INTRODUCTION

Acute respiratory distress syndrome (ARDS) is a common complication of critical illness and results in the sudden failure of the respiratory system. ARDS occurs when fluid builds up in the lungs and prevents normal gas exchange. The build up of fluid prevents oxygen from passing through to the bloodstream and can cause rapid, difficult breathing, low blood pressure, and organ failure. ARDS patients are often critically ill with coexisting conditions such as sepsis and multiple organ failure and require ventilator and fluid management support. Despite progress in understanding ARDS pathophysiology and treatment practices, mortality rates are still as high as 40-46% (1) and quality of life post ARDS is subpar.

DISEASE DESCRIPTION

ARDS is the acute onset of clinically significant hypoxemia with presence of diffuse pulmonary edema resulting from increased pulmonary vascular permeability (2). ARDS is used to describe worsened conditions of severe hypoxemia in acute lung injury (ALI). Diagnostic criteria for ALI and ARDS are the same except for oxygenation standards. Partial arterial pressure of oxygen (PaO₂)/fractional concentration of oxygen in inspired air (FiO₂) in ALI is <300 mg Hg, whereas (PaO₂)/(FiO₂) in ARDS <200 mg Hg. ALI and ARDS are both characterized to have <18 mm Hg pulmonary acute wedge pressure with no signs of left arterial hypertension or fluid overload (3). ARDS is a leading cause of prolonged intensive care unit (ICU) stays and has been a subject of great interest.
ETIOLOGY

ALI/ARDS is the result of direct injury or indirect injury to the lungs. Direct injuries may be related to pneumonia, gastric aspiration, drowning, fat and amniotic-fluid embolism, pulmonary contusion, alveolar hemorrhage, smoke and toxic gas inhalation, reperfusion, and unilateral lung re-implantation. Indirect injuries include severe sepsis, transfusions, shock, narcotic overdose, and pancreatitis (2). Factors that appear to increase the risk of developing ALI/ARDS include advanced age, cigarette smoking, alcohol use, and gender (female) indicated during trauma cases. Recent studies suggest a genetic link between variations of the FAS gene and ALI susceptibility (4). Further research is needed to understand causes of ALI/ARDS.

PATHOPHYSIOLOGY

Acute Exudative Phase

ARDS occurs in two stages: the acute exudative phase and fibroproliferative phase. Early histologic changes in the acute exudative phase are characterized by disruption of the capillary endothelium-alveolar epithelium interface, protein-rich pulmonary edema, and increased neutrophilic alveolar infiltrates (3). Direct or indirect injury to the capillary endothelium-alveolar epithelium interface allows protein-rich fluids to leak into the interstitium and alveolar space. Initial injury and presence of pulmonary edema causes diffuse alveolar damage, and stimulates an inflammatory response to release cytokines (e.g. TNF, IL-1, IL-6) and attract neutrophils to the lungs. Neutrophilic activity produces reactive oxygen species and proteases and causes oxidative cell damage. Oxidative stress and protease activity reduce surfactant activity as well as inactivate remaining surfactant
promoting diffuse atelectasis (2). Additionally, protease or elastase activity damages the structural framework of the lung and increased permeability exacerbates pulmonary edema. The presence of excess fluid and atelectasis leads to ventilation-perfusion mismatch and reduced lung compliance, as evidenced by hypoxemic vasoconstriction (3). Sustained pulmonary artery pressures may lead to right heart failure. Oxygen demands will eventually exceed the lungs ability to perform normal gas exchange and result in hypoxemic, hypercarbic respiratory failure.

