JOURNAL OF INTERNATIONAL CRISIS AND RISK COMMUNICATION RESEARCH
ISSN: 2576-0017
2024, VOL 7, NO S9

A guide for nurses and respiratory therapists for ARDS Treatment: Bridging the Gap Between Theory and Practice

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Abstract

Acute respiratory distress syndrome (ARDS) remains a significant challenge in intensive care units despite advancements in management. This review provides a comprehensive overview of ARDS, focusing on fluid management strategies. The Berlin definition categorizes ARDS severity based on the degree of hypoxemia. Sepsis is the most common etiology, with other causes including pneumonia, shock, surgery, and trauma. Pathophysiologically, ARDS is characterized by increased alveolar-capillary permeability, leading to pulmonary edema and impaired gas exchange. Management involves treating the underlying cause, nutritional support, thromboprophylaxis, and lung-protective ventilation strategies. Low tidal volumes (6 mL/kg) and driving pressure are associated with improved survival. Prone positioning is recommended for severe ARDS, while the roles of inhaled nitric oxide, corticosteroids, and extracorporeal membrane oxygenation require further research. Conservative fluid management is favored over liberal strategies, as it improves oxygenation and lung function without impacting mortality. Central venous pressure and pulmonary artery wedge pressure have limitations in guiding fluid therapy. Pulse pressure variation and lung ultrasound are emerging tools for assessing fluid responsiveness and extravascular lung water, respectively. Albumin offers no advantage over crystalloids, and hydroxyethyl starch is not recommended. A restrictive transfusion strategy is appropriate in ARDS patients. Prognosis depends on the

underlying cause, with sepsis-related ARDS having the lowest survival. Recent studies suggest declining mortality rates, likely due to advancements in ventilation strategies and supportive care. Further research is needed to refine management approaches and identify new therapeutic avenues for ARDS.
Keywords: ARDS, acute respiratory distress syndrome, treatment, nurses, respiratory therapists.

Introduction

Since its first characterization in 1967 by Ashbaugh and colleagues, acute respiratory distress syndrome (ARDS) remains a significant and frequent challenge in contemporary intensive care units. Despite substantial advancements in its management, mortality and morbidity rates continue to be concerning, with the severe form demonstrating a mortality rate of 45%. The need for innovative therapeutic modalities necessitates a deeper understanding of the underlying pathophysiology, as well as refinement of the tools currently available for treatment. Fluid therapy is among the interventions employed in managing ARDS patients. However, it represents a distinct dilemma, functioning as a double-edged sword. On one side, fluid therapy is often essential in ARDS patients, especially in the presence of hypoperfusion. On the other side, fluid infusion and the resultant volume overload can aggravate pulmonary edema, exacerbating the existing gas exchange abnormalities.

This review provides a comprehensive overview of ARDS management, with an emphasis on fluid management. It examines the underlying pathophysiological mechanisms influencing fluid dynamics across the alveolar-capillary membrane, alongside major clinical trials addressing optimal fluid therapy strategies in this syndrome.

Definition And Clinical Picture

Following two decades of reliance on the 1994 American/European Consensus Conference definition of ARDS, a revised definition, known as the Berlin definition, was introduced. This updated definition specifies that ARDS must develop within one week of an identifiable clinical insult, accompanied by bilateral radiographic opacities. Importantly, these pulmonary opacities must not be solely attributable to systolic heart failure or fluid overload. The severity of gas exchange impairment, measured at a PEEP $\geq$ 5 cm H_2O, is used to categorize ARDS into mild ($200 < PaO_2/FiO_2 \leq 300$), moderate ($100 < PaO_2/FiO_2 \leq 200$), or severe ($PaO_2/FiO_2 \leq 100$). Noninvasive ventilation is deemed feasible in the mild category. The term Acute Lung Injury (ALI), previously used to describe the mild form of ARDS, has been removed from this new definition.

