls below 7.15.

»

[VIDEO: Tracheal intubation: animated demonstration]

[VIDEO: Bag-valve-mask ventilation: animated demonstration]

adjunct **prone positioning**

Treatment recommended for SOME patients in selected patient group

» Prone positioning can improve oxygenation in patients with ARDS and has been shown to reduce mortality in patients with severe ARDS (PaO₂/fraction of inspired oxygen [FiO₂] <150).[^60] [^73] [^74] [^75] [^76] [^77] One systematic review found that reduced mortality was contingent upon patients remaining prone for at least 12 hours daily.[^78] Given the potential complications of prone positioning, including facial edema, pressure sores, and dislodgement of catheters and endotracheal tubes, prone positioning should only be considered in patients with severe ARDS (PaO₂/FiO₂ <150).

adjunct **intravenous fluids**

Treatment recommended for SOME patients in selected patient group

» The patient's fluid balance should be maintained as slightly negative or neutral (providing the patient is not in shock). A central line is recommended to measure the central venous pressure (CVP), with regular assessments of fluid status. The goal is to keep the CVP <4 cm H₂O. The routine use of a pulmonary artery catheter (to measure pulmonary artery occlusion pressure) is not recommended as insertion is associated with more complications than a central line.[^41]

» A conservative fluid strategy reduced the duration of mechanical ventilation but had no effect on mortality in a large clinical trial in patients with ARDS who were not in shock.[^79] Similar results were reported in a systematic review and meta-analysis of adults
Acute respiratory distress syndrome (ARDS)

**Treatment**

**Acute**

and children with ARDS, sepsis, or systemic inflammatory response syndrome.[80]

**adjunct** antimicrobials + identification and treatment of source of infection

Treatment recommended for SOME patients in selected patient group

» In patients who have an infectious cause for ARDS (e.g., pneumonia or sepsis), the prompt initiation of antimicrobials is important.[90] [91] Empiric antibiotics targeted at the suspected underlying infection should be used as soon as possible after obtaining appropriate cultures including blood, sputum, and urine cultures. Antivirals or antifungals may be appropriate in patients with suspected or confirmed viral or fungal infections. Once culture results are available, the antimicrobial regimen can be tailored for the identified organism. There is no evidence to support the use of antibiotics in patients who have ARDS without infection.

**adjunct** supportive care

Treatment recommended for SOME patients in selected patient group

» Standard supportive care of critically ill patients includes prevention of deep vein thrombosis, blood glucose control,[92] prophylaxis against stress-induced gastrointestinal bleeding,[93] hemodynamic support to maintain a mean arterial pressure >60 mmHg, and transfusion of packed red blood cells in patients with hemoglobin <7 g/dL. Nutrition should be provided enterally where possible.[94] In a large randomized trial of 1000 patients with ARDS, low-dose enteral feeding for the first 5 days of ARDS had similar clinical outcomes compared with full-calorie feeding.[95] Supplemental nutrition with omega-3 fatty acids and antioxidants is not recommended.[96]

» Inhaled or intravenous beta-adrenergic agonists to promote alveolar fluid clearance and resolution of pulmonary edema are not recommended.[97] [98] Neither early nor late administration of corticosteroids has been shown to improve mortality in patients with ARDS, and their routine use is not recommended.[99] [100]

**adjunct** rescue therapies

Treatment recommended for SOME patients in selected patient group

» In patients with refractory hypoxemia despite a fraction of inspired oxygen (FiO₂) of 1.0 and high levels of positive end-expiratory pressure
Acute respiratory distress syndrome (ARDS)

**Treatment**

**Acute**

(PEEP), rescue therapies for oxygenation should be considered.

» Neuromuscular paralysis improves ventilator-patient synchrony and often improves oxygenation. Intermittent doses of paralytics can be used as effectively as a continuous intravenous infusion. If a patient is on a continuous intravenous infusion of a paralytic, train-of-four monitoring should be used to monitor the muscle fiber twitch response to the drug. Given findings from randomized controlled trials, neuromuscular blockade should be reserved for patients with severe ARDS and refractory hypoxemia despite low tidal volume ventilation and adequate sedation, particularly if there is still evidence of ventilator-patient dyssynchrony.[101] [102]

» Inhaled nitric oxide can improve oxygenation in patients with ARDS, but does not improve mortality and has been associated with acute kidney injury.[103] [104] [105] Thus, it should be used only as a rescue therapy for refractory hypoxemia. Inhaled prostacyclin is easier to administer than inhaled nitric oxide, and also has the potential to improve oxygenation in ARDS through better ventilation perfusion matching. However, there are currently no published large randomized controlled trials of inhaled prostacyclin; thus, it should be used cautiously and only as a rescue therapy.[106]

» Where available, extracorporeal membrane oxygenation (ECMO) should be considered (in conjunction with low tidal volume mechanical ventilation) in select patients with severe ARDS in whom standard therapies are failing (i.e., patients with profound refractory hypoxemia).[107] In patients with severe acute ARDS, venovenous ECMO is associated with reduced 60-day mortality compared with conventional mechanical ventilation, despite a moderate risk of major bleeding.[110]

» Routine use of high-frequency oscillatory ventilation (HFOV) in moderate-to-severe ARDS is not beneficial,[111] [112] [113] and may be harmful.[114] [115] However, HFOV may still have a role as a rescue therapy for patients with severe ARDS and refractory hypoxemia, because the use of HFOV often improves oxygenation.
Emerging

