Acute Respiratory Distress Syndrome

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Risk

- Recent data estimates the incidence at 190,000 cases per year in USA. True incidence is unknown due to difficulty in defining the disease and making the diagnosis.
- Represents 10.4% of all ICU admissions and 23.4% of pts requiring mechanical ventilation per a recent 2016 publication.
- Mortality rates vary from 25% to 40%. Mortality rate is strongly influenced by associated conditions (e.g., higher when associated with sepsis, liver disease, and advanced age; lower with trauma, transfusion-related lung injury, drug overdose, or other reversible conditions).

Perioperative Risks

- Increased risk of sudden and profound hypoxia secondary to loss of alveolar recruitment
- Worsening resp status due to effects of anesthesia and surgery
- Difficult balance between maintaining adequate intravascular volume and avoiding pulm edema and right heart strain leading to decreased oxygenation and ventilation

Worry About

- Maintaining required PEEP during pt transport with Ambu bag or Mapleson circuit. Transport with ICU ventilator may be necessary.
- Inability of standard OR ventilators to deliver required minute ventilation, high inspiratory pressures, and inverse ratio ventilation.

Overview

- Berlin definition of ARDS (published in 2012) requires each of the following criteria:
  - Timing—onset within 1 week of a known clinical insult or new or worsening resp symptoms.
  - Chest imaging (CXR or CT)—bilateral opacities; not fully explained by effusions, lobar/lung collapse, or nodules.
  - Origin of edema—resp failure not fully explained by cardiac failure or fluid overload, need objective assessment (ECHO) to exclude cardiogenic pulm edema.
  - Oxygenation:
    Mild—PaO_2/FiO_2 200 to 300 mm Hg with PEEP or CPAP ≥ 5 cm H_2O.
    Mod—PaO_2/FiO_2 100 to 200 mm Hg with PEEP ≥ 5 cm H_2O.
    Severe PaO_2/FiO_2 ≤ 100 mm Hg with PEEP ≥ 5 cm H_2O.
- Though classically defined by severe hypoxia, also can be associated with profound hypercarbia due to elevated alveolar dead space.
- Associated with low pulm compliance and lung volumes (due to alveolar edema and atelectasis) and, in certain pts, with abnormally low chest wall compliance.
- Most deaths are from sepsis or multisystem organ failure (more rarely from refractory hypoxemia or hypercarbia).

Etiology

- Direct or indirect lung injury leading to acute inflammatory alveolar damage characterized by increased microvascular permeability with interstitial and alveolar edema and often progressing to fibrosis.
- Precipitants include aspiration, pneumonia, sepsis, massive transfusion, pancreatitis, trauma, ischemia-reperfusion, drugs and alcohol, CNS injury, air embolism, cardiopulmonary bypass, genetic predisposition.
- Mechanical ventilation may worsen lung injury through alveolar overdistention and shear forces from cyclic opening and closing of collapsed alveoli (ventilator-associated lung injury).

Usual Treatment

- ARDS net trial (2000) demonstrated reduced mortality in pts ventilated with lower tidal volumes and decreased airway plateau pressures. Aim for TVs 6 to 8 mL/kg (ideal body weight) and plateau pressure ≤30 cm H_2O. Maintain ventilation with increased resp rate.
- Do not attempt to correct hypercarbia. Instead, direct ventilation toward maintaining acceptable pH (>7.15). There is no evidence that moderate acidemia is harmful in pts who do not have specific contraindications (i.e., intracranial hypertension).
- Apply PEEP to maintain alveolar recruitment and achieve O_2 saturation ≥88%. No consistent evidence shows benefit from high versus moderate levels of PEEP. Higher PEEP is reasonable if pt remains hemodynamically stable. Monitor for auto-PEEP (air trapping).
- Choose the lowest tolerated FiO_2 (actual FiO_2 associated with oxygen toxicity is unknown).
- Consider sedation, analgesia, and fever reduction to improve ventilator synchrony and decrease O_2 consumption.
- ACURASYS trial (2010) demonstrated reduced mortality in pts with severe ARDS (PaO_2/FiO_2 ratio ≤120) paralyzed with cisatracurium for 48 h.
- PROSEVA trial (2013) demonstrated reduced mortality in pts with severe ARDS (PaO_2/FiO_2 ratio ≤150) treated with intermittent (16 h/day) prone positioning. Prone positioning improves ventilation-perfusion matching of dependent (posterior) alveoli.
- OSCILLATE trial (2013) demonstrated possible increased mortality with HFOV.
- Potential role of ECMO and specifically veno-venous ECMO in treatment of most severe cases, but strong evidence and guidelines for use not yet established
- Role of steroids, incl pt selection, timing, and dosing, remains unclear.
- Diagnose and treat precipitating and underlying conditions.
- Prevent and treat fluid overload.