Estimates of the actual incidence of ARDS have varied over time, partly due to evolving definitions and inconsistent application. Using the 1994 criteria, a Scandinavian cohort study identified an incidence of 17.9 cases of ALI and 13.5 cases of ARDS per 100,000 individuals aged 15 years or older. A population-based cohort study conducted in Washington estimated the occurrence of ARDS at 64 cases per 100,000 person-years, while ALI was observed at 86 cases per 100,000 person-years. The incidence of ARDS and ALI may be decreasing, potentially due to a decline in hospital-acquired cases, although this trend requires further validation. Improved management of precipitating factors such as sepsis, along with more judicious ventilatory strategies designed to minimize barotrauma and volutrauma, may also contribute to this observed reduction. Furthermore, ARDS is likely underreported in resource-limited settings, where access to arterial blood gas analysis and chest radiography is often inadequate (Thompson et al., 2017).

Sepsis remains the most common etiology of ARDS, with approximately 40% of sepsis cases progressing to ARDS. Other contributing factors include pneumonia, shock, major surgical interventions, and trauma.

Clinically, ARDS is marked by the rapid onset of hypoxemic respiratory failure in the context of an underlying predisposing condition. The severity of hypoxemia often necessitates invasive

mechanical ventilation. Bilateral radiographic infiltrates are typically present and are frequently indistinguishable from those seen in cardiogenic pulmonary edema. Computed tomography findings usually reveal alveolar consolidation and filling, predominantly in dependent lung zones. In advanced stages, interstitial opacities and bullae formation may emerge. Complications associated with ARDS include pulmonary hypertension, right ventricular failure, and pneumothorax.

Pathophysiology

Fluid movement between the capillaries and the interstitial space is governed by hydrostatic and oncotic pressures across the capillary wall, as described by Starling's equation:

$Q_x = K_x[(P_x - P_i) - \sigma(\pi_x - \pi_i)]$

Here, Q_x represents the flow of fluid across the membrane, K_x denotes the capillary filtration coefficient, P_x is the capillary hydrostatic pressure, P_i is the interstitial hydrostatic pressure, σ refers to the oncotic reflection coefficient, π_x represents the capillary colloid osmotic pressure, and π_i denotes the interstitial colloid osmotic pressure.

These pressures determine both the direction and the quantity of fluid movement. Typically, net forces favor ultrafiltration at the arteriolar end of pulmonary capillaries, while reabsorption predominantly occurs at the venular end. The lymphatic system removes any fluid that accumulates in the interstitium. Additionally, the tight junctions between alveolar epithelial cells serve as a barrier, preventing alveolar flooding.

An increase in capillary hydrostatic pressure or a disruption in the integrity of the alveolar–capillary membrane, leading to increased permeability, can result in interstitial and alveolar flooding. ARDS exemplifies the latter scenario, characterized by alveolar edema or flooding occurring in the presence of normal capillary hydrostatic pressure. Injury to the flat type I cells, which comprise approximately 90% of the alveolar surface area, is a hallmark of ARDS. The more resilient cuboidal type II cells eventually differentiate into type I cells, restoring normal alveolar architecture if ARDS resolves. Damage to the alveolar-capillary membrane leads to significant leakage of fluid and plasma proteins into the alveolar space, resulting in the formation of hyaline membranes. Alveolar fluid clearance is also impaired.

Activated inflammatory cells, predominantly neutrophils and macrophages, accumulate in the interstitial space. Proinflammatory cytokines such as Interleukin (IL)-1β, IL-8, and tumor necrosis factor (TNF)-α are released into the lungs, contributing to microvascular injury, the cellular inflammatory response, and the extrapulmonary organ failures observed in ARDS.

Surfactant activity and composition are disrupted, leading to increased surface tension, alveolar collapse, decreased lung compliance, impaired gas exchange, and elevated pulmonary arterial pressures.

