

# Alcohol Abuse

## Risk

- Incidence in USA: 10% of Americans, incl physicians, will abuse alcohol at some point in their lives.
- 1:5 surgical pts has some form of alcohol use disorder.
- Third leading cause of death and disability, incl 30% of traffic fatalities.
- Male gender and family Hx major are risk factors.

## Perioperative Risks

- Severe malnutrition as significant as ETOH-induced end-organ injury.
- Risk of Htn, CVA, diabetes, GI disease.
- Liver is the most severely affected organ.
- Dilated cardiomyopathy.
- Withdrawal symptoms can be life threatening.

## Worry About

- Concomitant use of other drugs: Amphetamines, cocaine, benzodiazepines.
- Affects of chronic smoking, such as COPD and emphysema.

- Vasopressor effect of ETOH may cause Htn.
- Acute withdrawal and delirium tremens are life-threatening complications. Symptoms caused by sympathetic stimulation can range from mild tremors to electrolyte disturbances, seizures, and death.

## Overview

- Disease characterized by addiction (compulsion and craving despite consequences) to alcohol.
- Clinical syndromes related to direct effect of ETOH and secondary adaptive response to excess ETOH exposure.
- ETOH rapidly absorbed and metabolized.
- Hepatic dysfunction usually takes 10 to 15 y to develop.
- Cirrhosis may develop after 1 or more acute episodes.

## Etiology

- Unknown: Likely multifactorial with environmental, genetic, and psychosocial components

## Usual Treatment

- Recovery involves some or all of the following:
- Detoxification: Inpatient, residential, day treatment, or outpatient.
  - Evaluation for comorbid psychiatric disorder.
  - Referral to Alcoholics Anonymous or other alcohol programs.
  - Pharmacotherapy to help with withdrawal and prevent relapse:
    - Disulfiram (Antabuse): Acetaldehyde dehydrogenase inhibitor.
    - Naltrexone (Revia): Pure opioid receptor antagonist, blunts ETOH's pleasurable effects and reduces craving. Available as monthly IM depot. May interfere with opioids prescribed for periop pain.
    - Acamprostate (Campral): A synthetic derivative of homotaurine, a structural analog of GABA. Decreases excitatory glutamatergic neurotransmission during alcohol withdrawal.

## Assessment Points

System	Effect	Assessment by Hx	PE	Test
CV	Cardiomyopathy, arrhythmias, hypertension	Orthopnea, nocturnal urination, coughing, and leg swelling	Dyspnea BP lying and standing HR	ECG, ECHO Lytes
GI	Erosive gastritis, decreased lower esophageal sphincter tone, hepatic cirrhosis, acute hepatitis, pancreatitis, fatty liver	Hx of bleeding, easily bruised, anorexia, N/V	Ascites, jaundice Hepatomegaly, "spider" angiomas Abdominal pain Abdominal pain, hepatomegaly	Upper endoscopy, stool guaiac LFTs Serum amylase Mg <sup>2+</sup> , K <sup>+</sup>
ENDO	Gynecomastia, testicular atrophy, irregular menses			
HEME	Leukopenia, anemia, thrombocytopenia			CBC with differential
CNS	Wernicke's syndrome Korsakoff's syndrome Peripheral polyneuropathy Cerebellar degeneration	Amnesia, impaired reasoning	Sixth nerve palsy, ataxia Distal numbness and paresthesias Unsteady gait	MRI or CT scan, CNS exam

**Key References:** Jones K, Neumann T, Spies C: Perioperative management of patients with alcohol, tobacco and drug dependency, *Curr Opin Anaesthesiol* 23(3):384–390, 2010; Moran S, Isa J, Steinemann S: Perioperative management in the patient with substance abuse, *Surg Clin North Am* 95(2):417–428, 2015.

## Perioperative Implications

### Preoperative Preparation

- All pts should be screened for substance use routinely.
- Gastric prophylaxis.
- Blood ETOH and toxicology screen if indicated.

### Monitoring

- Standard ASA monitors.
- Consider invasive monitors for cardiomyopathy, hepatic dysfunction, and/or end-organ compromise.

### Airway

- Consider full stomach in acute intoxication.