Fibroproliferative Phase

The initial phase of fluid accumulation is followed by a proliferation phase. Chronic inflammation, fibrosis, and neovascularization occur during the fibroproliferative phase. However, time is the only feature used to distinguish between the exudative phase and fibroproliferative phase (2). The proliferation phase begins within 72 hours and lasts for more than 7 days. Noted activity during this phase includes proliferation of type II alveolar cells, fibroblast activity, and new matrix deposition (1). Type I alveolar cells provide surface area for gas exchange to occur. Type II alveolar cells act as progenitor cells for both type I and II alveolar cells, as well as synthesize, store, and release surfactant in order to maximize gas exchange (5). Fibroblast activity synthesizes collagen, the structural framework for connective tissue and promotes scarring. During the proliferation phase, type II cells proliferate with some alveolar epithelial cell regeneration and fibroblastic remodeling (6). It is not completely understood what factors influence progression into the proliferation phase or recovery. However, those who develop fibrosis have decreased lung compliance, as well as increased hypoxemia and risk of mortality (1).
METHODS OF MEDICAL DIAGNOSIS

There is no specific method for diagnosing ARDS. ARDS is diagnosed by ruling out other diseases or conditions that produce similar symptoms.

Imaging
A chest x-ray is a non-invasive test that creates pictures of the chest, heart, lungs, large arteries, ribs, and diaphragm using electromagnetic waves. Computerized tomography (CT) scan combines x-ray images taken from different cross-sectional views to provide information about structures within the heart and lungs. ARDS patients are diagnosed with bilateral pulmonary infiltrates seen on a front chest radiograph.

Lab Tests
Tests used to diagnose ARDS include arterial blood gas, CBC to indicate low oxygen levels or hypoxemic conditions, and tests for possible infections. Sputum cultures can be collected and analyzed to determine type of lung infection.

Heart Tests
ARDS is diagnosed by excluding cardiogenic pulmonary edema and other causes of hypoxemic respiratory failure. Electrocardiogram and echocardiogram are used because signs and symptoms of ARDS are similar to those of heart problems. For instance, an echocardiogram is used to rule out congestive heart failure, which can look similar to ARDS on a chest x-ray (7).

CURRENT MEDICAL THERAPIES
Currently there are no specific treatments for ARDS. Better understanding of the pathogenesis of ARDS and technological advancements has improved therapeutic
practices. Supportive care looks to treat the underlying cause of ARDS, as well as maintain adequate oxygenation and avoid iatrogenic complications (2). ICU patient beds will raise the head of bed 30 degrees to reduce risks of hospital-acquired pneumonia and aspiration.

Mechanical Ventilation

Mechanical ventilation relieves the respiratory muscles of their work by using supplying supplemental oxygen and pressure to expand atelectatic alveoli and assist with impaired gas exchange. The traditional approach of ventilation was to achieve near normal arterial blood gases, even if high tidal volumes and volume-cycled ventilation were used. It became apparent that high tidal volumes and increased extrinsic positive end expiratory pressure (PEEP) leads to over-distention and injury of compliant alveoli, as well as exacerbates the existing injury and inflammatory response (2). Current practices take a pressure and volume-limited approach with permissive hypercapnia.

Low Tidal Volume Ventilation

Tidal volume represents the volume of air displaced between inspiration and expiration. Studies show large tidal volumes and high inspiratory pressures results in development of hyaline membranes and exacerbates the inflammatory response (1). The National Heart Lung and Blood Institute (NHLBI) ARDS Network conducted a randomized controlled trial that compared low versus high tidal volume ventilated patients. Low tidal volume had higher survival rates and higher rates of weaning from mechanical ventilation (8). Current recommendations are to use 6 mL/kg predicted bodyweight tidal volume.
PEEP

Positive end expiratory pressure (PEEP) is the alveolar pressure that exists at the end of expiration. PEEP provides pressure support in atelectatic alveoli, increases functional residual capacity, and improves oxygenation. High PEEP levels could increase edema, over-distend compliant alveoli, worsen ventilation-perfusion matching, and cause hypotension. Research suggests higher PEEP levels are not harmful but pressure-limited mode of ventilation be used rather than volume-cycled ventilation (2). Current recommendations are to use the lowest PEEP-F,\textsubscript{1}O\textsubscript{2} that produces acceptable oxygenation 88%-95%.