By the seventh day of ARDS, interstitial inflammation and fibrosis become the predominant pathological findings. In a subset of patients, pulmonary fibrosis develops, which is closely associated with mortality in advanced ARDS.

General Management

Prompt recognition and treatment of the underlying cause, such as infection, is critical. Moreover, adequate nutritional support and prophylaxis against thromboembolic complications should be incorporated into the management plan (Sweeney & McAuley, 2016).

Mechanical Ventilation

Most patients with ARDS require endotracheal intubation and mechanical ventilation to address the severe hypoxemia associated with the condition. Special consideration must be given to tidal volume to prevent ventilator-induced lung injury.

A pivotal ARDSnet study involving 861 patients compared tidal volumes of 12 mL/kg versus 6 mL/kg of predicted body weight (with plateau pressures ≤50 vs. ≤30 cm H_2O). This study established the use of low tidal volume (6 mL/kg) as the standard of care, demonstrating lower

mortality in this group (31.0% vs. 39.8%, p = 0.007). In a porcine model of pulmonary edema, a tidal volume of 6 mL/kg was associated with lower extravascular lung water (EVLW) measured using the double indicator method, compared with 12 mL/kg.

A meta-analysis of nine randomized trials by Amato et al. revealed that driving pressure, defined as the ratio of tidal volume to respiratory system compliance, was strongly correlated with survival in ARDS, even among patients receiving protective ventilation (Amato et al., 2015). These findings suggest driving pressure may represent a more effective therapeutic target in future research.

While the use of low tidal volume may lead to CO_2 retention and respiratory acidosis, this permissive hypercapnia can typically be managed by increasing the respiratory rate.

Positive End Expiratory Pressure (PEEP) is primarily utilized to improve oxygenation by increasing functional residual capacity and preventing alveolar and airway collapse, thereby enhancing ventilation-perfusion (V/Q) matching. The impact of PEEP on EVLW, as measured by transpulmonary thermodilution, appears to be minimal (Jozwiak et al., 2015). However, PEEP may cause side effects, including circulatory depression and barotrauma.

Multiple trials have assessed whether higher PEEP levels improve outcomes, but no significant mortality differences were observed. This indicates that the lowest PEEP level sufficient to maintain acceptable oxygenation and airway pressures may be optimal. Nonetheless, the 2016 Surviving Sepsis Campaign guidelines suggest higher PEEP levels over lower levels for adults with sepsis-induced moderate to severe ARDS (weak recommendation, moderate-quality evidence) (Rhodes et al., 2017).

In contrast, a recent trial found worse outcomes when using a strategy involving lung recruitment and PEEP titration based on respiratory system compliance compared with a strategy of low PEEP (Writing Group for the Alveolar Recruitment for Acute Respiratory Distress Syndrome Trial (ART) Investigators et al., 2017).

Prone Position

The prone position has been shown to improve oxygenation in cases of moderate to severe ARDS. The mechanisms behind this improvement are not entirely understood but likely include an increase in lung volume, reduced atelectasis and shunt fraction, improved V/Q matching, and the alleviation of heart weight effects on the left lung. Interestingly, a small study noted an increase in the EVLW index measured by transpulmonary thermodilution after proning (12.7 ± 4.7 vs. 14.8 ± 7.8 mL/kg), though the clinical relevance of this increase was negligible.

While earlier studies failed to demonstrate a mortality benefit with prone positioning, a more recent trial in patients with severe ARDS (PaO_2/FiO_2 < 150 mm Hg) showed reduced 28-day mortality when the prone position was maintained for at least 16 consecutive hours (Guérin et al., 2013). As a result, current evidence supports the use of prone positioning in patients with severe ARDS, including those with sepsis-induced ARDS and PaO_2/FiO_2 < 150 mm Hg (Rhodes et al., 2017).

