

**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<sub>2</sub>O for 40 s) followed by increased PEEP setting.

**Postoperative Period**

- Continued careful monitoring of hemodynamic and volume status.
- Reassess ventilator settings and reduce FiO<sub>2</sub> 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 regimens

**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<sup>+</sup>, K<sup>+</sup>, 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.