Common Medical Illnesses that Affect Anesthesia and Their Anesthetic Management

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Any patient undergoing an office-based anesthetic will require a thorough preoperative evaluation to identify medical illnesses that can affect the anesthetic management. The literature is abundant with information about general anesthetic management of patients with medical illnesses. However, some of such considerations may not be applicable to the open-airway office-based anesthesia. This article addresses common medical illnesses seen in oral surgery offices, with specific focus on the preoperative and perioperative anesthetic considerations of open-airway office-based anesthesia.

HYPERTENSION

Background

Hypertension is a common disease affecting more than 30% of adults in America. The diagnosis of hypertension is having a blood pressure higher than 140/90 mm Hg measured on a minimum of 2 occasions over a 1- to 2-week span.1 Classifications of hypertension are listed in Table 1.

Hypertension is a risk factor for ischemic heart disease, congestive heart failure, cerebral vascular accident, arterial aneurysm, and end-stage renal disease. Essential hypertension is a diagnosis obtained when a true identifiable cause cannot be found, and accounts for more than 95% of diagnoses. Most often there are familial factors and/or biochemical factors leading to the hypertension. Biochemically, hypertension is a result of a disruption in the complex interplay of endogenous vasoconstrictors and the hormones within the renin-angiotensin system. Other major medical contributors in causing and perpetuating hypertension are involved in 5% of cases; this is known as secondary hypertension. The most common cause of secondary hypertension is renovascular disease.
Treatment of hypertension usually begins with lifestyle modifications that include weight loss, moderation of alcohol, physical activity, moderation of dietary salt intake, and smoking cessation. In addition, pharmacologic therapy should be initiated and managed by the primary care physician. Initial therapy for uncomplicated hypertension is a thiazide diuretic such as hydrochlorothiazide. Other classes of medications such as angiotensin-converting enzyme (ACE) inhibitors, Angiotensin receptor blockers (ARBs), β-blockers, or calcium-channel blockers may be added if necessary. Commonly used antihypertensive agents are listed in Table 2. If patients have other comorbidities, specific classes of drugs may be selected for their pleiotropic benefits.

<table>
<thead>
<tr>
<th>Category</th>
<th>Systolic Blood Pressure (mm Hg)</th>
<th>Diastolic Blood Pressure (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>&lt;120</td>
<td>&lt;80</td>
</tr>
<tr>
<td>Prehypertension</td>
<td>120–139</td>
<td>80–89</td>
</tr>
<tr>
<td>Stage 1</td>
<td>140–159</td>
<td>90–99</td>
</tr>
<tr>
<td>Stage 2</td>
<td>≥160</td>
<td>≥100</td>
</tr>
</tbody>
</table>


Table 2
Commonly used antihypertensive drugs

<table>
<thead>
<tr>
<th>Class</th>
<th>Subclass</th>
<th>Generic Name</th>
<th>Trade Name</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diuretics</td>
<td>Thiazides</td>
<td>Hydrochlorothiazide</td>
<td>HydroDiuril</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Metolazone</td>
<td>Lozol</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Bumetanide</td>
<td>Bumex</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Furosemide</td>
<td>Lasix</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Amiloride</td>
<td>Midamor</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Spironolactone</td>
<td>Aldactone</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Triamterene</td>
<td>Dyrenium</td>
</tr>
<tr>
<td>Adrenergic antagonists</td>
<td>β-Blockers</td>
<td>Atenolol</td>
<td>Tenormin</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Metoprolol</td>
<td>Lopressor</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Propranolol</td>
<td>Inderal</td>
</tr>
<tr>
<td></td>
<td>α1-Blockers</td>
<td>Doxazosin</td>
<td>Cardura</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Prazosin</td>
<td>Minipress</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Terazosin</td>
<td>Hytrin</td>
</tr>
<tr>
<td></td>
<td>α- and β-Blockers</td>
<td>Carvedilol</td>
<td>Coreg</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Labetalol</td>
<td>Normodyne</td>
</tr>
<tr>
<td></td>
<td>Central acting</td>
<td>Clonidine</td>
<td>Catapres</td>
</tr>
<tr>
<td></td>
<td>Vasodilators</td>
<td>Hydralazine</td>
<td>Apresoline</td>
</tr>
<tr>
<td>ACE inhibitors</td>
<td></td>
<td>Benzepril</td>
<td>Lotensin</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Captotropril</td>
<td>Capoten</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Enalapril</td>
<td>Vasotec</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Lisinopril</td>
<td>Prinivil</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Ramipril</td>
<td>Altace</td>
</tr>
<tr>
<td>Angiotensin receptor blockers</td>
<td></td>
<td>Candesartan</td>
<td>Atacand</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Irbesartan</td>
<td>Avapro</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Losartan</td>
<td>Cozaar</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Olmesartan</td>
<td>Benicar</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Telmisartan</td>
<td>Micardis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Valsartan</td>
<td>Diovan</td>
</tr>
<tr>
<td>Calcium-channel blockers</td>
<td>Dihydropyridine</td>
<td>Amlodipine</td>
<td>Norvace</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Nicardipine</td>
<td>Cardene</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Nifedipine</td>
<td>Procardia</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Felodipine</td>
<td>Plendil</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Diltiazem</td>
<td>Cardizem</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Verapamil</td>
<td>Tiazac</td>
</tr>
<tr>
<td>Nondihydropyridine</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Anesthetic Management

Preoperative

- Determine adequacy of blood pressure control by asking if the patient checks the blood pressure at home and have knowledge of its average range.
  - Preanesthesia blood pressure can often be elevated, and is noted with increased age. 
- Review pharmacology of the drugs used in control.
  - Often the more severe cases require multiple classes of drugs.
- Perform thorough history and physical examination evaluating the heart, kidney, or eyes for end-organ damage (left ventricular hypertrophy, proteinuria, reduced renal function, and retinopathy).
- Assume that the patient may have some form of ischemic heart disease or renal dysfunction.
- Continue all prescribed home medications; office-based procedures are of low surgical risk.
  - Although ACE inhibitors/ARBs have been associated with intraoperative hypotension with general anesthesia, they are continued for outpatient sedation. Hypotension associated with ACE inhibitors/ARBs is more common with patients experiencing high intraoperative blood loss, hypovolemia, or fluid shifts. In the oral surgery office, these risks are negligible and the hazards of increase in blood pressure would be more concerning when stopping these medications.
- There are no universal guidelines on when to postpone elective procedures, but anecdotally a diastolic pressure higher than 110 mm Hg would be a reason to delay the procedure. A diastolic pressure lower than 110 mm Hg has been shown not to have any increased incidence of postoperative complications.

Perioperative

- Most classes of anesthetic drugs used in the oral surgery office such as benzodiazepines, opioids, propofol, and barbiturates are safe.
- Ketamine is a general anesthetic agent used as an adjunct during office anesthesia. However, its indirect effects of increasing heart rate and blood pressure should deter its use in patients with hypertension and other cardiovascular diseases.
- Pain control with local anesthesia and/or sedation is important in preventing stimuli-induced hypertension.
- Management of persistent hypertension is not addressed in this article. However, drugs to consider in the office would be medications such as hydralazine or labetalol.
- Hypotension may be seen with the use of propofol/barbiturates, opioids, and benzodiazepines. However, the hypotension is usually transient and can be addressed, if necessary, with the administration of intravenous fluid. Sympathomimetic medications such as phenylephrine or ephedrine may be used if intravenous fluids fail to address the problem.
- Local anesthesia is safe, and dosages are known to most practitioners. In patients with poorly controlled hypertension (Stage II or upward), consider eliminating the use of epinephrine if possible. If vasoconstriction is needed, consider a maximum dose of 0.04 mg epinephrine.

CORONARY ARTERY DISEASE

Background

Coronary artery disease (CAD), also known as ischemic heart disease, often manifests itself through angina, acute myocardial infarction (MI), and sudden cardiac death. Risk factors for CAD include male gender, increasing age, dyslipidemia, hypertension, smoking, diabetes, obesity, sedentary lifestyle, and family history.

Angina is retrosternal chest discomfort, pain, pressure, or heaviness caused by imbalance from coronary blood flow and consumption of myocardial oxygen. Pain may radiate to the neck, left shoulder, left arm, jaw, and sometimes the back. When classified as stable angina this pain lasts several minutes and is often triggered by physical exertion, emotional tension, and cold weather. It is best relieved with rest and/or nitroglycerin. Chronic stable angina is pain that occurs with a repeatable level of exertion or stimulation for more than 2 months. Unstable angina is classified as pain at rest, new onset, or increasing in severity or frequency.

Treatment consists of lifestyle changes, pharmacologic therapy, and coronary revascularization. Often patients will be on a statin drug to reduce low-density lipoprotein to less than 100 mg/dL, and this has been shown to aid in secondary prevention. Hypertension is controlled with β-blockers, calcium-channel blockers, or ACE inhibitors. Patients with stable disease should be on low-dose aspirin therapy (commonly 81 mg). If a patient has undergone revascularization with an intracoronary stent then often aspirin and an adenosine diphosphate blocker such as clopidogrel (Plavix) or ticlopidine (Ticlid) are prescribed.
Patients with bare-metal stents will require uninterrupted antiplatelet therapy for a minimum of 6 weeks, whereas patients with drug-eluting stents will require uninterrupted antiplatelet therapy for a minimum of 1 year. Thus if the patient has received these interventions within these time frames the antiplatelets should not be stopped, owing to the risk of stent thrombosis. If possible, surgery should be deferred until after the dual antiplatelet therapy is complete.5

**Anesthetic Management**

**Preoperative**

- A thorough history and physical examination can determine the severity, progression, and any functional limitations caused by CAD.
- Assess if any cardiac clinical risk factors (Box 1) are present, in which case cardiac consultation may be needed for risk stratification.
  - Determine the patient’s functional status using activities of daily living and metabolic equivalents (METs). A patient capable of performing activities of greater than 4 METs is considered to have a good functional capacity (Table 3).
- If there is a positive history for an MI, elective procedures should be delayed at least 6 weeks following the incident and after clearance from the cardiologist.6 This recommendation of 6 weeks of waiting has changed from the previously recommended delay of 6 months after MI.
- Recommendations for timing of elective noncardiac surgery after coronary intervention are listed in Table 4. Any deviations should be carried out in consultation with the cardiologist.
- Using the guidelines published by the American College of Cardiology/American Heart Association, most oral surgery office-based procedures are low risk. If a patient has had coronary revascularization within the past 5 years or has had an appropriate coronary evaluation (ie, physical/chemical stress test or appropriate nuclear imaging) in the past 2 years, with no subsequent deterioration of cardiac status, further cardiac evaluation is not warranted.6
  - If the patient has any clinical risk factors, consultation with the cardiologist is recommended.
- Continue all home medications including antiplatelets.
  - The decision to hold any antiplatelet should be based on the risk of surgical bleeding and discussion with the cardiologist.