Other Supportive Therapies

Inhaled Nitric Oxide (iNO), a potent vasodilator, enhances ventilation–perfusion mismatching and leads to dose-dependent improvements in oxygenation. In a small animal model of acute lung injury, iNO demonstrated a reduction in edema formation associated with fluid resuscitation. Clinical trials evaluating its use in ARDS consistently showed improved oxygenation but no significant impact on mortality. As with any salvage therapy, iNO may be considered for patients with refractory hypoxemia, with awareness of its potential side effects and the likelihood of its benefits being transient.

Iloprost, a stable prostacyclin analog, has been shown to improve gas exchange in patients with ARDS and pulmonary hypertension (Sawheny et al., 2013). However, unlike iNO, iloprost did not reduce lung edema in an ovine model of lung injury.

The use of corticosteroids in ARDS remains controversial. Over recent decades, studies have reported mixed results regarding their effect on mortality. Current guidelines from the Society of Critical Care Medicine and the European Society of Intensive Care Medicine recommend corticosteroids in patients with early moderate to severe ARDS ($PaO_2/FiO_2 < 200$ mm Hg and within 14 days of onset) as a conditional recommendation based on moderate-quality evidence (Annane et al., 2017). The potential benefits must be weighed against risks such as infections and neuromuscular weakness.

Extracorporeal Membrane Oxygenation (ECMO) provides extracorporeal oxygenation and carbon dioxide removal. It is a salvage option for patients with severe ARDS; however, robust controlled trials demonstrating a clear survival benefit are lacking. A recent international trial found no significant reduction in 60-day mortality with ECMO compared to conventional mechanical ventilation strategies, which included ECMO as salvage therapy (Combes et al., 2018). Additional research is required to establish ECMO's role in ARDS management.

High-frequency oscillatory ventilation, which delivers small tidal volumes at high frequencies, has been evaluated in ARDS. However, a trial in moderate-to-severe ARDS found that early use of this technique did not reduce and may even increase in-hospital mortality, leading to its non-recommendation in ARDS (Ferguson et al., 2013).

Neuromuscular blockade has been shown to improve oxygenation in ARDS. A 2010 French trial demonstrated that early administration of Cisatracurium in severe ARDS (within 48 hours of onset) improved adjusted 90-day survival without increasing the risk of muscle weakness. Further studies are needed to validate this finding and assess whether similar benefits are seen with other neuromuscular blocking agents.

Edema clearance in ARDS relies on active sodium transport, with water following the sodium gradient. In hydrostatic pulmonary edema, fluid clearance is typically maximal or submaximal in most patients compared to ARDS (Neamu & Martin, 2013). Enhancing alveolar fluid removal in ARDS is an appealing therapeutic approach, potentially upregulated through catecholamine-dependent and -independent mechanisms, including beta-2 adrenergic agonists. A small randomized trial demonstrated that intravenous salbutamol reduced lung water and plateau pressure in patients with ALI/ARDS. However, a subsequent ARDS Network trial found no clinical benefit of aerosolized albuterol (5 mg every 4 hours for up to 10 days) compared to saline placebo. As a result, routine use of beta-2 agonists solely for alveolar edema clearance in ARDS is not recommended.

Fluid Management and Responsiveness

Fluid management in ARDS presents a complex challenge. Many patients require fluid resuscitation, particularly in the context of sepsis or septic shock. However, given the pathophysiology of normal-pressure pulmonary edema, fluid administration can increase left atrial and pulmonary venous pressures, exacerbate alveolar flooding, reduce the PaO_2/FiO_2 ratio, and necessitate careful monitoring of gas exchange and hemodynamic parameters. Conversely, induced hypotension, which reduces cardiac output and pulmonary blood flow (as seen in hemorrhagic shock), can increase alveolar and physiological dead space, leading to impaired gas exchange and elevated $PaCO_2$. Moreover, therapies or conditions that lower pulmonary arterial pressure, such as vasodilator treatment for pulmonary hypertension, may increase intrapulmonary shunting and worsen hypoxemia. This underscores the importance of maintaining adequate volume status in these patients.