Weaning

As soon as the condition that caused respiratory failure has started to improve, weaning from ventilation may begin (9). The transition requires patients to be hemodynamically stable, able to take spontaneous breaths, and need <50% supplemental O\textsubscript{2} and <8 cm H\textsubscript{2}O PEEP (2).

Prone Positioning

Improved oxygenation has been reported in ARDS patients when in the prone position. Proposed benefits from prone positioning include increased chest wall elastance, improved ventilation due to decreased ventilation-perfusion mismatch, and reduced ventilator-associated lung injury (1). However, evidence does not support routine use of the prone position in improving survival rates (10).

Corticosteroids

Corticosteroids are used to regulate the inflammatory response through increased
encoding for anti-inflammatory proteins and decreased expression of inflammation genes. Evidence for the use of corticosteroids to prevent or treat early ALI is inconclusive, but may be beneficial in late persistent ALI (2).

Fluid Management
Increased lung capillary permeability leads to an accumulation of fluids in the interstitium and alveolar space. The NHLBI ARDS network recommends conservative fluid administration to improve lung function and shorten duration of mechanical ventilation (1). Fluid restrictions and diuretic use help to alleviate pulmonary edema seen in ARDS.

Vasodilators
Pulmonary hypoxemic vasoconstriction is part of ARDS pathophysiology and selective vasodilation helps to improve blood flow and gas exchange in ARDS patients. Although oxygenation and pulmonary resistance are improved, evidence does not indicate better clinical outcomes (2).

Extracorporeal Support
An extracorporeal membrane oxygenator (ECMO) is a device used to provide respiratory support by circulating blood through an artificial lung, then returning the blood to the patient’s circulatory system (11). ECMO uses a blood-gas interface for the diffusion of gas exchange. Extracorporeal support provides temporary improvement in those with severe ARDS, but does not seem to affect clinical outcomes (1).

APPROPRIATE TOOLS IN NUTRITION ASSESSMENT
Nutrition support is essential to meet energy requirements and maintain muscle strength.
An accurate assessment of needs is important in ALI/ARDS. Underfeeding can prevent unsuccessful ventilator weaning and increase susceptibility of infection, whereas overfeeding can lead to hypercapnea and delay ventilator weaning (3). Indirect calorimetry (IC) should be used to obtain initial needs assessment and repeated during length of stay.

MNT

Basic medical nutrition therapy goals are to meet basic nutritional requirements, preserve lean body mass, restore respiratory muscle mass and strength, maintain fluid balance, improve resistance to infection, and facilitate weaning from oxygen support and mechanical ventilation by providing energy substrates without exceeding the capacity of the respiratory system to clear CO2 (12). Nutritional implications of hypoxemic state as a result of ARDS condition include anorexia, early satiety, malaise, bloating, constipation and diarrhea. A person with respiratory failure can burn more calories breathing than a healthy person does. It is easy to lose weight without trying.

Obesity and Underfeeding

In obese patients, caloric restriction can reduce the hypercapnea and fluid retention that is seen in ARDS. Protein provision should be a priority as it promotes fat oxidation and prevents further muscle wasting. It is recommended to provide 60-70% of target or 11-14 kcal/kg and 1.5-2.0 g/kg of protein (3).

Hypercapnea and Overfeeding

Permissive hypercapnia from mechanical ventilator support has nutritional implications. Carbohydrates have high CO2 production during metabolism. In order to achieve a respiratory quotient of ~ .7, ARDS patients should consume a high fat diet. Overfeeding
increases lipogenesis, increases glucose levels, leads to hepatic dysfunction and the inability to wean from the ventilator (3). Constant assessment of nutritional needs is important to prevent overfeeding.