Assessment Points

System	Effect	Assessment by Hx	PE	Test
CV	Pulm Htn RV and/or LV dysfunction Septic shock Fluid overload	Hypotension, ↓ renal and hepatic function, metabolic acidosis	Cool extremities, narrow pulse pressure, JVD, RV heave, peripheral edema, enlarged liver, abdominal distension	PA cath, ECHO, mixed venous oxygen saturation
RESP	Ventilator-associated lung injury Pneumothorax	Increased airway pressures, impaired resp mechanics, worsening blood gases	Bilateral rhonchi, crackles decreased or absent breath sounds, tracheal deviation	CXR, CT chest
ID	Ventilator associated pneumonia Line sepsis	Increased WBC/bandemia, new infiltrates, hypotension	Fever, purulent secretions	CXR, CT chest, blood and sputum culture
GI	Hemorrhage	Decreased Hct	Melena, bloody NG output	Esophagogastroduodenoscopy
GU	Acute kidney injury	Oliguria, increased creatinine	Peripheral edema	Serum creatinine
MS	Prolonged weakness Diaphragm atrophy	Pharmacologic paralysis, high-dose steroids, sepsis, prolonged ventilation	Polyneuropathy, myopathy	Electromyography, muscle biopsy

Key References: Bernard GR: Acute respiratory distress syndrome, *Am J Respir Crit Care Med* 172:798–806, 2005; Guldner A, Kiss T, Serpa Neto A, et al: Intraoperative protective mechanical ventilation for prevention of postoperative pulmonary complications: a comprehensive review of the role of tidal volume, positive end-expiratory pressure, and lung recruitment maneuvers, *Anesthesiology* 123:692–713, 2015.

Perioperative Implications

Preoperative Preparation

- Assess current ventilator mode and settings in ICU and review last blood gas.
- Assess pt preop hemodynamic and intravascular volume status.
- Use PEEP valve for pt transport or consider transportation to OR on ICU ventilator.
- Consider use of ICU ventilator intraop with concurrent total intravenous anesthesia, particularly when very high minute ventilation and airway pressures are required.
- Maintain comparable levels of mean airway pressure and minute ventilation when transitioning between modes or ventilators and when paralyzing the pt.

Airway

- Avoid suctioning and unnecessary ETT disconnection. Even transient loss of PEEP may result in lung derecruitment and severe hypoxemia that is difficult to correct.

Monitoring

- In most severe pts, PA cath or intraop TEE may be helpful in estimating intravascular volume status and ventricular function.
- Closely monitor airway pressures (peak, plateau, mean airway), tidal volumes, minute ventilation.
- Monitor oxygen saturation and obtain frequent blood gases. $ETCO_2$ may not be representative of arterial PCO_2 due to increased dead space.

Preinduction/Induction

- Expect increased shunt with elevated FiO_2 and/or PEEP requirements due to loss of hypoxic pulm vasoconstriction caused by anesthetics.
- Prepare for worsening resp mechanics and decreased ventilation in spontaneously breathing pt given anesthetics, narcotics, or muscle relaxants.
- Prepare for elevated airway pressures with supping positioning and increased risk of aspiration (suction stomach via NG/OG tube before lying supine).

Maintenance

- Attention to fluid management to avoid worsening pulm edema and right heart strain from excessive fluid administration.
- Consider treating worsening hypoxemia with recruitment maneuvers (apply continuous airway pressure of 40 to 50 cm H_2O for 40 s) followed by increased PEEP setting.

Postoperative Period

- Continued careful monitoring of hemodynamic and volume status.
- Reassess ventilator settings and reduce FiO_2 and airway pressures as tolerated.

Anticipated Problems/Concerns

- Sudden and profound hypoxia can occur if lung recruitment is lost during transport, movement, positioning, or surgical retraction.

Addison Disease

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Risk

- Prevalence 1:100,000 persons
- M:F ratio: 1:1.8

Perioperative Risks

- CV instability, labile BP, hypotension, shock
- Hypovolemia, hyperkalemia, cardiac dysrhythmia
- Limited response to vasopressors

Worry About

- N/V and diarrhea leading to dehydration, electrolyte imbalances, and acid/base disorder.
- Acute adrenal insufficiency leading to hypotension and refractory distributive shock.
- Cardiac dysrhythmia caused by hyperkalemia.
- Hypoglycemia and uremia, muscle weakness, decreased level of consciousness.