Determining intravascular fluid status and evaluating the contribution of impaired cardiac function to oxygenation deficits is clinically challenging. Chest X-rays and blood gas measurements are limited in their ability to quantify pulmonary edema (Sakka, 2013). Balancing tissue perfusion and oxygenation remains a significant challenge, and identifying the optimal intravascular volume–pressure with the best risk–benefit ratio is often difficult.

Optimal Volume Status

The optimal intravascular volume strikes a balance between ensuring adequate tissue perfusion and minimizing alveolar flooding. Theoretically, maintaining a "dry" state in ARDS patients may improve pulmonary function, including gas exchange, potentially resulting in better outcomes. Retrospective studies provide some support for this hypothesis. Alsous et al. demonstrated that in septic shock patients, achieving at least one day of negative fluid balance within the first three days was associated with improved survival, even after adjusting for age, APACHE II scores, SOFA scores on days 1 and 3, and the requirement for mechanical ventilation. Notably, five of these patients developed ARDS/ALI by day 3. Similarly, Humphrey reported that reducing pulmonary artery wedge pressure (PAWP) was linked to increased survival in ARDS. Using logistic regression, Simmons et al. identified a correlation between weight loss, negative fluid balance, and survival in ARDS. Another observational study utilizing prospectively collected data revealed that excessive fluid administration in trauma-related ARDS patients was independently associated with higher mortality. This study accounted for variables such as demographics, severity scores, injury-admission delay times, first 24-hour transfusion, and septic and organ failure complications. However, these studies were neither prospectively randomized nor based on a consistent definition of ARDS.

A large prospective, randomized controlled trial compared conservative and liberal fluid management strategies in 1,000 patients with ALI. Patients were randomized to either pulmonary artery catheter (PAC) or central venous catheter (CVC) groups, with management guided by four variables: central venous pressure (CVP) or PAWP (depending on catheter type), and the presence or absence of shock, oliguria, or ineffective circulation. Fluid administration, diuretics, or inotropic agents were used to meet target variables. Over the first seven days, the mean cumulative fluid balance was –136 ± 491 mL in the conservative group and 6992 ± 502 mL in the liberal group ($p < 0.001$). Compared to the liberal strategy, the conservative strategy resulted in improved oxygenation index, lung injury score, and ventilator-free days. However, there were no significant differences in shock incidence, dialysis use, or 60-day mortality (25.5% in the conservative group vs. 28.4% in the liberal group, $p = 0.30$). Additionally, vasopressor use did not differ significantly between the groups. These findings support the adoption of a conservative fluid management approach in ALI patients.

Assessing Volume Status/Fluid Responsiveness

CVP/PAWP

Central venous pressure (CVP) and pulmonary artery wedge pressure (PAWP) have traditionally been utilized to guide fluid management in various clinical settings, including ARDS. However, it is important to note that the relationship between these pressures and cardiac preload is highly variable (Hu et al., 2014). Although the measurement of these pressures could theoretically improve outcomes, there is a poor correlation between CVP and blood volume, and CVP fails to reliably predict the hemodynamic response to fluid challenges. Wheeler et al. compared the efficacy and risks of CVCs and PACs in ALI management. Their findings indicated that PAC-guided therapy neither improved survival nor organ function and was associated with a higher incidence of complications compared to CVC-guided therapy. Complications of PAC use included arrhythmias, air embolisms, catheter malfunction, and insertion-site bleeding. Consequently, PACs should not be routinely employed in the management of ALI.