Early corticosteroid administration

An open-label randomized controlled study of patients with moderate-to-severe ARDS found that early dexamethasone resulted in a substantial increase in ventilator-free days (4.8 days), and a 15% reduction in mortality, compared with placebo.[116] These findings need to be validated and must be considered cautiously given serious concerns about the safety of high-dose glucocorticoids in critically ill patients, including risk of nosocomial infections and critical illness polyneuropathy/myopathy.
Recommendations

Monitoring

No long-term monitoring is needed in patients who survive ARDS, unless they continue to have shortness of breath. In that instance, yearly pulmonary function tests are used to monitor their course.
## Complications

<table>
<thead>
<tr>
<th>Complications</th>
<th>Timeframe</th>
<th>Likelihood</th>
</tr>
</thead>
<tbody>
<tr>
<td>death</td>
<td>short term</td>
<td>medium</td>
</tr>
<tr>
<td>Mortality for patients with ARDS is estimated at 30% to 50%. [35] [117]</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ventilator-associated pneumonia</td>
<td>short term</td>
<td>medium</td>
</tr>
<tr>
<td>Can develop in any patient who requires mechanical ventilation for more than 48 hours.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Signs and symptoms include a new fever, elevated white blood cell count, new infiltrate on chest x-ray, increased or changing pulmonary secretions, and hypotension.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>multiple organ failure</td>
<td>short term</td>
<td>medium</td>
</tr>
<tr>
<td>In addition to respiratory failure, the most common manifestations in patients with ARDS are renal failure, shock, acute delirium, or coma. Less common are hepatic and hematologic failure.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Treatment includes supportive therapy as well as specific interventions for each organ: mechanical ventilation for respiratory failure, dialysis for renal failure, and vasopressors for hypotension.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>pneumothorax</td>
<td>short term</td>
<td>low</td>
</tr>
<tr>
<td>Most often a complication due to pulmonary barotrauma. Barotrauma occurred in 13% of patients enrolled in the ARDS Network low tidal volume trial and was associated with higher levels of positive end-expiratory pressure (PEEP). [123]</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Signs and symptoms include tracheal deviation, sudden worsening hypoxemia, high peak and plateau pressures on the ventilator, hypotension, and cardiovascular collapse.</td>
<td></td>
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<tr>
<td>Chest x-ray can confirm the presence of a pneumothorax. Treated with insertion of a chest tube.</td>
<td></td>
<td></td>
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<tr>
<td>persistent dyspnea</td>
<td>variable</td>
<td>high</td>
</tr>
<tr>
<td>Persistent dyspnea is particularly present during exercise. A majority of patients who survive ARDS have a mild to moderate decrease in carbon monoxide diffusion in the lung, [121] but steady improvement is seen in the first year. [122]</td>
<td></td>
<td></td>
</tr>
<tr>
<td>abnormal lung function</td>
<td>variable</td>
<td>medium</td>
</tr>
<tr>
<td>In one study, 40% of patients had either restriction or obstruction 1 year after ARDS, [121] but similar abnormalities were not observed in another study. [122]</td>
<td></td>
<td></td>
</tr>
<tr>
<td>reduced quality of life</td>
<td>variable</td>
<td>medium</td>
</tr>
<tr>
<td>Studies looking at quality-of-life scores found a reduction in quality of life for at least the first year after surviving ARDS. [121] [122]</td>
<td></td>
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</tr>
</tbody>
</table>
Prognosis

Mortality in patients who develop ARDS is 30% to 50%.\cite{35} \cite{117} Death is most often due to multiple organ failure rather than purely to respiratory failure.\cite{118} Low tidal volume ventilation reduced in-hospital mortality from 40% to 31% in the 2000 ARDS Network trial.\cite{57} Being of a younger age may also increase the chances of survival.\cite{119} Patients who do survive their illness usually have some residual decrease in lung function, although it may not always cause symptoms.\cite{120} \cite{121} Muscle weakness, neuropathies, joint disorders, and chronic pain are also common in survivors of ARDS at 1 year.\cite{122}
Treatment guidelines

International

Mechanical ventilation in adult patients with acute respiratory distress syndrome [60]

Published by: American Thoracic Society; European Society of Intensive Care Medicine; Society of Critical Care Medicine

Guidelines on the management of acute respiratory distress syndrome [107]

Published by: The Faculty of Intensive Care Medicine; Intensive Care Society

Last published: 2017

Last published: 2018
Key articles


References


Acute respiratory distress syndrome (ARDS)

References


Acute respiratory distress syndrome (ARDS)

References


Acute respiratory distress syndrome (ARDS)  References


REFERENCES


85. Abbott Northwestern Hospital Internal Medicine Residency. Internal jugular central venous line. 2015 [internet publication]. Full text


100. Steinberg KP, Hudson LD, Goodman RB, et al; National Heart, Lung, and Blood Institute Acute Respiratory Distress Syndrome (ARDS) Clinical Trials Network. Efficacy and safety of corticosteroids


Images

Figure 1: Chest x-ray image of bilateral infiltrates in a patient with ARDS

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DISCLOSURES: LW has received advisory board fees from Bayer, Quark, and CSL Behring and contractual research support to her institution from Global Blood Therapeutics, Boehringer Ingelheim, and CSL Behring.

// Acknowledgements:

Dr Lorraine Ware would like to gratefully acknowledge Dr Richard Fremont, a previous contributor to this topic.
DISCLOSURES: RF declares that he has no competing interests.

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