Overview

- Addison disease is a specific type of adrenal insufficiency due to a primary inadequate production of glucocorticoids, mineralocorticoids, and androgens by the adrenal glands.
- Nonspecific symptoms and insidious disease progression often result in a delay in diagnosis until after the development of addisonian crisis after a significant stressor or illness.
- Pts often present with chronic fatigue as well as GI disturbances; pain, nausea/vomiting, diarrhea, and may develop episodes of mental status changes.
- Diagnosed by cosyntropin stimulation test; administration of cosyntropin will stimulate ACTH secretion by pituitary but will not increase cortisol levels.
- May be associated with other autoimmune conditions
- Drugs that inhibit cortisol biosynthesis will trigger addisonian crisis; etomidate, antifungals.
- See also Adrenal Insufficiency, Acute or Secondary.

Etiology

- 80% of cases are due to immune destruction of the adrenal cortex by autoantibodies.
- Most often an antibody against 21-hydroxylase. Presence of these autoantibodies may predate development of clinical disease by decades.
- Other causes include infection (TB, histoplasma, HIV, CMV), cancer metastases, bilateral adrenalectomy, sepsis especially meningococcal, hemorrhage, and infiltrative diseases.

Usual Treatment

- Lifelong hormone replacement therapy. Glucocorticoid: Prednisone 3 to 5 mg daily and hydrocortisone 5 to 25 mg divided into 2 to 3 times/d. Mineralocorticoid: Fludrocortisone 0.05 to 0.2 mg daily. Men do not need androgen replacement as their androgens are produced in the testes. Women may benefit from DHEA 25 to 50 mg daily.
- Acute adrenal insufficiency treatment: Supportive treatment with rapid isotonic solution, hydrocortisone IV 100 mg q8h, and electrolyte replacement
- See Adrenal Insufficiency, Acute or Secondary for procedure-adjusted stress dose regiments

Assessment Points

System	Effect	Assessment by Hx	PE	Test
CV	Hyponatremic hypovolemia, hypotension, CV instability	Postural symptoms, salt cravings, weight loss	Low BP, orthostatic changes, dry mucous membranes, poor cap refill	CBC, chemistry, BUN/Cr, ACTH stimulation test
MS	Muscle weakness, high urea	Fatigue, anorexia, N/V	Decreased level of consciousness, potentiation of neuromuscular blockade	BUN, nerve stimulator
GI	Dehydration, pH disturbances	Abdominal pain, N/V, diarrhea	See CV	Chemistry panel
ENDO	Hyperkalemia, hyponatremia, hypoglycemia	Weakness, cardiac dysrhythmia, depression	Inability to stand from seated position, flat affect	Chemistry panel, ECG
DERM	Excess corticotropin release	Vitiligo, changes in skin color	Hyperpigmentation	ACTH stimulation test

Key References: Jung C, Inder WJ: Management of adrenal insufficiency during the stress of medical illness and surgery, *Med J Aust* 188(7):409–413, 2008; Michels A, Michels N: Addison disease: early detection and treatment principles, *Am Fam Physician* 89(7):563–568, 2014.

Perioperative Implications

Perioperative Preparation

- Glucocorticoid and mineralocorticoid levels should be checked and optimized.
- Stress dose steroid coverage in periop period.
- Measure electrolytes, BUN, creatinine, glucose, and correct abnormalities.
- Ensure normovolemia.

Monitoring

- Standard ASA monitors.
- Arterial line and central line may be necessary in acute adrenal insufficiency.
- Na^+, K^+, pH, glucose.

Airway

- No specific recommendations

Premedication/Induction

- Avoid etomidate; may be associated with increased mortality in this population.

Maintenance

- Anticipate hypotension; dose adjustment may be required for muscle relaxants.

Extubation

- Appropriate muscle relaxant reversal must be achieved.

Adjuvants

- Glucose/dextrose, pressor drips, fluids, hormone replacement.

Postoperative Period

- Monitor pts for acute adrenal insufficiency, high-risk period.
- Stress dose replacement may be required several days postop.
- Assess pts for complications of steroid use; ulcers, infection, poor wound healing, glucose intolerance.

Anticipated Problems/Concerns

- Previously undiagnosed Addison disease presenting as unrecognized acute adrenal insufficiency resulting in a delay of adequate management.
- Vasopressor-resistant hypotension, acute mental status changes; confusion, lethargy, coma, delayed emergence.
- Refractory hypotension should alert clinicians toward adrenal insufficiency.
- Glucocorticoid replacement and supportive care are the mainstays of treatment in periop period.