Lesson 310: Preanesthetic Assessment of the Patient With Addison’s Disease

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Read this article, reflect on the information presented, then go online and complete the lesson post-test and course evaluation before the termination date below. (CME credit is not valid past this date.) You must achieve a score of 80% or better to earn CME credit.

TIME TO COMPLETE ACTIVITY: 2 hours
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Professional Gaps

Addison’s disease is a well-recognized but seldom encountered disease perioperatively. Recent guidelines have summarized strategies for steroid supplementation based on the results of large studies and evidence-based medicine.

Learning Objectives

At the completion of this activity, the reader will be able to:

1. Describe the adrenal gland and hypothalamic–pituitary–adrenal (HPA) axis.
2. Classify adrenal insufficiency.
3. Describe Addison’s disease.
4. Describe the systemic changes caused by Addison’s disease.
5. Define adrenal crisis.
6. Explain the effects of etomidate as a triggering cause of Addison’s disease.
7. Describe etomidate analogues for possible future use.
8. Apply appropriate preoperative testing and evaluation of the patient suspected to have Addison’s disease.
9. Develop an anesthetic plan for the patient with a previous incident or family history of Addison’s disease.
10. Anticipate, recognize, and manage likely perioperative complications related to adrenal insufficiency disease.
Case History

A 41-year-old man presented to the emergency room with a 2-day history of nausea, vomiting, and pain in his right lower quadrant. Vital signs demonstrated a slight increase in temperature, and lab studies showed leukocytosis with a white cell count of 12.5. Chest radiograph was within normal limits. Acute appendicitis was diagnosed and appendectomy scheduled. The patient gave a 3-year history of Addison’s disease, which was initially diagnosed with weakness, hypotension, and weight loss. The patient also stated he has been stable on his current medications, which included fludrocortisone 0.2 mg per day and prednisone 4 mg twice daily. An electrolyte panel was within normal limits.

Adrenal Gland and HPA Axis

The adrenal gland consists of a cortex or outer regions and a medulla or central region. The cortex is made up of 3 regions: the zona glomerulosa, zona fasciculata, and the zona reticularis, which produce mineralocorticoids (eg, aldosterone), glucocorticoids (eg, cortisol), and sex hormones, respectively. The medulla produces catecholamines, such as epinephrine and norepinephrine. Direct destructive insults to the adrenal gland or to the hypothalamus or pituitary gland can cause adverse effects resulting in disruption in the production or release of these hormones.

The HPA axis controls the overall output of glucocorticoids from the adrenal cortex. Specifically, the hypothalamus releases corticotrophin-releasing hormone (CRH), which stimulates the anterior pituitary gland to produce adrenocorticotropic hormone (ACTH). After release, ACTH stimulates the adrenal cortex to produce cortisol. Cortisol provides negative feedback to both the hypothalamus and pituitary gland to control the release of CRH and ACTH.

Cortisol plays an important role in metabolic and endocrine functions essential for human survival, particularly during stress conditions such as surgery, anesthesia, trauma, severe illnesses or infection, exercise, or burns when an intact or normal functioning HPA axis responds by increasing the release of ACTH and cortisol. The degree of increase in cortisol levels depends on the severity of the stressful event. Normal physiologic cortisol output in a non-stress state may range between a total of 15 and 30 mg per day, whereas a stressful state may cause a normally functioning HPA axis to increase the output of cortisol to 60 to 100 mg/m² per day. Although the adrenal glands are capable of secreting up to 300 mg per day, output rarely exceeds 150 mg even in response to major surgical stress. This HPA axis response to stress is necessary for maintaining proper physiologic functioning.