Pulse Pressure Variation

Pulse pressure (PP), defined as the difference between systolic and diastolic pressure, reflects ventricular stroke volume. Pulse pressure variation (PPV) is the difference between maximal (PPmax) and minimal (PPmin) pulse pressure values divided by the mean pulse pressure over a single respiratory cycle. Accurate measurement requires a tidal volume ≥ 8 mL/kg, sinus rhythm, and the absence of spontaneous ventilator triggering. The variation in PP during

positive pressure ventilation is thought to reflect the patient's position on the Frank–Starling curve, with fluid-responsive patients located on the steep portion of the curve. Fluid-responsive patients typically exhibit significant PPV (>10–12%) during mechanical ventilation (Teboul & Monnet, 2013). However, the utility of PPV in ARDS patients receiving protective ventilation is limited, partly due to insufficient pleural pressure changes. Additional factors that restrict its effectiveness include arrhythmias and spontaneous respiratory efforts.

Lung/Central Vascular Ultrasound

Lung ultrasound is a relatively recent modality used to evaluate extravascular lung water (EVLW) by identifying reverberation artifacts, or B lines, originating from the pleural line. These B lines are thought to result from interlobular septal thickening due to fluid accumulation. However, the presence of B lines has not been found to predict pulmonary artery wedge pressure (PAWP), which is expected as pulmonary edema can stem from both cardiogenic and non-cardiogenic causes. Conversely, a horizontal reverberation pattern (A lines) has been shown to predict low PAWP (≤18 mmHg) with a sensitivity of 50% and a specificity of 93%.

A B-line score (BLS) developed to estimate EVLW was found to correlate with radiological EVLW scores in patients admitted to medical and cardiac intensive care units. In patients undergoing hemodialysis, BLS measured one hour post-dialysis decreased by 2.7 B-lines for every 500 mL of fluid removed ($p = 0.02$), suggesting that lung ultrasound may be a useful tool for monitoring the resolution of pulmonary edema caused by hypervolemia. Additionally, in patients with high-altitude pulmonary edema, higher BLS scores were observed compared to control subjects (31 ± 11 vs. 0.86 ± 0.83), with oxygen saturation decreasing by 0.67% for every one-point increase in the BLS ($p < 0.001$ for both comparisons).

In ARDS patients, Zhao et al. developed a lung ultrasound score (LUS) using a 12-region method (anterior, lateral, and posterior; upper and lower; right and left chest wall), with higher scores indicating reduced aeration or pulmonary consolidation (Zhao et al., 2015). In this study, non-survivors had significantly higher LUS on day 1 compared to survivors (20 ± 5 vs. 15 ± 5, $p = 0.022$). Correlations were also identified between LUS and EVLW indices, lung injury score, and PaO2/FiO2 ratio ($r^2 = 0.906, 0.361, 0.472$; $p < 0.01$). In another study involving 32 patients with septic shock and ARDS, Caltabeloti et al. demonstrated that lung ultrasound could detect aeration changes in response to early fluid loading, suggesting its role in preventing excessive fluid administration in these patients (Caltabeloti et al., 2014). In a study on vascular ultrasound, Allyn et al. found that inferior vena cava (IVC) diameter, its respiratory cycle variations, and IVC distensibility did not predict tolerance to negative fluid balance (assessed by hypotension, acute kidney injury, or fluid expansion requirements) in 45 ARDS/ALI patients.

Overall, lung ultrasound may serve as a practical bedside tool for detecting EVLW and assessing septal edema during fluid removal or administration. It may also have potential for predicting ARDS-related mortality, but further studies are required to establish its role in guiding therapy for this patient population.

Extravascular Lung Water (EVLW)

The measurement of EVLW has been proposed as a strategy to guide fluid management in ARDS patients, with evidence suggesting that maximal alveolar fluid clearance may improve clinical outcomes. Compared to patients with cardiac pulmonary edema, those with ARDS typically exhibit higher EVLW and lower PAWP. A normal EVLW index is defined as <7 mL/kg of predicted body weight, with 10 mL/kg considered the upper limit of normal. Jozwiak et al. identified EVLW and pulmonary vascular permeability indices (measured via the thermodilution curve using the PiCCO device, Pulsion Medical Systems) as independent risk factors for 28-day mortality in ARDS patients. However, other studies have found no

distinction in survival between patients with higher or lower EVLW measurements. Furthermore, EVLW did not correlate with oxygenation, suggesting that while pulmonary edema is present in these patients, it may not be the primary cause of hypoxemia. A recent small study by Hu found that using EVLW to guide fluid management reduced mechanical ventilation duration and ICU stays but did not affect survival when compared to PAWP-based management (Hu et al., 2014). Nevertheless, bedside interpretation of EVLW measurements remains challenging, and larger prospective studies are needed to define its role in ARDS management.