**Oxepa**

Oxepa is the recommended formula for ARDS patients on nutrition support. Oxepa contains fatty acids, vitamins A, C, and E, and is volume reduced. Lipids reduce carbohydrate load and are increasingly used in immunonutrition. Oxepa decreases pulmonary inflammation and edema, facilitates pulmonary vasodilation, improves oxygenation, decreases time on a ventilator, reduces new organ failures, decreases ICU length and stay, lowers mortality rates, and is less expensive (13).

**Post ICU**

After weaning from ventilator support/EN support it is important to eat a calorically dense diet including a variety of foods. Eating several small meals a day of favorite foods will help to consume necessary amount of calories without feeling uncomfortable.

**LONG TERM PROGNOSIS**

Several different pathways are involved in the development of ARDS, and there isn’t a single biomarker that can predict the outcome of ARDS patients. Multiple Organ Dysfunction Syndrome (MODS) is the leading cause of death in ARDS (1). Post ICU patient survivors reported reduced physical function and quality of life despite normal or almost normal pulmonary function.
CONCLUSION

ALI/ARDS continues to affect morbidity and mortality rates in critically ill patients. Treatment of the underlying cause of ARDS and as well as mechanical ventilation support can help to attenuate conditions of respiratory failure. Early nutrition support is critical to meet increased energy and protein needs related to hypoxemia and muscle wasting. Fluid management and proper MNT help to shorten time on ventilator support and reduce ICU length of stay.
REFERENCES


(11) Leonard PC. *Building a Medical Vocabulary.* 8th ed. St. Louis County, MO: Elsevier Inc; 2012


Acute Respiratory Distress Syndrome (ARDS)
ARDS

- ARDS: a life-threatening condition characterized by severe hypoxia, bilateral pulmonary fluid infiltration, and decreased lung compliance; usually occurring without prior lung disease but secondary to catastrophic illness
Etiology

- The most common underlying causes of ARDS include:
  - Sepsis
  - Inhalation of harmful substances
  - Severe pneumonia
  - Head or chest injury
Risk Factors

- **Direct lung injury**
  - Pneumonia, gastric aspiration, drowning, fat and amniotic-fluid embolism, pulmonary contusion, alveolar hemorrhage, smoke and toxic gas inhalation, reperfusion, and unilateral lung re-implantation

- **Indirect lung injury**
  - Severe sepsis, transfusions, shock, narcotic overdose, and pancreatitis
Several risk factors appear to increase the risk of ARDS after an inciting event including:

- Advanced age
- Female sex (noted only in trauma cases)
- Cigarette smoking
- Alcohol use
Diagnosis

- There is no specific test to identify ARDS
- Diagnosis is reached by ruling out other diseases and conditions
<table>
<thead>
<tr>
<th></th>
<th>Timing</th>
<th>Oxygenation</th>
<th>Chest Radiograph</th>
<th>Pulmonary Acute Wedge Pressure</th>
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<tbody>
<tr>
<td>ALI criteria</td>
<td>Acute onset</td>
<td>$P_{a02}/F_{I02} &lt; 300\text{mg Hg}$</td>
<td>Bilateral infiltrates seen on frontal chest radiograph</td>
<td>$&lt;18\text{mm Hg when measured or no clinical evidence of left atrial hypertension}$</td>
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<tr>
<td>ARDS criteria</td>
<td>Acute onset</td>
<td>$P_{a02}/F_{I02} &lt; 200\text{mg Hg}$</td>
<td>Bilateral infiltrates seen on frontal chest radiograph</td>
<td>$&lt;18\text{mm Hg when measured or no clinical evidence of left atrial hypertension}$</td>
</tr>
</tbody>
</table>
Bilateral pulmonary infiltrates detected

- **Chest X-rays**
  - A chest X-ray can reveal which parts of the lungs have fluid in them and whether the heart is enlarged

- **CT Scan**
  - A CT scan can provide detailed information about the structure within the heart and lungs
Lab Tests

- **Blood testing**
  - Arterial blood gas, CBC to indicate low oxygen levels or hypoxemic conditions, and tests for possible infections