**Box 1**

Clinical predictors of increased perioperative cardiovascular risk

<table>
<thead>
<tr>
<th>Major</th>
<th>Intermediate</th>
<th>Minor</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unstable coronary syndromes</td>
<td>Mild angina pectoris</td>
<td>Advanced age (&gt;70 years)</td>
</tr>
<tr>
<td>Acute or recent MI with evidence of important ischemic risk based on</td>
<td>Previous MI based on history or Q waves on</td>
<td>Abnormal ECG (left ventricular hypertrophy, left bundle branch block,</td>
</tr>
<tr>
<td>clinical symptoms or noninvasive study</td>
<td>electrocardiogram (ECG)</td>
<td>ST-T abnormalities)</td>
</tr>
<tr>
<td>Unstable or severe angina</td>
<td>Compensated or previous heart failure</td>
<td>Rhythm other than sinus</td>
</tr>
<tr>
<td>Decompensated heart failure</td>
<td>Diabetes mellitus (particularly insulin dependent)</td>
<td>Low functional capacity</td>
</tr>
<tr>
<td>Significant dysrhythmias</td>
<td>Renal insufficiency</td>
<td>History of stroke</td>
</tr>
<tr>
<td>High-grade atrioventricular block</td>
<td>Supraventricular dysrhythmias with uncontrolled</td>
<td>Uncontrolled systemic hypertension</td>
</tr>
<tr>
<td>Symptomatic ventricular dysrhythmias in the presence of underlying</td>
<td>ventricular rate</td>
<td></td>
</tr>
<tr>
<td>heart disease</td>
<td>Severe valvular disease</td>
<td></td>
</tr>
</tbody>
</table>


- For most oral surgical procedures it is suggested to continue antiplatelets, as the risk of significant bleeding on dual antiplatelets is not well established.7
- In cases of bleeding risk and with cardiology consultation, it is possible to stop the clopidogrel 5 days before the procedure.
while maintaining aspirin. The oral medications should be resumed as soon as possible after the procedure.

### Perioperative

- Prevent any myocardial ischemia by limiting tachycardia, sympathetic nervous system responses, hypotension, arterial hypoxemia, and systolic hypertension.
- Monitor for myocardial injury by electrocardiogram (ECG) monitoring of ST-segment elevations or depression, the most commonly monitored leads being II and V5.

### VALVULAR HEART DISEASE

#### Background

At present, the prevalence of valvular heart disease in the United States is approximately 2.5% and increases with age. Valvular disease is a significant risk factor for perioperative complications during anesthesia. The common valve disorders may cause a pressure-overload problem (mitral/aortic stenosis) or a volume-overload problem (mitral/aortic regurgitation).

Heart murmurs are a common physical finding, and represent turbulent blood flow. During systole, murmurs arise from stenosis of the aortic/pulmonary valves or incompetent closure of mitral/tricuspid valves. Diastolic murmurs usually arise from incompetent aortic/pulmonary valves or stenosed mitral/tricuspid valves. Commonly these murmurs decrease myocardial work, so the patient may develop symptoms such as dyspnea, orthopnea, fatigue, anxiety, diaphoresis, or resting tachycardia. In addition to valvular heart disease, congestive heart failure or cardiac dysrhythmias (especially atrial fibrillation in mitral diseases) are commonly found.

ECG findings include enlarged P waves suggesting left atrial enlargement, and findings of left ventricular hypertrophy that can include increase voltage in precordial waves or deviation of the left axis.

Mitral stenosis is most commonly caused by rheumatic heart disease. Often symptoms arise 20 to 30 years after experiencing rheumatic fever, owing to its slow progression. The stenosis occurs from leaflet thickening, calcifications, and decreased orifice size. The high atrial pressure causes increased pulmonary pressure, the symptoms of which are dyspnea on exertion, orthopnea, and paroxysmal nocturnal dyspnea. Mild mitral stenosis can be treated with diuretics.

Mitral valve prolapse (MVP) is the most common valvular heart disease, affecting 1% to 2.5% of the United States population, and found more often in...
young women. It can be associated with Marfan syndrome, rheumatic carditis, myocarditis, thyrotoxicosis, and systemic lupus erythematosus. Usually MVP is a benign condition, but it can give rise to serious events such as thromboembolic stroke, infective endocarditis, dysrhythmias, and so forth. Clinically a midsystolic click and late systolic murmur are noted. The diagnosis is based on echocardiographic findings.

In addition, there can be mitral regurgitation associated with mitral valve diseases, commonly from papillary muscle dysfunction or ruptured chordae tendineae. The left atrial volume overload from the incompetent valve can lead to slow progression of pulmonary overload. On physical examination, a holosystolic murmur can be heard radiating to the axilla. Asymptomatic patients often do not need treatment; symptomatic patients require surgery for mitral valve replacement.

Aortic stenosis is an increasingly common valvular lesion in the United States. Increasing age results in degeneration and calcification of the aortic leaflets. Patients with congenital bicuspid aortic valves can develop aortic stenosis earlier in life. The stenosis leads to ventricular hypertrophy and increases the risk for MI. Symptoms include angina, syncope, and dyspnea on exertion. About 75% of symptomatic patients will succumb to death within 3 years if their valve is not replaced. A systolic murmur radiating to the neck can be auscultated, mimicking a carotid bruit. Asymptomatic patients are treated with medical management, whereas symptomatic patients should have valve surgery.

Prosthetic valves may be mechanical or bioprosthetic. Mechanical valves are very durable, lasting at least 20 to 30 years, whereas bioprosthetic valves last about 10 to 15 years. Mechanical valves are highly thrombogenic and require long-term anticoagulation. Because bioprosthetic valves have a low thrombogenic potential, long-term anticoagulation is not necessary; however, future replacement valves are required. Mechanical valves are preferred in patients who are young, have a life expectancy of more than 10 to 15 years, or require long-term anticoagulation therapy for another reason such as atrial fibrillation. Bioprosthetic valves are preferred in elderly patients and in those who cannot tolerate anticoagulation. The risk of a thromboembolic event with a mechanical heart valve is 5% to 8%, compared with chronic atrial fibrillation which has an embolic stroke rate of 7% to 15%.

Medical management of valve disease usually requires heart-rate control with β-blockers, calcium-channel blockers, or digoxin. Blood pressure should be controlled with ACE inhibitors or vasodilators. Congestive heart failure can be controlled with diuretics or inotropes.

## Anesthetic Management

### Preoperative

- A thorough evaluation includes assessment of the severity of the disease, degree of impaired myocardial contractility, and presence of any major organ disease. Also, define a patient’s exercise tolerance using the METs scale (see Table 3), as with ischemic heart disease.
- Obtain a cardiology consult if there is any evidence of symptomatic heart disease. In general, asymptomatic systolic clicks or murmurs may not require preoperative cardiology consultation.
- Consider ECG if there is concern for any arrhythmias associated with valvular disease. Be wary of atrial fibrillation in mitral diseases or left atrial abnormalities.
- In general, aortic stenosis carries a high risk for perioperative complications. For patients with anything more than mild, asymptomatic diseases, consider hospital-based surgery.
- If a patient has a prosthetic heart valve or another high-risk condition for endocarditis (Box 2), provide the appropriate antibiotic prophylaxis (Table 5).
- In general, continue all home medications to maintain a controlled rate and normotension.
- Check for diuretic-induced hypokalemia (if applicable).

### Perioperative

In aortic and mitral stenosis the filling time of the chamber is compromised, so these patients generally require a slow heart rate to allow adequate filling.

- Avoid anticholinergics and ketamine.
- Most other intravenous medications are safe.
- Treat tachycardias with short-acting β-blockers.

In aortic and mitral regurgitations a slow heart rate or hypertension accentuates the regurgitation, so bradycardia and hypertension must be avoided.

- Avoid potent narcotic-induced bradycardia.
- Maintain intravascular volume with intravenous fluids.
- Provide good local anesthetic to reduce sympathetic stimulation.
- Avoid ketamine in patients with MVP.
CONGESTIVE HEART FAILURE

Background

Congestive heart failure (CHF) is a complex disease process whereby the heart is unable to fill with or eject blood to meet bodily demands. Etiology most often involves impaired contractions secondary to ischemic heart disease, valve abnormalities, systemic hypertension, and disease of the pericardium or cor pulmonale.

The most common aspect of CHF is systolic heart failure caused by decreased ventricle wall motion, often secondary to ischemic complications. Left heart failure shows signs of pulmonary edema (orthopnea, dyspnea, paroxysmal nocturnal dyspnea), whereas right heart failure results in venous hypertension and peripheral edema. A commonly used classification of heart failure is that of the New York Heart Association (NYHA), and is based on functional status:

- **Class I**: Ordinary physical activity does not cause symptoms
- **Class II**: Symptoms occur with ordinary exertion
- **Class III**: Symptoms occur with less than ordinary exertion
- **Class IV**: Symptoms occur at rest

Symptoms are dyspnea on exertion (an early symptom), orthopnea (typically dry nonproductive cough when supine), paroxysmal nocturnal dyspnea, fatigue, weakness, nausea, and abdominal pain (from liver congestion). On examination, the physician may note tachypnea, “moist” rales (mostly in lung bases), resting tachycardia, third heart sound, cool and pale extremities, right upper quadrant pain, and/or bilateral lower extremity pitting edema.

Typical treatment depends on symptoms and staging of heart failure. The primary treatment is lifestyle modifications consisting of low sodium diet, weight control, smoking cessation, and glycemic control. Patients with heart failure will be under treatment with various classes of cardiac medications. Classes may include ACE inhibitors/ARBs, aldosterone antagonists, β-blockers, diuretics, vasodilators, and statins.

**Anesthesia Management**

**Preoperative**

- It is important to assess precipitating factors for heart failure, and confirm control and management. Heart failure can be one of the most important risk factors for perioperative cardiac morbidity. Common signs and symptoms of poor control can include:
  - Shortness of breath with minimal exertion (use METs criteria)
  - Peripheral edema: look for swollen ankles and feet
  - Orthopnea: ask about the use of 2 or more pillows
  - Fluctuations in body weight
- Obtain cardiology clearance and classification of disease.
- If on diuretics, check potassium and electrolytes for disturbances. Remember the need for frequent urination when administering intraoperative fluids.
- For the office-based patient, continue all medication unless there is concern for diuretic hypokalemia or volume depletion.

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**Box 2**

Cardiac conditions associated with the highest risk of adverse outcome from endocarditis for which prophylaxis with dental procedures is reasonable

- Prosthetic cardiac valve or prosthetic material used for cardiac valve repair
- Previous infective endocarditis
- Congenital heart disease (CHD)a
- Unrepaired cyanotic CHD, including palliative shunts and conduits
- Completely repaired congenital heart defect with prosthetic material or device, whether placed by surgery or by catheter intervention, during the first 6 months after the procedureb
- Repaired CHD with residual defects at the site or adjacent to the site of a prosthetic patch or prosthetic device (which inhibit endothelialization)
- Cardiac transplantation recipients who develop cardiac valvulopathy

a Except for the conditions listed above, antibiotic prophylaxis is no longer recommended for any other form of CHD.

b Prophylaxis is reasonable because endothelialization of prosthetic material occurs within 6 months after the procedure.

Consider only moderate sedation for patients with stable heart failure who can tolerate ordinary activities (NYHA Class I).

Perioperative

- Moderate-sedation drugs are safe and well tolerated.
- Minimize fluctuations in blood pressure and heart rate with gradual titration of sedation and careful selection of anesthetics. Compensated heart failure can easily decompensate.
- Carefully administer intravenous fluids.
- As for other cardiac conditions, avoid medications that have significant hemodynamic effects (eg, ketamine, glycopyrrolate, atropine).

**ATRIAL FIBRILLATION**

**Background**

Atrial fibrillation occurs when multiple areas within the atria depolarize, causing a quivering of the atrium. The atrioventricular (AV) node sporadically reacts, leading to an irregular heart rate usually in the 180s.

Signs and symptoms of atrial fibrillation include palpitations, fatigue, weakness, CHF, and hypotension. The most important consequence is an increased risk for thromboembolic events, which are often prevented by anticoagulation therapy. Long-term anticoagulation is commonly managed with warfarin (Coumadin), a vitamin K antagonist that has many drug interactions and a narrow therapeutic window requiring frequent monitoring. Recently a new oral anticoagulant, dabigatran (Pradaxa), was approved in 2010. Dabigatran is a direct thrombin inhibitor with half-life of 12 to 17 hours; however, there is no available reversal agent apart from plasma replacement products. New-onset atrial fibrillation is treated by either electrical cardioversion (if within 24–48 hours) or pharmacologic cardioversion (if within 7 days). In either case, rate control is necessary and is accomplished with β-blockers, calcium-channel blockers, or digoxin. Clinically the palpable pulse will be “irregularly irregular.”