### Preinduction/Induction

- Consider long-acting benzodiazepine, barbiturate, or  $\alpha_2$ -adrenergic agonist.
- Anesthetic doses increased in chronic disease.
- Decreased dose in acute intoxication.

- Rapid sequence in acute intoxication.
- Consider Rx of nutritional/metabolic deficiencies.

### Maintenance

- Requirements vary by age, general health, nutrition and hydration states, concomitant disease.
- Acute intoxication may decrease MAC requirement and lower BIS monitoring values.

### Extubation

- Ensure return of airway reflexes.

### Postoperative Period

- Provide adequate analgesia in PACU.
- Anxiety can worsen withdrawal symptoms.
- Withdrawal syndrome may develop within 6 to 8 h; treat based on symptoms—benzodiazepines for agitation and seizures, alpha-2-agonists for autonomic signs and neuroleptics or olanzapine for hallucinations.

- DTs develop in 5% of pts in withdrawal.
- Ten percent mortality secondary to hypotension, electrolyte disturbances, seizures and/or arrhythmias.

### Adjuvants

- Long-term consumption of ETOH impairs hepatic metabolism.
- Short-term consumption inhibits drug metabolism.
- Polyneuropathy is a relative contraindication to regional anesthesia.
- Consider periop clonidine patch.

## Anticipated Problems/Concerns

- Recognition and treatment of withdrawal important, as significant mortality occurs if inadequately treated.

# Allergy

## Risk

- Incidence in USA: 5% of adults are allergic to one or more drugs.
- During surgery, the risk of anaphylaxis is ~1:2500 to 1:20,000 depending on the agent, with a mortality rate of 4%.
- Females > males (1.6:1).

## Perioperative Risks

- Intensity of Sx variable: From an isolated cutaneous eruption to CV collapse and death.
- CV, cutaneous (incl angioedema), resp systems are mostly involved.
- Increased morbidity and hospitalization time if intensive care required.

## Worry About

- Pt's Hx: Knowledge of prior allergic event leads to avoiding drugs or other components involved; however pt may not know.
- Hypotension/shock, bronchospasm, and angioedema may become life-threatening events.

**Overview**

- IgE anaphylaxis (type I immediate hypersensitivity reaction): Adverse response of host; mediated by antibodies, the antigen bridges with two IgE on the surface of basophils and mast cells; can be reproduced if foreign substance is reinjected. However, IgG reactions with complement may manifest similarly.
- Anaphylactoid reactions or histamine release: Describes a clinically indistinguishable syndrome probably involving similar mediators but not mediated by IgE antibody and not necessarily requiring

previous exposure to the inciting substance, associated with vancomycin, benzylisoquinolinium-derived muscle relaxants, but term should be avoided.

**Etiology**

- Clinical history of allergy or perianesthetic allergic reaction considered to put pt at increased risk for a reaction from neuromuscular blocking agents and induction agents

**Usual Treatment**

- Preventive therapy with corticosteroids and antihistamines is of unproven value.

- Severe allergic therapy: Stop antigen, maintain the airway with 100% O<sub>2</sub> and intubate if necessary; discontinue all anesthetic drugs, volume expansion, epinephrine (5 to 10 µg IV boluses as starting doses and titrate upward), antihistamines, β-sympathomimetic in case of bronchospasm, arginine vasopressin and/or norepinephrine for refractory shock, phosphodiesterase inhibitors for RV dysfunction, airway evaluation prior to extubation, ICU observation.

**Assessment Points**

System	Effect	PE	Test
CV	Hypotension, tachycardia, dysrhythmias Pulm Htn Cardiac arrest	BP	ECG PA pressure
RESP	Dyspnea, sneezing Coughing, wheezing Laryngeal edema Fulminant pulm edema Acute respiratory failure	Chest exam	CXR PA cath ETCO <sub>2</sub> ABGs
DERM	Urticaria, flushing Perioral, periorbital edema	Skin exam	

**Key References:** Levy JH, Adkinson Jr NF: Anaphylaxis during cardiac surgery: implications for clinicians, *Anesth Analg* 106:392–403, 2008; Sampson HA, Muñoz-Furlong A, Bock SA, et al: Symposium on the definition and management of anaphylaxis: summary report, *J Allergy Clin Immunol* 115(3):584–591, 2005.