- **Sputum cultures**
  - Collected and analyzed to determine type of lung infection
Heart Tests

- Signs and symptoms of ARDS are similar to cardiogenic pulmonary edema and other causes of hypoxemic respiratory failure.
- An “objective assessment”—meaning an echocardiogram—should be preformed if there is no clear risk factor present like trauma or sepsis.
Pathology

Scientific study of the nature of disease and its causes, processes, development, and consequences.
Histopathology

Diffuse alveolar damage characterized by:

- Neutrophilic alveolar infiltrates
- Hemorrhage
- Protein-rich pulmonary edema
Acute Exudative Phase

- Cytokines (e.g. TNF, IL-I, IL-8) $\Rightarrow$ inflammatory response

- Oxidative stress & protease activity
  - Reduces surfactant activity, inactivates remaining surfactant $\Rightarrow$ atelectasis
  - Damage of structural framework
    - Increased permeability exacerbates alveolar edema

- Capillary thrombosis
Fibroproliferative Phase

- Chronic inflammation
- Pulmonary fibrosis
- Neovascularization
ARDS Stages

1. Exudative stage: Characterized by accumulation in the alveoli of excessive fluid, protein and inflammatory cells that have entered the air spaces from the alveolar capillaries. The exudative phase unfolds over the first 2 to 4 days after onset of lung injury.

2. Fibroproliferative (or proliferative) stage: Connective tissue and other structural elements in the lungs proliferate in response to the initial injury. Under a microscope, lung tissue appears densely cellular. Also, at this stage, there is a danger of pneumonia sepsis and rupture of the lungs causing leakage of air into surrounding areas.

3. Resolution and Recovery: During this stage, the lung reorganizes and recovers. Lung function may continue to improve for as long as 6-12 months and sometimes longer, depending on the precipitating condition and severity of the injury. It is important to remember that there may be and often are different levels of pulmonary recovery amongst individuals who suffer from ARDS.
Pathophysiology

- Inflammatory infiltrates, edema, atelectasis lead to:
  - Ventilation-perfusion mismatch $\rightarrow$ hypoxemia
  - Reduced lung compliance

- Hypoxemic vasoconstriction, capillary damage
  - Increases pulmonary-artery pressures
  - Sustained pressures $\rightarrow$ cor pulmonale
Pathophysiology Cont.

- Increased efforts of breathing
- Oxygen demands exceed respiratory capability
- \( \rightarrow \) hypoxemic, hypercarbic respiratory failure
Treatment

• No specific treatment

• Supportive care
  ○ Avoid iatrogenic complications
  ○ Treat underlying cause
  ○ Maintain adequate oxygenation

• Mechanical ventilation
  ○ Reduces airway pressure
Supportive Care

- Treat underlying cause of ARDS
- Prevent deep vein thrombosis, GI bleeding, pressure ulcers
- Bed head raised 30 degrees
  - Reduce risk of hospital-acquired pneumonia
- Enteral nutrition
  - Glucose control
Endotracheal tube goes through patient's mouth and into the windpipe.

Mechanical ventilator blows air, or air with increased oxygen, through tubes into the patient's airways.

Nasogastric tube goes through patient's nose and into the stomach.

Air flowing to the patient passes through a humidifier, which warms and moistens the air.

Exhaled air flowing away from the patient.

Nurse periodically checks the patient.
Mechanical Ventilation, Oxygen, PEEP

- Lung capacity affected by age, gender, height
- Mechanical ventilation used to relieve respiratory muscles of their work
- Usually tracheal-tube-delivered ventilation
Mechanical Ventilation, Oxygen, PEEP

- **High tidal volume ventilation**
  - Over-distention and injury of compliant alveoli
  - Exacerbate existing injury
  - Local, systemic inflammation

- **Positive-pressure ventilation**
  - Supplemental oxygen
  - PEEP
Mechanical Ventilation, Oxygen, PEEP