**Anesthesia Management**

**Preoperative**

- Continue all antiarrhythmic medications.
- Check electrolytes, specifically magnesium and potassium, if the patient is on digoxin.
- Coordinate care with the primary care team for anticoagulation management or transitions if needed for surgical procedures.

**Perioperative**

- Maintain rate control; use β-blockers or calcium-channel blockers if necessary.
- Avoid use of medications that can induce tachycardias (ketamine, glycopyrrolate, atropine).

**PACEMAKERS AND IMPLANTABLE CARDIOVERTER-DEFIBRILLATORS**

**Background**

Pacemakers

Permanently implanted pacemakers are most commonly used for patients with sick sinus syndrome, and are the only long-term treatment...
for symptomatic bradycardia. Improvements in pacemakers now allow for dual-chamber sensing, pacing, and variable response. Pacing modes are designated by a 5-letter generic code of which the first 3 letters give the most important information.

First letter denotes the chamber being paced (A atrium, V ventricle, D dual chamber)
Second letter denotes chamber being sensed (A atrium, V ventricle, D dual chamber)
Third letter denotes the response to sensed signals (I inhibition, T triggering, D both)

For example, DDI pacing paces both chambers, senses both chambers, and only inhibits if it senses a response. The mode can give insight for the reason for pacing, such as AV dyssynchrony, atrial tachyarrhythmias, or sinoatrial node disease.

**Implanted cardioverter-defibrillators**
The implanted cardioverter-defibrillator (ICD) can be used independently from a pacemaker, and provides a shock within 15 seconds of sensing a dysrhythmia. Timely defibrillation is the most important factor in survival from cardiac arrest. ICDs are typically given to patients at significant risk for sudden cardiac death, patients with advanced heart failure (ejection fraction <30%), or patients at high risk for ventricular tachycardia/fibrillation.

**Anesthetic Management**

**Preoperative**

- Focused history and physical examination in coordination with the patient’s cardiologist is necessary.
  - Determine the reason for pacemakers/ICD.
    - Ask about bradyarrhythmias, AV nodal ablation, or lack of escape rhythm
  - Assess current function, and check ECG to determine pacing spikes.
- Battery depletion may be seen through decreases in heart rate, often 10% lower than the initial planned rate (commonly 70–72 beats/min).
- The ECG may not show pacemaker function if intrinsic heart rate is adequate.
- If there is no plan for electromagnetic interference (EMI) such as cautery use, then no modifications are required. If EMI is planned then determining pacemaker or ICD function is important; in coordination with cardiology determine a plan to reprogram the pacemaker, suspend rate-adaptive functions, or suspend antiarrhythmia function.

**Perioperative**

- Monitor ECG for proper functioning of the pacemaker.
- Ensure backup equipment is available in case of unexpected failure.
- There is no evidence that anesthetic drugs alter the stimulation threshold for pacemakers.
- A magnet can be placed externally over the pacemaker to convert it to asynchronous mode. Although this is unnecessary for most office-based procedures, if required its use needs to be coordinated with the patient’s cardiologist.
- Avoid monopolar electrocautery if possible by using a bipolar or harmonic scalpel to reduce any potential damage to the pulse generator.
  - Of note, new shielding reduces problems associated with electrocautery. However, this cannot be guaranteed, so consultation with the patient’s cardiologist and the manufacturer is recommended.

**ACUTE RESPIRATORY INFECTION**

**Background**

Uncomplicated upper respiratory infection (URI), also known as the common cold or infectious nasopharyngitis, is a viral infection that comprises 95% of URIs. Symptoms include sneezing, runny nose, fever, purulent discharge, cough, and general malaise. Noninfectious nasopharyngitis, with a somewhat similar presentation without the fever, often has an allergic or vasomotor origin; diagnosis is based on the clinical history and the variety of symptoms.

**Anesthetic Management**

**Preoperative**

- In pediatric patients there is an increased risk of respiratory complications associated with the copious amount of secretions during a URI, which lead to airway irritability and increase the risk of laryngospasm and bronchospasm.
- It is recommended to delay the procedure in patients with obvious symptoms such as rhinitis, cough, rhonchi, and/or fever, for ideally 6 weeks, but the procedure can be completed once most symptoms have resolved.
- For URI patients who are improving and on the latter end of symptoms, consider proceeding with the procedure. The rationale for this is that it takes 6 weeks for airway
hyperreactivity to resolve, by which point another URI can take effect. For patients who have preexisting airway comorbidity such as asthma or chronic obstructive pulmonary disease (COPD), the procedure should be postponed until they are asymptomatic.

**Perioperative**
- Adequately hydrate with intravenous fluids.
- Manage secretions judiciously, taking care to monitor secretions from the nasopharynx.
- The use of bronchodilators to reduce bronchospasm prophylactically has not been established.
- The use of Afrin (oxymetazoline hydrochloride) or decongestants preoperatively to reduce nasal secretions has not been well studied.

**ASTHMA**

**Background**
Asthma affects 4% to 5% of the United States population and is characterized by chronic airway inflammation, temporary expiratory airflow obstruction, and bronchial mucosal reactivity. Acute exacerbations of asthma are short lived, usually lasting minutes to hours. Signs and symptoms include wheeze, cough (productive or nonproductive), dyspnea, and chest tightness. The greatest risk factor for asthma is atopy. Asthmatic disease can be seen in patients with allergic diseases such as rhinitis, urticaria, and eczema. Symptoms of asthma, which can occur during the day or even at night, may be provoked by exercise, viral infections, inhalant allergens (animal dander, dust, pollen), irritants (smoke, chemicals), changes in weather, strong emotions, stress, and/or menstrual cycles.

The diagnosis of asthma is typically based on quantifying the obstruction using spirometry, commonly using the forced expiratory volume in 1 second. Other classifications may be based on symptoms (Table 6).

Treatment of asthma is based on preventing and controlling bronchial inflammation. Table 7 lists common medications used in the treatment of obstructive airway diseases. There are 2 component strategies in the treatment:
- Control airway inflammation and irritability using inhaled/systemic steroids, antileukotrienes, and theophylline.
- Provide rescue relief during acute phases, often using inhaled β-adrenergic agonists.

**Anesthetic Management**

**Preoperative**
- Preoperative evaluation is needed to determine the severity of the disease and effectiveness of medication. Box 3 lists questions to ask patients to determine the severity of their disease.
  - Poorly controlled disease can be indicated by a recent visit to the emergency room or hospitalization within the past month, increasing use of short-acting inhalers, and/or recent flare-ups requiring oral corticosteroids.

---

**Table 6**

<table>
<thead>
<tr>
<th>Classification of asthma for patients older than 12 years</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Symptoms</strong></td>
</tr>
<tr>
<td>Night-time</td>
</tr>
<tr>
<td>awakennings</td>
</tr>
<tr>
<td>Rescue inhaler use for symptoms</td>
</tr>
<tr>
<td>Interference with daily activities</td>
</tr>
<tr>
<td>Lung function</td>
</tr>
<tr>
<td></td>
</tr>
</tbody>
</table>

*Abbreviations: FEV₁, forced expiratory volume in 1 second; FVC, forced vital capacity.*

Auscultate lungs for wheezing/crepitation and look for use of accessory muscles. Pulmonary function tests can be reviewed; however, in the office-based setting these are unlikely to change management. All medications should be continued throughout the preoperative and perioperative period.16

**Perioperative**

- Before induction, use a short-acting inhaler if the patient has any evidence of wheezing.15 Do not start anesthesia until wheezing has resolved.
- Common office-based medications are safe to use in asthmatics; propofol has bronchodilating effects, ketamine produces smooth-muscle relaxation.16
- Drugs that induce histamine release such as morphine, meperidine, and succinylcholine should be avoided except in emergent situations.17
- Consider the use of a precordial stethoscope to monitor airway resistance.

**SMOKING**

**Background**

Tobacco smokers can have a significant impact on both anesthetic and surgical management. The risk of pulmonary complications can be 5 to 7 times higher in smokers than in nonsmokers. The effects of smoking on the heart and lung are listed in **Box 4**.

The carbon monoxide in smoke preferentially binds hemoglobin, decreasing oxygen delivery.
and shifting the oxygen dissociation curve to the left. These effects are short term, usually 4 to 6 hours, and normal hemoglobin function can return in 48 hours.

Nicotine, another component, has known sympathomimetic effects and also can induce hepatic enzyme function, which can elevate metabolism.15 The half-life of nicotine is 20 to 50 minutes.

The long-term effects of decreased mucociliary function, hyperreactivity of the airway, and reduced immune function caused by smoking can take up to 6 weeks to resolve after smoking cessation.15 Thus short-term cessation before an anesthetic may not be as beneficial as was once thought.

Smoking cessation may be advised through counseling, nicotine replacement therapies, or use of the antidepressant bupropion.

**Anesthetic Management**

**Preoperative**

- If surgery is more than 4 weeks away, smoking cessation should be advised.15
- Immediate cessation before surgery:
  - Can be detrimental owing to increased sputum production, nicotine withdrawal, irritability, restlessness, sleep disturbances, and anxiety15
  - Can improve carboxyhemoglobin levels and improve oxygenation15
- Auscultate lungs and question the patient regarding the degree of dyspnea (if any) with various levels of activity.

**Perioperative**

- Management of the patient who smokes is similar to that for the asthmatic patient.

**CHRONIC OBSTRUCTIVE PULMONARY DISEASE**

**Background**

COPD is a disease process largely related to smoking, hallmarked either by chronic productive cough or progressive exercise limitations. Symptoms include cough, dyspnea, and orthopnea. COPD often can be categorized as chronic bronchitis, emphysema, or a combination of both (Table 8).

Patients with chronic bronchitis, sometimes called “blue bloaters,” often are noted for chronic productive cough and frequent respiratory infections, and can be barrel chested. It is also important to bear in mind that chronic hypoxemia and hypercapnia causes respiratory acidosis, pulmonary hypertension, cor pulmonale (right heart failure), and secondary erythrocytosis.

Patients with emphysema, sometimes called “pink puffers,” are typically thin, often have distant breath sounds, and show hyperlucency on chest radiographs. In emphysema, the alveolar walls and pulmonary capillaries are destroyed. This process reduces oxygen diffusion capacity but does not lead to pulmonary vasoconstriction and its secondary effects.
Smoking cessation is the ultimate goal, as this can alter the progression of COPD. Home oxygen is often used, especially in cases of chronic hypoxemia with arterial oxygen pressure (PaO\(_2\)) less than 55 mm Hg. Oxygen via nasal cannula at 2 L/min can restore PaO\(_2\) to between 60 and 80 mm Hg. Symptomatic relief is accomplished using bronchodilator therapy; however, unlike asthma, anticholinergics may be more effective than \(\beta_2\)-agonists. Inhaled steroids are also used in COPD to reduce airway inflammation. Another common finding is frequent respiratory infections, which are managed with intermittent antibiotic therapy.

**Anesthetic Management**

**Preoperative**

- Determine the severity of exercise tolerance, chronic cough, wheezing, and incidence of respiratory tract infections.
- Risk factors for pulmonary complications include age older than 60 years, American Society of Anesthesiologists (ASA) Class II or higher, smoking, pulmonary disease, surgery longer than 3 hours, and general anesthesia.\(^{15}\)
- Continue all home medications. Table 7 lists common drugs used in obstructive pulmonary diseases.
- Patients may be referred to their pulmonologist for optimization if home medications are ineffective or there is clinical evidence of poor lung function.