**Perioperative Implications****Preoperative Preparation**

- Prick tests, intradermal testing: Anesthetic drugs (NM blocking agents)
- Most of the allergic reactions are unexpected. In case of established allergy, those drugs or latex should be strictly avoided.

**Monitoring**

- Routine.

- If major anaphylaxis occurs, consider pulm and radial arterial catheterization to guide therapeutic interventions.

**Airway**

- None, except specific care for the asthmatic pt

**Preinduction/Induction/Maintenance/Extubation**

- Slow injection of drugs, use burette for antibiotics. Avoid histamine-releasing drugs in high-risk pts.

**Anticipated Problems/Concerns**

- For each pt who has a periop allergic reaction, consider evaluation 1 mo after with skin testing, antigen-specific IgE level dosage (ELISA).
- Measure tryptase if there is an anaphylactic reaction within 1 to 2 h of reaction, then 24 h later to support diagnosis.
- Latex allergy should be considered. Healthcare workers are at greater risk, and Hx has to be evoked at the preanesthetic evaluation.

## Alpha<sub>1</sub>-Antitrypsin Deficiency

Paul S. Myles

**Risk**

- One of the most common inherited disorders (1 in 2500 in case of European ancestry; uncommon in Asians)
- Less than 10% of individuals with AAT deficiency are currently identified.
- AAT deficiency is the most common genetic cause of liver disease in neonates and children.
- About 1% to 5% of pts with COPD have AAT deficiency.
- Approximately 15% of adults with AAT deficiency develop liver cirrhosis.

**Perioperative Risks**

- Dynamic hyperinflation (air-trapping or auto-PEEP) with positive pressure ventilation, leading to hypotension and CV collapse
- Resp failure
- Hepatic impairment
- Poor wound healing (panniculitis)

**Worry About**

- Missed or incorrect (e.g., asthma) diagnosis
- Liver cirrhosis
- Glomerulonephritis and nephrotic syndrome
- Gastrointestinal complications incl ascites
- Panniculitis
- Vascular disease

**Overview**

- AAT is secreted in the liver as the most abundant of the serine protease inhibitors (serpins), with over 100 genetic variants of AAT identified.
- Panacinar pulm emphysema is the most common manifestation, and is the major cause of disability and death.
- Most commonly presents with slowly progressive dyspnea in mid-life, typically 2 to 3 decades earlier than do smokers with emphysema and normal AAT levels.
- Some pts present with otherwise unexplained hepatic dysfunction.
- Cigarette smoking greatly accelerates the progression of emphysema in AAT deficiency.
- AAT deficiency may present early after birth as neonatal jaundice and hepatitis, in infancy as cholestatic jaundice, or in children as liver cirrhosis or failure.
- AAT deficiency is the most common condition requiring liver transplantation in children.

**Etiology**

- Autosomal recessive disorder; the most common form is associated with allele Z, or homozygous PiZ (ZZ).
- Emphysema results from the unimpeded neutrophil elastase destruction of the lung alveolar basement membranes.

- Liver disease results from the accumulation of unsecreted AAT protein within the hepatocyte.
- Nonsmokers with the homozygous Z phenotype have minimal symptoms and an almost normal life span.
- Serum levels of AAT in the deficiency states are 10% to 15% of normal levels.
- Emphysema develops in most (but not all) individuals with serum levels less than 9 µmol/l; levels greater than 11 µmol/l seem to be protective.

**Usual Treatment**

- Treatment of emphysema: smoking cessation, preventive vaccinations, bronchodilators, supplemental oxygen when indicated.
- Replacement (“augmentation”) therapy with purified AAT or synthetic elastase inhibition to prevent progression of emphysema.
- End-stage lung or liver disease is treated with transplantation.
- Approximately 12% of all lung transplants are performed for emphysema secondary to AAT deficiency.
- Alternative treatments for emphysema include lung volume reduction surgery or endobronchial valves.
- Emerging therapies include recombinant AAT augmentation/leukoprotease inhibitors, retinoic acid receptor agonists, and gene therapies.