- **PEEP**
  - Pressure support in atelectic alveoli, increases functional residual capacity, improves oxygenation
  - Could increase edema, over-distend compliant alveoli, worsen ventilation-perfusion matching, cause hypotension

- Pressure-limited mode of ventilation better than volume-cycled ventilation
Recommendations

- Pressure and volume-limited approach with permissive hypercapnia
- 6 mL/kg adjusted bodyweight tidal volume
- Lowest PEEP-$F_iO_2$ that produces acceptable oxygenation 88%-95%
Weaning from ventilators

- Hemodynamically stable
- Spontaneous breaths
- <50% supplemental O\(_2\), <8 cm H\(_2\)O PEEP

- Modify inflammation, change mechanical properties of lungs
  - No conclusive evidence
Treatment cont.

- Prone Positioning
- Corticosteroids
- Fluid management
  - Diuretic use to reduce lung water
- Vasodilators
- Extracorporeal support
Actions of Corticosteroids

- Decreased extravasation of fluid through intercellular junction
- Inhibition of adhesion of neutrophils to endothelial cell

Leukocyte

- Corticosteroid
- Corticosteroid receptor
- Nucleus
- Cytoplasm
- Increased encoding of anti-inflammatory proteins
- Steroid-sensitive genes
- Decreased expression of inflammation genes
Extracorporeal Support (ECMO)

- Oxygenates and removes $\text{CO}_2$ from the blood

- Blood withdrawn into oxygenator, returned
  - Blood-gas interface for diffusion of gases
**Table 1. Indications and Contraindications for ECMO in Severe Cases of ARDS.**

**Indications**

- Severe hypoxemia (e.g., ratio of $P_aO_2$ to $F_iO_2 < 80$, despite the application of high levels of PEEP [typically 15–20 cm of water]) for at least 6 hr in patients with potentially reversible respiratory failure†
- Uncompensated hypercapnia with acidemia (pH < 7.15) despite the best accepted standard of care for management with a ventilator
- Excessively high end-inspiratory plateau pressure (>35–45 cm of water, according to the patient’s body size) despite the best accepted standard of care for management with a ventilator

**Relative contraindications**

- High-pressure ventilation (end-inspiratory plateau pressure >30 cm of water) for >7 days
- High $F_iO_2$ requirements (>0.8) for >7 days
- Limited vascular access
- Any condition or organ dysfunction that would limit the likelihood of overall benefit from ECMO, such as severe, irreversible brain injury or untreated metastatic cancer

**Absolute contraindication**

- Any condition that precludes the use of anticoagulation therapy‡
Basic MNT Goals

- Meet basic nutritional requirements
- Preserve lean body mass
- Restore respiratory muscle mass and strength
- Maintain fluid balance
- Improve resistance to infection
- Facilitate weaning from oxygen support and mechanical ventilation by providing energy substrates without exceeding the capacity of the respiratory system to clear CO2
MNT Cont.

- Negative Nitrogen balance
  - Protein (1.5-2 g/kg)
- Pulmonary rehabilitation programs
  - Small portions
  - Favorite foods
- Inadequate oxygen
  - Anorexia
  - Early satiety
  - Malaise
  - Bloating
  - Constipation
  - Diarrhea
Obesity and Underfeeding

- Underfeeding delays ventilator weaning
- Factors associated with obesity:
  - Chest physiology
    - Reduced functional reserve capacities
    - Smaller lung volumes
    - Hypercapnea
  - Glucose resistance
  - Mobilization of fat stores
Obesity and Underfeeding

- **Protein provision**
  - Promotes endogenous fat oxidation
  - Shifts from utilization of muscle stores

- **Caloric restriction**
  - Improves glycemic control
  - Reduces hypercapnea and fluid retention
  - Endorse hypocaloric feeding for obese patient with goals of:
    - 60-70% of target or
    - 11-14 kcal/g with 2.0-2.5 g/kg of protein
Hypercapnia and Overfeeding