**Perioperative**

- Inhalation anesthesia with nitrous oxide should be avoided because of the risk of gas being trapped within the bullae, which can potentially rupture.\(^{15}\)
- Supplemental oxygen can be used, and should be titrated to maintain adequate oxygen saturation.
- Note that in advanced disease, patients adapt to hypercarbia and then rely on arterial oxygen levels to trigger their respiratory drive (hypoxicemic drive). Excess administration of oxygen could lead to bradypnea, especially if under the influence of anesthetic medications.\(^{17}\)
- Opioids should be used cautiously, as central nervous system depression can reduce any hypercapnic respiratory drive and cause prolonged ventilatory depression.
- In severe disease, consider minimal conscious sedation and good local anesthesia.
- Avoid early-morning appointments, as patients can have significantly increased coughing as they clear excess mucus accumulated overnight.\(^{17}\)

**OBESITY AND OBSTRUCTIVE SLEEP APNEA**

**Background**

Obesity is an epidemic with significant comorbidities including cardiovascular, pulmonary, and metabolic diseases. As the age and weight of the United States population increases, the incidence of obstructive sleep apnea (OSA) also increases. Obesity currently is best measured with body mass index, calculated as weight in kilograms divided by height in meters squared.

OSA is characterized by 5 or more episodes of apnea, each lasting 10 seconds or more, or a decrease in oxygen saturation of 4% or more from baseline. The polysomnogram is the gold standard for the diagnosis of OSA. The severity of obstructions is measured by the average number of apnea-hypopnea episodes per hour, the

---

**Table 8**

<table>
<thead>
<tr>
<th>Feature</th>
<th>Chronic Bronchitis</th>
<th>Emphysema</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mechanism</td>
<td>Mucus and inflammation of airway lumen</td>
<td>Loss of elastic recoil</td>
</tr>
<tr>
<td>Dyspnea</td>
<td>Moderate</td>
<td>Severe</td>
</tr>
<tr>
<td>FEV(_1)</td>
<td>Decreased</td>
<td>Decreased</td>
</tr>
<tr>
<td>PaO(_2)</td>
<td>Marked decrease (blue bloater)</td>
<td>Modest decrease (pink puffer)</td>
</tr>
<tr>
<td>PaCO(_2)</td>
<td>Increased</td>
<td>Normal to decreased</td>
</tr>
<tr>
<td>Diffusing capacity</td>
<td>Normal</td>
<td>Decreased</td>
</tr>
<tr>
<td>Hematocrit</td>
<td>Increased</td>
<td>Normal</td>
</tr>
<tr>
<td>Cor pulmonale</td>
<td>Marked</td>
<td>Mild</td>
</tr>
<tr>
<td>Prognosis</td>
<td>Poor</td>
<td>Good</td>
</tr>
</tbody>
</table>

apnea-hypopnea index. Alternatively, a screening tool such as the STOP-BANG questionnaire (Table 9) is helpful in identifying patients at risk and stratifying risk management.

Comorbidities associated with obesity and OSA that require investigation include hypertension, pulmonary hypertension, heart failure, dysrhythmias, MI, stroke, diabetes, nutritional deficiencies, and fatty liver infiltration.

Patients with obesity and OSA are at risk for both difficult mask ventilation and difficult airway intubations. Because of increased abdominal fat the functional residual capacity will be reduced, thus leading to quicker desaturation as a result of decreased reserves. The risk of pulmonary aspiration is increased, owing to incidence of hiatal hernias or acid reflux disease from increased intragastric pressures associated with obesity.

Many OSA patients are treated with nighttime dental appliances or continuous positive airway pressure (CPAP), and some receive upper airway surgery to relieve obstructions. Obesity management includes weight loss, medical therapy, and surgery.

Anesthetic Management

Preoperative

- If OSA is suspected or the STOP-BANG questionnaire suggests high risk, avoid sedation and have the patient undergo a formal assessment. Alternatively, proceed with local anesthesia only if feasible.
- Assess the cardiorespiratory system by conducting a thorough cardiac and respiratory review of systems including symptoms of chest pain, shortness of breath, sleeping position, exertional dyspnea, fatigue, and so forth.
- Assess the risk of pulmonary aspiration by questioning for symptoms of gastroesophageal reflux disease (GERD) (coughing, inability to lie flat without coughing, heartburn).
- Perform a thorough airway examination looking for a short neck, large tongue, small mouth, large tonsils, and/or excessive palatal soft tissues.
- If a cephalometric radiograph is available, measure the distance from hyoid to mandibular plane. A distance greater than 20 mm has been correlated with OSA.
- For OSA patients who are successfully treated using CPAP and whose coexisting illnesses are effectively managed, outpatient sedation can be considered.
  - Keep in mind that patients must be able to use their CPAP machine during the postoperative period.

Perioperative

- Keep the patient in a sitting position to minimize the effects of gravity on airway collapse.
- A dedicated head holder is crucial in helping to maintain a patent airway.
- If possible, use conscious sedation only with anesthetics such as midazolam or dexmedetomidine to limit respiratory depression.
- Avoid opioids, owing to the risk of pharyngeal collapse and respiratory depression.
  - Also must be considered during the postoperative period, as respiratory events can occur even 1 week after anesthesia.
  - It is important to maintain a closely monitored follow-up period and to manage analgesia with nonopioids when possible postoperatively.
  - Adjust medications to ideal body weight (110 lb [50 kg] + 5 lb [2.3 kg] for every inch [2.54 cm] above 5 feet [152.4 cm]); see Table 10.
- Have the patient bring the CPAP machine to the office; use postoperatively if necessary.

Table 9

<table>
<thead>
<tr>
<th>STOP-BANG questionnaire</th>
<th>Yes or No</th>
</tr>
</thead>
<tbody>
<tr>
<td>Snoring: Do you snore loudly</td>
<td>Yes or No</td>
</tr>
<tr>
<td>(louder than talking or loud enough to be heard through</td>
<td></td>
</tr>
<tr>
<td>closed doors?)</td>
<td></td>
</tr>
<tr>
<td>Tired: Do you often feel tired, fatigued, or sleepy during</td>
<td>Yes or No</td>
</tr>
<tr>
<td>the daytime?</td>
<td></td>
</tr>
<tr>
<td>Observed: Has anyone observed you stop breathing during your</td>
<td>Yes or No</td>
</tr>
<tr>
<td>sleep?</td>
<td></td>
</tr>
<tr>
<td>Blood pressure: Do you have or are you being treated for</td>
<td>Yes or No</td>
</tr>
<tr>
<td>high blood pressure?</td>
<td></td>
</tr>
<tr>
<td>Body mass index: &gt;35 kg/m²?</td>
<td>Yes or No</td>
</tr>
<tr>
<td>Age: Older than 50 y?</td>
<td>Yes or No</td>
</tr>
<tr>
<td>Neck circumference: &gt;40 cm?</td>
<td>Yes or No</td>
</tr>
<tr>
<td>Gender: Male?</td>
<td>Yes or No</td>
</tr>
</tbody>
</table>

Three or more Yes answers indicate high risk for obstructive sleep apnea (OSA). Less than 3 indicate low risk for OSA.


DIABETES

Background

Diabetes mellitus (DM) is a result of either an inadequate production of insulin (type 1) or inadequate

---

Table 10

<table>
<thead>
<tr>
<th>Anesthetic Management</th>
<th>Yes or No</th>
</tr>
</thead>
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<tr>
<td>Preoperative</td>
<td></td>
</tr>
<tr>
<td>- If OSA is suspected or the STOP-BANG questionnaire</td>
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<tr>
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<td>- Assess the cardiorespiratory system by conducting a</td>
<td></td>
</tr>
<tr>
<td>thorough cardiac and respiratory review of systems</td>
<td></td>
</tr>
<tr>
<td>including symptoms of chest pain, shortness of breath,</td>
<td></td>
</tr>
<tr>
<td>sleeping position, exertional dyspnea, fatigue, and so</td>
<td></td>
</tr>
<tr>
<td>forth.</td>
<td></td>
</tr>
<tr>
<td>- Assess the risk of pulmonary aspiration by</td>
<td></td>
</tr>
<tr>
<td>questioning for symptoms of gastroesophageal reflux disease</td>
<td></td>
</tr>
<tr>
<td>(GERD) (coughing, inability to lie flat without coughing,</td>
<td></td>
</tr>
<tr>
<td>heartburn).</td>
<td></td>
</tr>
<tr>
<td>- Perform a thorough airway examination looking for a</td>
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<td>short neck, large tongue, small mouth, large tonsils, and</td>
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<td>- If a cephalometric radiograph is available, measure the</td>
<td></td>
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<td>distance from hyoid to mandibular plane. A distance greater</td>
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<tr>
<td>than 20 mm has been correlated with OSA.</td>
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<tr>
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<tr>
<td>CPAP and whose coexisting illnesses are effectively</td>
<td></td>
</tr>
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<td>managed, outpatient sedation can be considered.</td>
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</tr>
<tr>
<td>- Keep in mind that patients must be able to use their CPAP</td>
<td></td>
</tr>
<tr>
<td>machine during the postoperative period.</td>
<td></td>
</tr>
<tr>
<td>Perioperative</td>
<td></td>
</tr>
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<td>- Keep the patient in a sitting position to minimize the</td>
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<tr>
<td>effects of gravity on airway collapse.</td>
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</tr>
<tr>
<td>such as midazolam or dexmedetomidine to limit respiratory</td>
<td></td>
</tr>
<tr>
<td>depression.</td>
<td></td>
</tr>
<tr>
<td>- Avoid opioids, owing to the risk of pharyngeal collapse</td>
<td></td>
</tr>
<tr>
<td>and respiratory depression.</td>
<td></td>
</tr>
<tr>
<td>- Also must be considered during the postoperative period,</td>
<td></td>
</tr>
<tr>
<td>as respiratory events can occur even 1 week after</td>
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<tr>
<td>anesthesia.</td>
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<td></td>
</tr>
<tr>
<td>up period and to manage analgesia with nonopioids when</td>
<td></td>
</tr>
<tr>
<td>possible postoperatively.</td>
<td></td>
</tr>
<tr>
<td>- Adjust medications to ideal body weight (110 lb [50 kg]</td>
<td></td>
</tr>
<tr>
<td>+ 5 lb [2.3 kg] for every inch [2.54 cm] above 5 feet</td>
<td></td>
</tr>
<tr>
<td>[152.4 cm]); see Table 10.</td>
<td></td>
</tr>
<tr>
<td>- Have the patient bring the CPAP machine to the office;</td>
<td></td>
</tr>
<tr>
<td>use postoperatively if necessary.</td>
<td></td>
</tr>
</tbody>
</table>
use of the insulin produced (type 2). Type 1 DM is caused by autoimmune destruction of β cells within the pancreas, resulting in essentially a complete absence of circulating insulin. Patients with type 1 DM require exogenous insulin for survival. Type 2 DM results from defect(s) in the insulin receptors or pathways, making insulin ineffective and secondarily decreasing its production. Type 2 DM accounts for almost 90% of diabetics.

The diagnosis of diabetes requires 1 of the following criteria:
1. Hemoglobin A1c (HbA1c) greater than 6.5%
2. Fasting glucose greater than 126 mg/dL
3. Two-hour plasma glucose greater than 200 mg/dL after 75 g oral glucose tolerance test
4. Symptoms of hyperglycemia with random glucose greater than 200 mg/dL

Symptoms of hyperglycemia can include polyphagia, polydipsia, polyuria, fatigue, and unexplained weight loss.