Fluid Type

There is limited research regarding the optimal fluid type for ARDS patients. In the ARDS Network trial comparing conservative and liberal fluid strategies, clinicians were allowed to select the type of fluid—such as isotonic crystalloids, albumin, or blood products—while adhering to specified fluid volumes. In a large ICU study, 6% hydroxyethyl starch (HES) was compared with 0.9% saline for resuscitation. Although the study did not specify the proportion of ARDS patients, no difference in 90-day mortality was observed. However, HES use was associated with an increased need for renal replacement therapy. A meta-analysis of critically ill patients also linked HES to significantly higher risks of mortality and acute kidney injury, leading to its recommendation against use in ARDS patients.

In an open-label trial, patients with severe sepsis were randomized to receive either 20% albumin and crystalloids or crystalloids alone (Caironi et al., 2014). While the majority of patients (approximately 80%) required mechanical ventilation, ARDS-specific data were not provided. Patients in the albumin group showed higher mean arterial pressure and lower net fluid balance, but 28- and 90-day mortality rates were similar. Another double-blind trial comparing 4% albumin and normal saline in a heterogeneous ICU population also found no significant differences in mortality, including in a pre-specified ARDS subgroup (RR 0.93, 0.61–1.41). A large meta-analysis also showed no evidence that colloids reduced mortality compared to crystalloids in trauma, burns, or surgical patients.

Thus, current evidence suggests that albumin offers no advantage over crystalloids, and HES should be avoided in ARDS patients.

Blood Transfusion

Blood transfusion aimed at increasing oxygen delivery was investigated in a trial where septic shock patients were randomized to receive transfusions when hemoglobin levels were ≤7 g/dL or ≤9 g/dL (Holst et al., 2014). Approximately 70% of these patients required mechanical ventilation, although ARDS-specific data were not provided. Mortality at 90 days was similar between the two groups, supporting a conservative transfusion strategy in ARDS patients with underlying septic shock. Another study showed that a restrictive transfusion strategy is at least as effective as a liberal one—possibly superior—except in cases of acute myocardial infarction or unstable angina. Based on this evidence, a conservative transfusion approach appears appropriate for ARDS patients.

Prognosis and Outcome

The prognosis of ARDS largely depends on the underlying cause of lung injury. Survival to home discharge is lowest in sepsis-related ARDS and highest in trauma-related ARDS. Additional predictors of mortality include age, the severity of hypoxemia, and APACHE scores. Historically, ARDS mortality rates ranged from 40% to 60%, with most deaths attributed to sepsis and non-respiratory organ dysfunction. Recent studies indicate that mortality rates may be declining. This improvement is likely due to advancements in low tidal volume ventilation, improved supportive care, and more effective management of sepsis.

Conclusion

In summary, acute respiratory distress syndrome (ARDS) remains a critical and multifaceted challenge in intensive care, despite decades of research and advancements in supportive care.

The management of ARDS, particularly fluid therapy, demands a delicate balance to address hypoperfusion without exacerbating pulmonary edema. Key insights from pathophysiological studies and clinical trials underscore the importance of individualized fluid management strategies, low tidal volume ventilation, and adjunct therapies such as prone positioning. Emerging modalities like lung ultrasound and EVLW measurement show promise for tailoring treatment to optimize outcomes. However, further research is essential to refine these approaches and identify new therapeutic avenues. Continued multidisciplinary efforts are critical to improve survival rates and reduce the burden of morbidity associated with ARDS.

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