- Limit tidal volumes to prevent further inflammation and barotrauma
- Increases CO2 production
- Can lead to:
  - Increased lipogenesis
  - Increased glucose levels
  - Hepatic dysfunction
  - Inability to wean from the ventilator
Lipids in ARDS

- Anti-inflammatory lipid substrates
- Omega-3 fatty acids
- Fish oils (DHA and EPA)
- Resolvins
- Neuroprotectin
EN

- Always given when gut is functional
- Early EN (within 24-48 hours of arrival)
- Feed directly into small bowel
- Supplementation with vitamins A, C, E
- Phosphate
PN

- Shock
- Nonfunctional gut
- Peritonitis
- A marker for severity of disease
Pulmonary Inflammation

All further deteriorating a patient's condition

Impaired gas exchanged and poor oxygenation

Pulmonary inflammation with edema and vasoconstriction

Proinflammatory eicosanoids and free radicals produced

Clinical catastrophe

Increase in neutrophil recruitment
How Oxepa Works

- **Borage Oil** (GLA) → **DGLA** → **Cyclooxygenase, Lipoxygenase**
  - Replacing amino acids with GLA results in...
  - **Results in decreased proinflammatory eicosanoids (LTB₄, TXA₂, PGE₂)**

- **Arachidonic Acid**
  - **Fish Oil** (EPA) → **Eicosanoids** that are less inflammatory (LTB₃, TXA₃, PGE₅)

- **Replacing Amino Acids with EPA results in...**
Benefits of Oxepa

- Decreases pulmonary inflammation and edema
- Facilitates pulmonary vasodilation
- Improves oxygenation
- Decreases time on ventilator
- Reduces new organ failures
- Decreases ICU length of stay
- Lowers mortality rates
- Less expensive
Clinical Outcomes

- Time on Ventilator
  - Control: 17.6 days
  - Oxepa®: 13.1 days

- ICU LOS
  - Control: 18.4 days
  - Oxepa®: 14.8 days

28 Day Survival

Survival Distribution Function

P = 0.037

19.4% Absolute Risk Reduction in Mortality

67.3% Oxepa®

47.9% Control

Time (Days)

## Cost of Care

<table>
<thead>
<tr>
<th>Patient</th>
<th>Average Cost Per Day</th>
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<td>ARDS Patient</td>
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<tr>
<td>ICU Bed</td>
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<td>Ventilator Support</td>
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<td><strong>Oxepa</strong></td>
<td><strong>$25</strong></td>
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Enteral Feedings in ARDS

- Oxepa for 60kg (132lb) Adult
- 25 kcal/kg diet for 24hrs.

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<th>Amount(g)</th>
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<td>Fluid</td>
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<tr>
<td>Total Calories / 24hrs</td>
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</table>
Quality of Life

- Health-related quality of life (HRQL) - a self-reported measure of one’s own physical, mental, and social well-being
- Short Form-36 (SF-36) health survey - The most commonly used and best-validated tool to measure HRQL
- Nottingham Health Profile (NHP) - NHP gives more coverage to emotional reaction, pain, and physical mobility and unlike SF-36, NHP includes problems of sleep disturbance
- distance walked in 6 minutes with continuous oximetry (6MWD)
One-year post-ICU in ARDS

- 104 patients
- 6MWD improved over first year post-ICU
- All domains of SF-36 improved from 3 to 12 months and physical role and physical functioning domains showed greatest improvement
- when they were compared to general population at 1 year, all domains except emotional role were reduced
Radar plots summarizing SF-36 scores and pulmonary function in survivors of acute lung injury (ALI)/acute respiratory distress syndrome (ARDS) at 3, 6, 12 and 24 months.

Source: Data for PFT graph from Herridge et al., 2003 and Cheung et al., 2006.
Figure 2. Representation of quality of life in patients with ARDS 6 months after the event and in the reference population. The black line represents quality of life 6 months after ARDS. The gray line represents quality of life of the healthy reference population. *P < .05.
Radar plots summarizing SF-36 scores and pulmonary function in survivors of acute lung injury (ALI)/acute respiratory distress syndrome (ARDS) at 3, 6, 12 and 24 months.