Acute hyperglycemia causes dehydration, impaired wound healing, and an increased rate of infection and hyperviscosity with thrombogenesis. The possibility of infection (especially skin and soft tissue) and the delay in wound healing result from a reduction in neutrophil number, impaired chemotaxis/phagocytosis, a reduction in capillary volume, a decrease in tensile wound strength, a decrease in fibroblast and collagen synthesis, and an increase in edema.

Chronic hyperglycemia in diabetics has long-term consequences.

- Microvascular complications include nephropathy, peripheral neuropathy, retinopathy, and autonomic nephropathy.
- Macrovascular complications are associated with dyslipidemia leading to cardiovascular, cerebrovascular, and peripheral vascular diseases.

Untreated hyperglycemia can lead to diabetic ketoacidosis (mostly type 1 patients) or hyperglycemic hyperosmolar syndrome (mostly type 2 patients).

Treatment of diabetics is often multifold. Type 2 diabetics often need weight loss, exercise therapy, and antidiabetic agents. Further comorbidities of the disease, or those associated with it, may necessitate other medications such as antihypertensives and statins. Common antidiabetic drugs and insulin preparations are listed in Tables 11 and 12.

It is important to remember that the stress response of anesthesia and surgery creates a hyperglycemic challenge through activation of the sympathetic nervous system. This process can turn a well-controlled diabetic into one with hyperglycemia or turn a poorly controlled diabetic into one with severe metabolic effects, including ketoacidosis.

### Anesthetic Management

#### Preoperative

- The following areas should have a complete physical examination and review of systems, as they have implicit effects on the anesthetic management:
  - Cardiac
    - Be wary of silent ischemia in chronic diabetics and keep a high suspicion for myocardial disease
    - Patients should be on β-blockers if CAD is present
    - Patients with concomitant renal disease should be on ACE inhibitors
    - Autonomic neuropathy predisposes patients to dysrhythmias and associated hypotension
  - Renal
    - Maintain adequate hydration
    - Avoid nephrotoxins
    - Maintain renal blood flow with adequate perfusion from fluid and blood pressure
  - Neurologic
    - Gastroparesis is common, thus patients can have an increased risk of aspiration
    - In chronic diabetes, autonomic dysfunction may lead to hyperglycemic unawareness
  - Musculoskeletal
    - Patients may have limited joint mobility of the neck secondary to glycosylation of proteins within the joints
    - This can result in increased difficulty with intubation

#### Table 10

<table>
<thead>
<tr>
<th>Total Body Weight</th>
<th>Lean Body Weight</th>
</tr>
</thead>
<tbody>
<tr>
<td>Propofol: loading dose</td>
<td>Propofol: maintenance dose</td>
</tr>
<tr>
<td>Midazolam</td>
<td>Fentanyl</td>
</tr>
<tr>
<td>Succinylcholine</td>
<td>Remifentanil</td>
</tr>
</tbody>
</table>

Ask the patient to put palms of hands together (Prayer sign) and assess if the palms of the hands are touching. If the palms are unable to touch because of stiff joints, be wary of limited neck mobility. Question the patient on history of hypoglycemia at night, in the morning, or with missed meals, as this will give insight regarding any necessary modification of medication regimens. In general, modification of a patient’s medication regimen for diabetes is based on whether a normal diet can be resumed that day. If a procedure is short and the patient can resume normal oral intake, minimal modifications are necessary.22

- The medications of concern that require modifications are those that can directly cause hypoglycemia (sulfonylureas, meglitinides, insulin preparations)
- These medications may need to be held until after the patient resumes normal intake

### Noninsulin medications
- All medications can be taken the day before surgery and on the morning of surgery; however:
  - Consider holding sulfonylureas, meglitinides, and noninsulin injectables on the morning of surgery, owing to potential risk of hypoglycemia.22

---

### Table 11: Common noninsulin antidiabetic medication

<table>
<thead>
<tr>
<th>Drug Class</th>
<th>Generic (Trade) Name</th>
<th>Mechanism of Action</th>
<th>Half-Life (h)</th>
<th>Side Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Biguanides</td>
<td>Metformin (Glucophage)</td>
<td>Decreases hepatic gluconeogenesis, increases insulin sensitivity</td>
<td>6–18</td>
<td>Diarrhea, nausea, vomiting, lactic acidosis (avoid in renal, liver, and congestive heart failure patients)</td>
</tr>
<tr>
<td>Sulfonylureas</td>
<td>Chlorpropamide (Diabenese) Tolbutamide (Orinase) Glimperide (Amaryl) Glipizide (Glucotrol) Glyburide (DiaBeta, Micronase)</td>
<td>Stimulate insulin secretion, decrease insulin resistance</td>
<td>2–10</td>
<td>Hypoglycemia, gastrointestinal disturbance</td>
</tr>
<tr>
<td>Meglitinides</td>
<td>Repaglinide (Prandin) Nateglinide (Starlix)</td>
<td>Stimulate pancreatic insulin secretion</td>
<td>1</td>
<td>Hypoglycemia (less than sulfonylureas)</td>
</tr>
<tr>
<td>Thiazolidinediones</td>
<td>Rosiglitazone (Avandia) Pioglitazone (Actos)</td>
<td>Regulate carbohydrate and lipid metabolism, reduce insulin resistance, and hepatic glucose production</td>
<td>3–8</td>
<td>Fluid retention, increased cardiac risk, hepatotoxicity.</td>
</tr>
<tr>
<td>α-Glucosidase Inhibitors</td>
<td>Acarbose (Precose) Miglitol (Glyset)</td>
<td>Reduce intestinal absorption of ingested glucose</td>
<td>2–4</td>
<td>Gastrointestinal irritation, flatus</td>
</tr>
<tr>
<td>Dipeptidyl Peptidase-4</td>
<td>Sitagliptin (Januvia) Saxagliptin (Onglyza)</td>
<td>Reduce breakdown of gastrointestinal hormones (incretins), enhance insulin secretion, decrease glucagon</td>
<td>8–14</td>
<td>Infection</td>
</tr>
<tr>
<td>Noninsulin Injectables</td>
<td>Exenatide (Byetta) Pramlintide (Symlin)</td>
<td>Suppress glucagon secretion and hepatic glucose production, suppress appetite, delay gastric emptying</td>
<td>6–10</td>
<td>Nausea, vomiting, weight loss</td>
</tr>
</tbody>
</table>

Metformin has a concern for lactic acidosis; however, there seems to be a lack of evidence regarding this problem unless the patient has renal dysfunction or use of intravenous contrast. If there is a concern for renal dysfunction, hold metformin 24 to 48 hours before surgery.

Insulin medications

- Insulin doses on the day before surgery do not require change unless the patient suggests a history of hypoglycemia at night, in the morning, or without meals (Table 13).
- On the morning of surgery all rapid-acting and short-acting insulin should be held. Intermediate-acting insulin should be reduced to 50% to 75% and long-acting insulin, if taken in the morning, can be reduced to 75%.
- Check blood sugar on initial office visit and on the day of surgery.
- If the patient is hyperglycemic, try to determine if it is from poor long-term control or poor medication management preoperatively.
- There is no consensus or evidence to suggest that preoperative blood glucose is necessary for elective procedures.
- If a patient is hyperglycemic, practitioners can consider treating the patient with a rapid-acting insulin to lower blood sugar to less than 180 mg/dL before proceeding.
- Ask about recent HbA1c.
- Evidence suggests that HbA1c less than 7% has reduced incidence of infections and hospital stays.

Preoperative hyperglycemia management

- Typically, 1 unit of insulin will lower blood glucose levels by approximately 25 to 30 mg/dL.
There are many methods to approximate an appropriate correction factor for insulin. One recommendation is the “rule of 1500” for surgical patients.

- Take 1500 and divide it by the total number of units of insulin taken daily by the patient. The resulting correction factor is the expected decrease in blood glucose with each unit of insulin.
- With an appropriate correction factor, administer the amount of short-acting or rapid-acting insulin required to obtain a serum glucose level of less than 200 mg/dL.
- If the patient has never taken insulin before (insulin-naive type II DM), a sliding-scale dose regime with rapid-acting insulin can be used without any detrimental effects.
- It is recommended to use rapid-acting insulin for correctional doses as opposed to regular insulin because the peak blood level occurs earlier, thus reducing the observation time; this helps prevent “stacking” of correctional insulin doses.

**Table 13**

<table>
<thead>
<tr>
<th>Insulin Regimen</th>
<th>Day Before Surgery</th>
<th>Day of Surgery</th>
</tr>
</thead>
<tbody>
<tr>
<td>Insulin pump</td>
<td>No change</td>
<td>Use sick day or sleep basal rates</td>
</tr>
<tr>
<td>Long-acting peakless</td>
<td>No change</td>
<td>75%–100% of morning dose</td>
</tr>
<tr>
<td>Intermediate acting</td>
<td>No change in daytime dose</td>
<td>50%–75% of morning dose, 75% of dose if taken in evening</td>
</tr>
<tr>
<td>Fixed combinations</td>
<td>No change</td>
<td>50%–75% of intermediate acting (may have to substitute NPH)</td>
</tr>
<tr>
<td>Short and rapid acting</td>
<td>No change</td>
<td>Hold the dose</td>
</tr>
<tr>
<td>Noninsulin injectables</td>
<td>No change</td>
<td>Hold the dose</td>
</tr>
</tbody>
</table>


- Check serum glucose hourly until discharge (coincide serum glucose checks with the peak effects of any correctional insulin administered).
- To provide adequate nausea prophylaxis, dexamethasone may be considered, although the practitioner should be prepared to manage elevated blood sugar over the next 4 hours.
  - If not needed to prevent postoperative inflammation, suitable nausea prophylaxis may be achieved with 4 mg dexamethasone, and will result in less elevation of blood sugar in comparison with 8 mg.22

**CHRONIC LIVER DISEASE**

**Background**

Chronic liver diseases are further classified based on etiology such as viral hepatitis, autoimmune hepatitis, drug-induced hepatitis, and alcoholic hepatitis. Symptoms of chronic liver diseases may be minimal but can be complicated with malaise, jaundice, ascites, or cirrhosis. Common physical findings can include fetor hepaticus, spider angiomas, engorged abdominal veins (caput medusa), hemorrhoids, enlargement of the liver and spleen, palmar erythema, asterixis, testicular atrophy, or physical wasting, among others.

The most common causes of chronic hepatitis are alcoholic liver disease and hepatitis C infection.24 A diagnosis of liver disease should warrant an investigation for chronic alcohol use, substance abuse, exposure to toxic materials, or history of blood transfusions. Viral hepatitis (hepatitis A, B, C, D) is most often diagnosed on serologic testing, and treatment is only for symptom relief. Hepatitis A is most commonly associated with acute hepatitis, whereas hepatitis B and C usually develop into chronic disease.
Patients with cirrhosis and end-stage liver diseases should be adequately investigated for other organ diseases such as hepatic encephalopathy, pleural effusions, hepatopulmonary syndrome, hepatorenal syndrome, portal hypertension, cirrhotic cardiomyopathy, and coagulation disorders. These groups of patients are not recommended for outpatient sedation, although local anesthetic techniques can be used after conferring with a physician. In cases of severe cirrhosis, oxygenation must be monitored because of the possibility of portal hypertension causing shunting and elevating the diaphragm, leading to decreased functional residual capacity.

**Anesthetic Management**

**Preoperative**

- A review of symptoms of liver disease should include fatigue, malaise, nausea, vomiting, hematemesis, pruritus, jaundice, easy bleeding or bruising, hemorrhagic diathesis, abdominal distention, behavioral changes, or altered mental status.
- If a patient has a remote history of hepatitis but no active signs or symptoms of liver disease, outpatient sedation can be considered without further workup.
- Risk stratification for patients with liver disease is best accomplished using the Child-Pugh score (Table 14), evaluating the patient’s current condition, and through consultation with patient’s physician.
- In patients with chronic disease and/or history of bleeding, it is recommend to obtain a baseline complete blood count (CBC) and coagulation profile (prothrombin time, partial thromboplastin time, international normalized ratio), as this will assist in surgical workup as well.
- Consider proceeding with surgery in Child-Pugh Class A patients after consultation with the patient’s physician regarding office-based anesthesia and surgery. The remainder of patients with Child-Pugh Class B, C, and advanced liver disease are better served in a hospital setting.

**Perioperative**

- In liver disease, there is a decreased ability to metabolize drugs, leading to a lower anesthetic requirement and a longer half-life of drug.
- In general, benzodiazepines require a longer recovery, as they are metabolized by cytochrome P450 enzymes.
  - Single doses of midazolam may be acceptable but should be used cautiously, as some studies suggest it can be less effective.
- Propofol is metabolized by the liver through conjugation to glucuronide and sulfate, as well as undergoing extrahepatic metabolism through the lungs.
  - Chronic liver disease has been reported to have no significant alteration in the pharmacokinetic profile of propofol.
- Ketamine should be avoided because of concerns for the buildup of secondary metabolites and reduced elimination.
- Most opioids undergo hepatic metabolism.
  - Morphine, oxycodone, and meperidine have prolonged elimination half-lives and active metabolites, and should be avoided in outpatient sedation in this patient population.

<table>
<thead>
<tr>
<th>Table 14</th>
<th>Child-Pugh scoring system</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Sign of Hepatic Dysfunction</strong></td>
<td>1 Point</td>
</tr>
<tr>
<td>Encephalopathy</td>
<td>None</td>
</tr>
<tr>
<td>Ascites</td>
<td>Absent</td>
</tr>
<tr>
<td>Bilirubin (mg/dL)</td>
<td>&lt;2</td>
</tr>
<tr>
<td>Albumin (g/dL)</td>
<td>&gt;3.5</td>
</tr>
<tr>
<td>International normalized ratio</td>
<td>&lt;1.7</td>
</tr>
<tr>
<td><strong>Points</strong></td>
<td><strong>Class</strong></td>
</tr>
<tr>
<td>5–6</td>
<td>A</td>
</tr>
<tr>
<td>7–9</td>
<td>B</td>
</tr>
<tr>
<td>10–15</td>
<td>C</td>
</tr>
</tbody>
</table>

Fentanyl is well tolerated and does not have a significantly prolonged half-life. Modern volatile anesthetics tend to have a low hepatic metabolism and appear to be a good alternative. This includes the use of nitrous oxide, which has minimal metabolism within human tissues.

Most amide local anesthetics are metabolized by the liver; however, articaine is an alternative, as it has plasma esterase-based clearance.

**GASTROESOPHAGEAL REFLUX DISEASE**

**Background**

The primary cause of GERD is a decrease in resting tone of the lower esophageal sphincter. A reflux of gastric contents into the esophagus causes retrosternal discomfort commonly known as heartburn. The gastric content can also reach the pharynx, causing coughing, pharyngitis, morning hoarseness, and so forth. More than one-third of healthy adults experience reflux at least once a month.

Patients with GERD are at increased risk for aspiration. Common complications include esophagitis, esophageal stricture, laryngitis, bronchitis, bronchospasm, pneumonia, and pulmonary fibrosis.

Treatment modalities include use of H2-antagonists and proton-pump inhibitors (PPIs), which aim to increase gastric pH. Often patients self-medicate using over-the-counter antacids, which can cause rebound gastric acid production if stopped acutely, leading to worsened symptoms.

**Anesthetic Management**

**Preoperative**

- Include in the history and physical examination questions regarding any history of heartburn.
- Poor compliance or frequent as-needed dosing suggests poor control of the disease process.
- Determine the presence of other comorbidities, such as obesity, pregnancy, or diabetes, which could further increase the risk of aspiration.
- Continue home medications on the morning of surgery.

**Perioperative**

- Confirm fasting status using ASA guidelines (Table 15).
  - Anecdotally, consider extending fasting 1 to 2 hours if the patient carries higher risk.

<table>
<thead>
<tr>
<th>Ingested Material</th>
<th>Minimum Fasting Period (h)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clear liquids</td>
<td>2</td>
</tr>
<tr>
<td>Breast milk</td>
<td>4</td>
</tr>
<tr>
<td>Infant formula</td>
<td>6</td>
</tr>
<tr>
<td>Nonhuman milk</td>
<td>6</td>
</tr>
<tr>
<td>Light meal</td>
<td>6</td>
</tr>
<tr>
<td>Heavy meal</td>
<td>8</td>
</tr>
</tbody>
</table>

Examples of clear liquid include water, fruit juices without pulp, clear tea, and black coffee. With nonhuman milk, similar to solids, consider the amount ingested when making a decision. Light meal consists of toast and clear liquids. Fatty foods considerably delay gastric emptying time.

Data from American Society of Anesthesiologists Committee. Practice guidelines for preoperative fasting and the use of pharmacologic agents to reduce the risk of pulmonary aspiration: application to healthy patients undergoing elective procedures: an updated report by the American Society of Anesthesiologists Committee on Standards and Practice Parameters. Anesthesiology 2011;114(3):495–511.

- There is no evidence for the routine use of pharmacologic therapy to decrease aspiration risk in otherwise healthy individuals.
- For high-risk patients (diabetic, obese, pregnant), consider nonparticulate sodium citrate and a gastrokinetic agent such as metoclopramide.
- Avoid supine or Trendelenburg positions.
- If considering premedication for a high-risk GERD patient, H2-blockers or PPIs are effective choices.
  - H2-blockers: ranitidine or famotidine should be given at least 1 hour before planned procedure
  - PPI should be administered orally the night before in addition to the morning of the procedure

**COCAINE ABUSE**

**Background**

Cocaine is an indirectly acting sympathomimetic amine that works by blocking presynaptic uptake of norepinephrine and dopamine, increasing their levels in the synapse. It can be administered by multiple routes such as transmucosal, inhalation, and intravenous. Cocaine is metabolized by plasma cholinesterases within 2 hours of administration.

Acute administration causes coronary vasospasm, which can precipitate myocardial ischemia, myocardial infarction, ventricular dysrhythmias, hypertension, tachycardia, seizures,
and hyperthermia. The use of cocaine can sensitize a patient for myocardial ischemia and hypertension for as long as 6 weeks after discontinuation. Chronic cocaine abuse can lead to nasal septal erosion, depression, paranoid delusions, headaches, seizures, hyperpyrexia, lung damage, and pulmonary edema.

**Anesthetic Management**

**Preoperative**

- Determine last use of cocaine and severity of use.
  - Be wary, as many patients are not truthful regarding substance abuse
- Consider a 12-lead ECG if there is concern regarding recent use or if the patient’s history suggests cardiac disease.
- Urine testing can be done; however, this can detect cocaine and its metabolites for up to 6 days or even 10 days for chronic users.
- At present there is no consensus on the timing of treatment. Anecdotally, 8 hours is sufficient for general anesthesia if the patient is stable.
  - For office anesthesia, consider waiting 24 hours or consider treatment in a hospital setting

**Perioperative**

- Be prepared to handle hypertension or cardiac dysrhythmias.
  - Avoid use of β1-selective β-blockers, as there is concern for unopposed α-stimulation
  - Consider the use of labetalol or adding an α-blocker
- Benzodiazepines, opioids, and propofol are considered safe.
- Do not use ketamine, as it is an indirect sympathomimetic and could further worsen cardiac symptoms.
- Monitor use of local anesthetics containing epinephrine, as there is potential for dysrhythmic effects with both agents in conjunction.

**EXOGENOUS STEROIDS**

**Background**

The exogenous administration of glucocorticoids results in secondary adrenal insufficiency as in the adrenal glands innate secretion is repressed via the hypothalamic-pituitary axis (HPA). Cortisol is responsible for approximately 95% of the adrenal glands’ glucocorticoid activity, and participates in far-ranging processes from carbohydrate and protein metabolism and fatty acid mobilization to electrolyte and water balance, in addition to modulating inflammatory response. Daily estimates of cortisol production range from the equivalent of 15 to 25 mg/d of hydrocortisone to 5 to 7 mg/d prednisone. In response to surgery, the level of cortisol production increases from 2 to 10 times that of baseline.

Under similar surgical stress stimuli, patients who have adrenal suppression produce less cortisol than those who are not suppressed. Manifestations of adrenal crisis include dehydration, hypotension, and shock. However, as most exogenous steroids are glucocorticoid, the mineralocorticoid aldosterone functions remain intact. Therefore symptoms associated with exogenous steroid adrenal insufficiency are often less severe: hypoglycemia, weakness, gastrointestinal complaints, and evolving hypotension.

Clinical factors associated with activation of the HPA and adrenal insufficiency/crisis consist of length and extent of surgery, type and depth of anesthesia, perioperative pain control, and infections. The use of appropriate local anesthesia, postoperative analgesics, and good sedation all have been shown to control increases in cortisol production.

Patients taking less than 5 mg/d prednisone in a morning dose for any length of time do not demonstrate clinically significant HPA suppression. Any patient who has received the equivalent or greater than 20 mg/d prednisone for more than 3 weeks within the past year is assumed to have adrenal suppression. However, the onset of adrenal suppression can occur as early as 1 week after commencing corticosteroid therapy. Doses between 5 and 20 mg prednisone equivalent for longer than 3 weeks may harbor HPA suppression. Patients who receive more than 2 g/d of topical steroids or more than 1.5 mg/d beclomethasone equivalent of inhaled steroids on a long-term basis may also be suppressed. The recovery of normal adrenal function can take from 2 days to 9 months, depending on dosage and length of administration.

Of note, the majority of research into which levels of steroid administration lead to adrenal insufficiency has been based on patient response to biochemical testing of the HPA (corticotropin stimulation testing, among others). However, the results of these tests are not necessarily predictive of surgical or anesthetic outcomes. Hence clinical recommendations are probably based on data that are overly sensitive and inclusive, thus the consideration to provide a “stress dose” is likely based on the low risks and minimal side effects of additional steroids.
**Anesthetic Management**

**Preoperative**
- Perform a history and physical examination to determine why a patient is taking steroids, and at what doses and for what period of time.
  - The underlying disease should be evaluated for its stability and potential to affect treatment
  - Evaluate the patient for any side effects they may have experienced
- Be aware of various steroids and equivalents (Table 16) in considering which patients may be suppressed.

**Perioperative**
- Three popular management strategies for perioperative steroid coverage during surgical and dental care are available in the literature.
  - The most common is doubling the morning dose on the day of surgery
  - Administration can be based on the physiologic glucocorticoid production rate associated with the amount of stress a given procedure is likely to produce. Surgeries are categorized into minor, moderate, and major surgical stresses, which are associated with a daily goal of hydrocortisone administration
  - There may be no need for additional coverage as long as the patient has received the baseline dose
    - Although patients may experience lower cortisol levels than would otherwise be generated for a given surgical procedure/stress level, there appears to be little clinical significance
- Ultimately the risk and negative side effects of short-term glucocorticoid coverage in the amounts suggested is low in comparison with the possibility of adrenal crisis during or as a result of an outpatient procedure.
- Practitioners should use their clinical judgment to assess for a need for coverage beyond the patient’s baseline dose.

**EPILEPSY/SEIZURES**

**Background**

The diagnosis of epilepsy represents a broad grouping of neurologic disorders that can be further classified based on the etiology, age at onset, and seizure type, among others. In general, seizures result from excessive neuronal discharge from the brain and can be the result of multiple causes, all of which do not result in a diagnosis of epilepsy. Accordingly, the diagnosis of epilepsy can represent a spectrum of disorders, many of which are associated with syndromes or metabolic/structural disorders that have far-reaching consequences outside the implications of seizures.