Source: Data for PFT graph from Herridge et al., 2003 and Cheung et al., 2006.
4 years and 5.5 years post-ICU in ARDS

- 50 patients
- 54% survivors had normal lung function at 5.5 years
- Survivors had impairment of all domains SF-36 with 25% reduction physical functioning and physical role function
5 years post - ICU

- 81% Survived
- Only 86% of survivors available for follow-up at 5 years
- Also showed that both physical function and quality of life indices were reduced despite normal or almost normal pulmonary function.
Future Research Focus

- Strong relationship between the overall HRQL encountered at 1 month and at 6 months after ARDS, which indicates that identification of patients who will have a poor long-term quality of life is possible at an early time point.
- Impaired HRQL has little to do with pulmonary function per se, as symptoms are a result of impaired recovery of strength, immobilization, critically illness polyneuropathy, and entrapment neuropathies.
- Early intervention strategies to prevent or ameliorate physical morbidity may prove important for treating survivors of critical illness.
**Figure 5.** Comparative study between overall quality of life assessed at 1 month and 6 months after ARDS. HRQL = health-related quality of life.
Case Study

- Daishi Hayato
- 65yrs old male, lives with wife (62yrs old)
- “My husband had emphysema for many years. He was working in the yard today and got really short of breath. I called our doctor, and she said to go straight to the emergency room.”
Case Study Cont.

- Long-standing history of COPD due to tobacco use 2PPD for 50 yrs
- Dyspnea, swelling in lower extremities, pneumothorax, intermittent claudication
- Meds: Combivent, Lasix, O2 via nasal cannula at night
- nutrition Hx - Appetite has been decreased for past several weeks.
Case Study Cont.

- Ht 5’4”, Wt 122 lbs, UBW 135LBS
- HDL – C 32mg/dL - low
- LDL – 132 MG/dL - high
Case Study Cont.

• PES statement
• Increased nutrient needs related to long standing history of COPD and nutrient deprivation from Acute Respiratory Distress Syndrome as evidenced by decreased dietary intake and low body weight.
Acute Respiratory Distress Syndrome (ARDS)

What is acute respiratory distress syndrome?
Acute respiratory distress syndrome (ARDS) is the sudden failure of the respiratory system. It can occur in anyone over the age of one who is critically ill. ARDS can be life-threatening because normal gas exchange does not take place due to severe fluid buildup in both lungs. The condition is characterized by rapid breathing, difficulty getting enough air into the lungs and low blood oxygen levels.

Who has ARDS?
The incidence of ARDS has been difficult to determine partly because of the variety of causes, clinical manifestations and differing criteria used to define it. Various published estimates have ranged from 1.5 to 75 cases per 100,000 persons. In 2007, the National Heart, Lung and Blood Institute estimated that approximately 190,000 Americans are affected by ARDS annually.

What is the health impact of ARDS?
Lung function in most survivors of ARDS will return to normal or near normal within several months; however some will have lasting damage to their lungs or to areas outside the lungs. A study found that one year after discharge from the intensive care unit, ARDS survivors may still suffer side effects, mostly persistent muscle wasting and weakness. Quality of life in these survivors is compromised with poor mental and physical health outcomes.

Food plan for ARDS

Calorie intake to meet energy needs
A person with respiratory failure can burn more calories breathing than a healthy person does. They find that they lose weight without trying. Therefore, it is important for the patient to eat a calorically dense diet including a variety of foods.

Timing of meals
Eating several small meals a day instead of two or three large ones can help you eat well and not feel uncomfortable. Try dividing your day’s food into 4 to 6 small meals. You can eat a small breakfast, lunch, and dinner. Then get the rest of your nutritional needs for the day by eating two or three between-meal snacks.