Epilepsy has its highest incidence in the very young and the elderly. Although 10% of the population will experience one seizure in 80 years of life, only around 0.4% to 0.7% of the population is currently diagnosed with epilepsy, and there is an overall 3% lifetime prevalence. Epileptic seizures can be divided into generalized and focal seizures. Generalized seizures result in bilateral and generalized electroencephalographic abnormalities, whereas focal seizures are a result of abnormal neuronal networks that are initially localized within one hemisphere and, often, one area of the brain.

Symptoms of seizures can be very broad-ranging across auditory, motor, and sensory boundaries. Signs can range from simply staring off into space or clenching the jaw to convulsing on the floor.

Status epilepticus is an emergency situation that can result in patients who have previously diagnosed seizures and those who do not. It has been defined as a single seizure lasting more than 5 minutes or a series of seizures whereby consciousness is not recovered. Its occurrence has a similar bimodal distribution, occurring in both the very young and very old.

First-line therapy for epileptics is usually pharmaceutical and comes from a group of medications called antiepileptic drugs (AED). If monotherapy fails, further drug combinations are considered. These medications seem to work via increasing neuronal inhibition or decreasing

<table>
<thead>
<tr>
<th>Table 16</th>
<th>Steroid equivalents</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Name</strong></td>
<td><strong>Glucocorticoid Potency</strong></td>
</tr>
<tr>
<td>Cortisone acetate</td>
<td>0.8</td>
</tr>
<tr>
<td>Hydrocortisone</td>
<td>1</td>
</tr>
<tr>
<td>Prednisone</td>
<td>3.5–5</td>
</tr>
<tr>
<td>Prednisolone</td>
<td>4</td>
</tr>
<tr>
<td>Methylprednisolone</td>
<td>5–7.5</td>
</tr>
<tr>
<td>Triamcinolone</td>
<td>5</td>
</tr>
<tr>
<td>Dexamethasone</td>
<td>25–80</td>
</tr>
<tr>
<td>Betamethasone</td>
<td>25–30</td>
</tr>
</tbody>
</table>
excitability. Therapy is usually divided between treatment of generalized and focal therapy, although there are medications that are effective for both.

- Common first-line therapy for generalized seizures includes valproate, lamotrigine, phenytoin, phenobarbital, and ethosuximide.44
- Common first-line therapy for focal seizures includes phenytoin, carbamazepine, and valproate.45

Anesthetic Management

Preoperative

- History and physical examination.
  - Determine current control of seizure activity
  - Type, frequency, and duration of seizures
  - Time period the patient has been seizure free
- Consider hospital-based procedures for patients in whom seizure control is questionable. Such patients can include:
  - Newly diagnosed patient
  - Patient has changed medication regimen
  - Candidate for, or recipient of, vagal nerve stimulators
  - Patient has history of status epilepticus
- All antiepileptic medications should be continued without interruption of dosing schedules.
  - Low AED levels are one of the leading causes of seizures in epileptics43

Perioperative

- Benzodiazepines are safe, and have been shown to have a protective effect through dose-dependent anticonvulsant activity at the \( \gamma \)-aminobutyric acid A receptors.47
  - Flumazenil, a benzodiazepine reversal agent, should be limited to emergencies with epileptic patients, as its use is known to lower the seizure threshold and can elicit seizures.48,49
- Opioids such as fentanyl and morphine can be used, but caution is suggested because in animal models they can increase seizure thresholds but become proconvulsant at higher doses.50–52
  - Meperidine has a strong association with seizure activity and should be avoided in epileptic patients.53
- Propofol appears to raise the seizure threshold and to have a dose-dependent anticonvulsant effect, both alone and in combination with opioids in laboratory settings.54,55
  - Propofol has been found to have a greater effect than methohexital at raising the seizure threshold and shortening seizure duration.56
- Ketamine has been reported to have anticonvulsant effects at doses used for sedation/anesthesia, but may have proconvulsant properties at lower doses.57
- Barbiturates are known to increase the seizure threshold and have been established for treating status epilepticus; however, it has been suggested that methohexital appears to have the least protective effect of this class58 and has been known to activate epileptic foci.59
- Nitrous oxide has been suggested to provoke seizures in animal models, but this has not been demonstrated in humans.59,60
  - Given its widespread use and the rare reporting of it invoking seizure activity, nitrous oxide it is unlikely to provoke them when used alone
- As hypoglycemia can produce seizures, it may be warranted to use intravenous fluids that include glucose/dextrose.
  - Hypoglycemia should also be ruled out in any patient found to be experiencing seizure-like symptoms during sedation, as this is often readily correctable

CHRONIC KIDNEY DISEASE

Background

Chronic kidney disease (CKD) is a progressive and irreversible decline in renal function.61 It is most commonly caused by diabetic nephropathy followed by hypertensive nephrosclerosis. Progression of chronic renal failure is characterized by a steady decrease in glomerular filtration rate (GFR), which is normally at least 90 mL/min without evidence of disease. As glomerular filtration decreases, patients begin to experience the systemic effects of the accumulated nitrogenous waste normally filtered by the kidney. Patients are said to have CKD once GFR drops below 60 mL/min for 3 months or longer. Patients with a GFR of 40 to 60 mL/min are generally asymptomatic, but if filtration drops low enough (<25 mL/min) it can result in uremia/uremic syndrome. Once filtration drops below 15 mL/min, patients are said to have end-stage renal disease (ESRD) and are dependent on dialysis. The manifestations of CKD are broad, as a result of the many systems of the body affected (Box 5).

Systemic manifestations that must be considered with regard to outpatient sedation include anemia, which is associated with CKD progression as a result of decreased erythropoietin production.61–63
In addition, multiple electrolyte abnormalities may precipitate cardiac arrhythmias and can lead to decreased protein binding of certain drugs.\textsuperscript{61,63}

CKD has a profound effect on the cardiovascular system, significantly increasing cardiac morbidity and mortality.\textsuperscript{63} Physiologically, one of the most important effects is increased cardiac output to maintain oxygen delivery in the presence of anemia.\textsuperscript{61,62} Furthermore, sodium retention, induced abnormalities of the renin-angiotensin system, and dysfunction of the sympathetic nervous system result in arterial hypertension.\textsuperscript{61} Accelerated CAD is intimately linked with CKD, and more than 70% of patients with ESRD have CHF and/or ischemic disease.\textsuperscript{61,63}

Initially, treatment is focused on lifestyle modification and control of the underlying disease process that leads to CKD. With progression, patients often receive an ACE inhibitor or ARB. Anemia can be treated effectively with erythropoietin. Once a patient’s GFR begins to drop below 30 mL/min, discussion of transplantation or dialysis begins.

**Anesthetic Management**

**Preoperative**

- Assessment of CKD and ESRD revolves around the control and management of a patient’s disease by ascertaining any symptoms of disease, as listed in Box 5.
  - Evaluate the patient for cardiac and respiratory dysfunction, including evidence of fluid overload or CHF
  - Routine laboratory work such as a CBC and basic metabolic panel should be obtained to evaluate for anemia and electrolyte imbalances
  - If concerned for cardiac dysrhythmias, obtain an ECG and check electrolytes (specifically potassium)
- If the patient is on dialysis, ascertain the dialysis schedule. Often patients receive dialysis at least 3 days a week.
  - Patients on dialysis should be scheduled for dialysis on the day before sedation and the day after
  - Check for AV fistulas in the upper extremities; such extremities should be avoided for placement of blood pressure cuff and intravenous administration
- In general, because of the significant comorbidities associated with CKD, a consultation with the nephrologist is necessary before embarking on outpatient sedation.
  - Recommendations on any perioperative modifications, general medical status, and scheduling modifications should be reviewed

**Perioperative**

- Anesthetics used must be carefully selected to avoid active metabolites and overdosing for renally eliminated drugs.
  - Propofol is not significantly affected by renal impairment and is safe for use in reduced doses\textsuperscript{62,63}
  - Barbiturates, similarly, have unchanged pharmacokinetics but may have increased free barbiturate circulation because of decreased protein binding\textsuperscript{61}
  - Ketamine is minimally altered by CKD. However, it is known to have active
metabolites and has potential for accumulation. Be wary of its sympathomimetic effects, as CKD patients often have underlying cardiac disease.

- Benzodiazepines are metabolized in the liver but are eliminated renally.
  - Diazepam has active metabolites and should be avoided.
  - Short-acting midazolam in reduced dosage can be acceptable.
  - Oral variants for presedation anxiolysis should be avoided, as they are unable to be titrated and could easily lead to oversedation.
- Common opioids in sedation are generally inactivated by the liver or by plasma esterases, and are safe in reduced dosage (fentanyl, alfentanil, remifentanil).
- Fentanyl is safe in reduced doses but approximately 7% is excreted unchanged by the kidneys, suggesting a potential for accumulation.
- Meperidine and morphine have significant active metabolites, and can accumulate.
- Atropine and glycopyrrolate are eliminated by up to 50% in urine and have the potential for accumulation, therefore use in reduced doses if administration is necessary.
- Nitrous oxide is safe for this population, but administration is recommended at less than 50% to allow sufficient additional oxygen in the presence of anemia.
- Fluid administration should be carefully monitored; 250 mL is usually sufficient for short procedures.
  - Lactated Ringer should be avoided, as it contains potassium (4 mEq/L).
  - Use of 5% dextrose or normal saline (0.9% NaCl) is preferable.
- Patients with CKD can often be volume overloaded; however, patients who receive dialysis can often be volume depleted as a result of aggressive therapy.
- Hypotension can be managed with judicious administration of fluid.
- Moderate conscious sedation with good local anesthesia is recommended to limit exacerbation of cardiac or respiratory systems and to limit overall drug exposure/accumulation.
- There are no contraindications to the use of local anesthetics in patients with renal disease. However, dosages should be kept to a minimum owing to potential accumulation.

# DEPRESSION

**Background**

Major depressive disorder is the most common psychiatric disorder, currently affecting between 2% and 4% of the population. Physiologically, depression is thought to result from a decrease in serotonin and norepinephrine (NE) within the brain. However, the mechanism behind this deficiency and the resulting changes remain controversial.

The mainstay of treatment consists of pharmacotherapy and psychotherapy; however, some patients may benefit from electroconvulsive therapy. Pharmacotherapy for depression revolves around the modification of catecholamine or serotonin levels in the central nervous system. Common antidepressants (Table 17) can be categorized by their mechanism of action, as follows: selective serotonin reuptake inhibitor (SSRI), monoamine oxidase (MAO) inhibitor, tricyclic antidepressant (TCA), and atypical antidepressant.

Serotonin syndrome is a potentially life-threatening reaction resulting from elevated levels of serotonin, usually caused by the interactions between multiple drugs that affect the production, release, or metabolism of serotonin. It is characterized by autonomic hyperactivity, neuromuscular abnormalities, and changes in mental status leading to tachycardia, hyperthermia, hypertension, mydriasis, diaphoresis, tremor/clonus, agitation, and delirium. This potentially fatal syndrome can be caused by interactions between the common antidepressant classes (MAO inhibitors, SSRIs, TCAs) with medications used in outpatient sedation such as fentanyl, codeine, meperidine, ondansetron, and metoclopramide.

**Anesthetic Assessment**

**Preoperative**

- Depression is usually not a contraindication for sedation; however, the medications used to treat the patient can interact with and potentiate medications commonly used.
- Laboratory tests or forms of test are not generally required for patients being treated for depression.
- A preoperative interview should ascertain the following:
  - How long the patient has been on treatment
  - Has the patient recently switched medications
  - Does the patient take the medication as scheduled
  - History of any side effects of the medication.
Any interactions with other medications the patient has experienced

Does medication cause drowsiness or sedation

Patients who have been taking TCAs and MAO inhibitors should be queried for a history of palpitations, known arrhythmias, or orthostatic hypotension/syncopal episodes.

Avoid ketamine, ephedrine, and meperidine with TCAs and MAO inhibitors

Use local anesthetics containing epinephrine with caution

Patients with a positive history for arrhythmias or postural hypotension/syncope may require an ECG and/or medical consult before sedation.

**Perioperative**

- Perioperative management is based around awareness of a patient’s current regimen and any recent changes that have occurred.
- Most potential issues in this patient population come from drug interactions, which are listed in the aforementioned drug classes.
- Anesthetic agents should be titrated to effect, as most antidepressants produce some level of sedation at baseline and the effects with sedatives can be synergistic.
- Benzodiazepines and opioids should be administered in smaller doses to gauge response
- There is evidence that patients treated chronically with SSRIs or TCAs may potentiate the effects of propofol on NE and serotonin reuptake at the synapse, propagating its systemic effects and potentially causing disproportionate interaction.67

**THYROID DISEASES**

**Background**

**Hypothyroidism**

Hypothyroidism is more common in women than in men, and affects approximately 10% of adults older than 65 years.21 Primary hypothyroidism can be a result of autoimmune disease such Hashimoto thyroiditis or radioiodine therapy, whereas secondary hypothyroidism is the result of a dysfunctional pituitary axis.

The signs and symptoms of hypothyroidism are consistent with the far-reaching metabolic effects on the body. Symptoms include fatigue, cold intolerance, sleepiness, weight gain, deepening or hoarseness of the voice, and muscle aches.

---

**Table 17  
Antidepressant medications**

<table>
<thead>
<tr>
<th>Drug Class</th>
<th>Generic Name</th>
<th>Mechanism of Action</th>
<th>Adverse Effects</th>
</tr>
</thead>
</table>
| **Selective Serotonin Reuptake Inhibitors**    |                     | Block reuptake of serotonin from synaptic cleft | Few adrenergic, cholinergic, histaminergic, or dopaminergic effects31  
Associated with discontinuation syndrome and serotonin syndrome |
| Fluoxetine                                      |                     |                                              |                                                                  |
| Paroxetine                                     |                     |                                              |                                                                  |
| Sertraline                                      |                     |                                              |                                                                  |
| Citalopram                                     |                     |                                              |                                                                  |
| Escitalopram                                   |                     |                                              |                                                                  |
| Fluvoxamine                                    |                     |                                              |                                                                  |
| **Tricyclic Antidepressants**                   |                     | Block reuptake of serotonin and norepinephrine from synaptic cleft | Anticholinergic effects31,66 (dry mouth, blurry vision, constipation, urinary retention, increased body temperature)  
Sedation  
Postural hypertension  
Discontinuation syndrome |
| Amitriptyline                                   |                     |                                              |                                                                  |
| Nortriptyline                                  |                     |                                              |                                                                  |
| Imipramine                                     |                     |                                              |                                                                  |
| Trimipramine                                   |                     |                                              |                                                                  |
| Doxepin                                        |                     |                                              |                                                                  |
| Clomipramine                                   |                     |                                              |                                                                  |
| Desipramine                                    |                     |                                              |                                                                  |
| Norotiptyline                                  |                     |                                              |                                                                  |
| Protriptyline                                  |                     |                                              |                                                                  |
| **Monoamine Oxidase Inhibitors**               |                     | Prevent breakdown of catecholamines and serotonin | Hypertension with sympathomimetics  
Orthostatic hypotension  
Sedation  
Serotonin syndrome |
| Phenelzine                                     |                     |                                              |                                                                  |
| Tranylcypromine                                |                     |                                              |                                                                  |
| Isocarboxazid                                  |                     |                                              |                                                                  |
| Selegiline                                     |                     |                                              |                                                                  |
Common signs consist of bradycardia in the resting range of 60 to 80 beats/min (compared with a normal range of 72–84), dry skin, coarse or brittle hair, edema (especially periorbital), and difficulty with memory and concentration.\textsuperscript{21,68,69}

Physiologically, hypothyroidism is associated with decreased stroke volume and heart rate, resulting in decreased cardiac output. Peripheral vascular resistance is increased, and if left untreated can lead to reduced myocardial contractility, impaired baroreceptor function, decreased maximal breathing capacity, and decreased ventilatory response to hypercarbia and hypoxia.\textsuperscript{21,68,70–72} Patients with hypothyroidism can be sensitive to anesthetics and medications because of decreased cardiac output, abnormal baroreceptor function, decreased hepatic metabolism, and decreased renal excretion.\textsuperscript{21}

Levothyroxine (Levo-T4) is acceptable therapy for restoring levels of T3 and T4 for most patients, and consistent therapy can reverse or mitigate many of the systemic effects of hypothyroidism.

**Hyperthyroidism**

The overall incidence of hyperthyroidism in the United States is thought to be between 0.05% and 1.3%, and may be as high as 3% in patients older than 80 years.\textsuperscript{68,73} The majority of cases are subclinical in presentation.\textsuperscript{68,73} Usual causes include Graves disease, multinodular goiter, thyroid adenoma, or excessive exogenous thyroid hormones.

Common symptoms of hyperthyroidism consist of nervousness, fatigue, weakness, palpitations, heat intolerance, excessive sweating, dyspnea, diarrhea, insomnia, poor concentration, and oligomenorrhea. Signs consist of weight loss, hair loss, tachycardia, warm moist skin, hyperkinesis, exophthalmos, lid lag, emotional liability, hyperactive tendon reflexes, and, commonly, thyroid enlargement.\textsuperscript{21,68}

Physiologically, hyperthyroidism is associated with hyperdynamic cardiac functioning, possibly due to an increased number of $\beta$ receptors. The result is tachycardia, increased cardiac output, and profound hypertension.\textsuperscript{21,72} In addition, disturbances in cardiac rhythm are frequent, with sinus tachycardia being the most common, but with a significantly increased occurrence of atrial fibrillation.\textsuperscript{21,68,72,74}

Thyroid storm is a life-threatening exacerbation of hyperthyroidism and is a true medical emergency. Thyroid storm has been associated with administration of anesthesia and more commonly with the postanesthesia recovery period, and should be considered highly in the differential diagnosis of extreme anxiety, fever, tachycardia, cardiovascular instability, and prolonged recovery.

Treatment commonly consists of antithyroid medications such as methimazole and Propylthiouracil, which inhibit the synthesis of thyroid hormones. Symptoms are often controlled with a $\beta$-blocker such as propranolol, which controls the anxiety, tachycardia, and palpatations but also reduces the conversion of T4 to T3. Surgical treatments can include radioactive iodine and thyroidectomy. Patients can be left hypothyroid from both thyroidectomy and radioactive iodine treatment, depending on the extent of the gland removed or destroyed.

**Anesthetic Management**

**Preoperative**

- Patients with untreated, uncontrolled, or recently diagnosed thyroid disease are not candidates for outpatient sedation,\textsuperscript{75} and warrant prompt medical evaluation.
- Patients with diagnosed or suspected of having hypothyroidism or hyperthyroidism should be questioned regarding the degree of severity and the ability to control the condition.
- Emergent surgery should take place with the collaboration of a dedicated anesthesia team.
- Patients who have recently begun treatment for hypothyroidism may see rapid improvement in many symptoms; however, physiologically they may not have returned to normal, so consider delaying procedures.
- For example, decreased myocardial function and ventilatory drive takes 3 to 6 months to return to normal after treatment is initiated.\textsuperscript{21}
- Be cautious of patients with goiters, as there is a potential for airway obstruction and increased difficulty with intubation.
- Laboratory studies are not usually necessary in well-controlled patients who are followed regularly by their physicians.
- Consider ECG and medical clearance in patients with arrhythmias including atrial fibrillation, as this can represent refractory hyperthyroidism.

**Perioperative**

**Hypothyroidism**

- Patients with hypothyroidism have increased sensitivity to anesthetics.\textsuperscript{21}
  - Carefully titrate anesthetics to prevent excessive sedation.
Hypothyroid patients have a hypodynamic cardiovascular system, which can be unmasked by surgical stress and/or the cardiovascular depressant effects of anesthetic agents. The cardiovascular effects of hypothyroidism are generally reversed once patients are rendered euthyroid. There is evidence to suggest that patients rendered with subclinical hypothyroidism after treatment may have significant acceleration of cardiovascular disease. Avoid ketamine, atropine, and other medications that can increase the demand on the heart. Patients should maintain their perioperative dosing schedule, including morning doses even while NPO.

Hyperthyroidism
- Patients who have been euthyroid for at least 6 to 8 weeks have no contraindication for anesthesia. Those receiving propranolol should continue to receive their scheduled dose. Persistent signs of increased adrenergic activity beyond that normally associated with surgery and the selected anesthetic technique can be managed with β-blockers such as propranolol, esmolol, metoprolol, and atenolol, but should signal the termination of the surgery.
- Intraoperative hypotension can be managed with fluids, a decreased level of anesthesia, and phenylephrine if necessary.
- Avoid anticholinergic medications such as atropine and glycopyrrolate, as these drugs can precipitate a tachycardia and alter heat regulation.
- Sympathetic nervous system stimulants such as ketamine, ephedrine, and epinephrine should be avoided.
- There are no studies concerning local anesthesia with epinephrine in hyperthyroid patients; however, consider avoiding epinephrine in untreated or poorly controlled patients.
- Nitrous oxide, opioids, and benzodiazepines are generally considered safe for administration.

OPIOID TOLERANCE AND ABUSE

**Background**

Opioid abuse rarely develops from the treatment of immediate postoperative surgical pain. Often, abuse of these drugs is due to their euphoric and analgesic effects. Common routes of administration of opioids are oral, subcutaneous injection, or intravenous injection. Problems from opioid abuse include hepatitis, cellulitis, endocarditis, septic thrombophlebitis, malnutrition, AIDS, and transverse myelitis, among others. Tolerance can develop to the analgesic, sedative, and euphoric effects of opioids, requiring higher concentrations of the drug. However, side effects such as miosis or constipation always remain. Often the tolerance to opioids can lead to opioid-induced hyperalgesia. The magnitude of the phenomenon of clinical opioid tolerance is controversial.

Although withdrawal is rarely life threatening, one must watch out for symptoms in the postoperative setting. Withdrawal symptoms can include sympathetic overactivity (diaphoresis, mydriasis, hypertension, and tachycardia), laceration, piloerection, tremors, insomnia, abdominal cramps, and/or muscle spasms. Opioid dependence is often treated with methadone, levomethadyl, or buprenorphine (Subutex). Buprenorphine can be combined with the antagonist naloxone (Suboxone), which prevents patients from dissolving the tablets and using the drugs intravenously.

**Anesthetic Management**

**Preoperative**
- Patients on preoperative opioids or methadone should continue their prescribed dosages throughout the preoperative period.
- A thorough history and physical examination should be obtained, specifically targeting any complications secondary to opioid abuse.

**Perioperative**
- Avoid any opioid agonist-antagonist or partial antagonist drugs, owing to concern for developing acute withdrawal.
- Patients may have exaggerated postoperative pain, which needs to be managed.
  - Continue home dosages of methadone or opioids
  - Add appropriate narcotics and analgesics (nonsteroidal anti-inflammatory agents) for postoperative pain control
  - Give good local anesthetic and consider long-acting local anesthetics
  - Do not attempt to wean these patients during the perioperative period
  - Continue perioperative and postoperative pain adjuncts such as antidepressants or antianxiety medications
REFERENCES