Cortisol is required for the metabolism of carbohydrates, lipids, and proteins, and for the maintenance of vascular tone and endothelial integrity. Cortisol also facilitates the effects of catecholamines to increase vascular tone, vasoconstriction, and exert positive inotropic effects. Surgery has been found to be one of the most potent activators of the HPA axis. Studies have reported that maximum levels of ACTH and cortisol are reached during the early postoperative period, particularly following reversal of anesthesia and endotracheal extubation. Widmer et al reported that in patients undergoing coronary artery bypass graft surgery, plasma cortisol levels increase significantly during the operation, with peak cortisol levels achieved 30 minutes after extubation. Reports have also shown that ACTH levels return to normal within 24 hours, whereas cortisol levels decline more slowly, reaching high normal values approximately 48 to 72 hours after surgery.
Adrenal Insufficiency

Classification of Adrenal Insufficiency

Adrenal insufficiency (AI) may be classified as primary, secondary, or tertiary. Primary AI (Addison’s disease) is caused by disease, destruction, or malfunction of the adrenal glands. Secondary AI results from disease or insult to the pituitary gland, which reduces production and release of ACTH and leads to atrophy of the adrenal cortex. Secondary AI can stem from iatrogenic causes such as exogenous administration of corticosteroids. Administration of exogenous glucocorticoids suppresses both hypothalamic CRH and pituitary ACTH. In general, patients who receive the equivalent of 20 mg per day of prednisone for more than 5 days are at increased risk for suppression of the HPA axis. Patients treated for approximately 1 month may be at risk for HPA suppression for up to 6 to 12 months after stopping therapy. Receiving an equivalent dose of 5 mg or less of prednisone over any period of time usually will not significantly suppress the HPA axis. Tertiary AI results from hypothalamic disease or may have an iatrogenic etiology. Presenting signs and symptoms of AI typically occur when greater than 80% to 90% of the adrenal gland is destroyed or is nonfunctional.

Addison’s Disease

Primary AI or Addison’s disease results in a deficiency of both cortisol and mineralocorticoid. In 1930, tuberculosis accounted for 70% of cases of Addison’s disease. Today, however, the most common cause is autoimmune adrenalitis, which can be sporadic or familial in nature and can involve other endocrine organs including parathyroid glands, thyroid glands, ovaries, and islet cells of the pancreas. Other less common causes of Addison’s disease include fungal infections such as histoplasmosis, coccidiodomycosis, and cryptococcosis. Primary AI may occur in patients with HIV secondary to dissemination of infectious agents such as cytomegalovirus and Mycobacterium avium-intracellulare. Bilateral adrenal hemorrhage can result from clotting abnormalities triggered by anticoagulant drugs, heparin therapy, coagulopathy, thromboembolic disease, or a hypercoagulable state. Disseminated infections such as meningococcemia (Waterhouse-Friderichsen syndrome), physical trauma, postoperative states, sepsis, and severe stress also may cause bilateral adrenal hemorrhage. Bilateral hemorrhaging of the adrenal glands inhibits adrenal function and therefore may cause primary AI. Additional causes of Addison’s disease include metastases, amyloidosis, sarcoidosis, and drugs such as ketoconazole, rifampin, and etomidate.

Presenting signs and symptoms of Addison’s disease include hypotension; fatigue; weakness; anorexia; nausea and vomiting; weight loss; and hyperpigmentation of the skin, skin creases, and buccal mucosa. Hyperpigmentation occurs as a result of the elevated levels of ACTH found in primary AI and the relationship between ACTH and melanocyte-stimulating hormone. Secondary and tertiary AI result in depressed levels of ACTH and are not associated with hyperpigmentation. Laboratory examination is likely to reveal hypoglycemia, eosinophilia, hypoaldosteronism, hyponatremia, and hyperkalemia. The low aldosterone levels in patients with primary AI cause hyponatremia and water loss with subsequent hypovolemia and hypotension. Other classic laboratory findings in AI are hypercalcemia, azotemia, and mild metabolic acidosis. As reported by Frederick et al, a random serum cortisol level of less than 10 mcg/dL during stress is suggestive of Addison’s disease, and less than 5 mcg/dL is virtually diagnostic. Patients with Addison’s disease also have suppressed levels of 24-hour urine cortisol and 17-hydroxycorticosteroids. Secondary and tertiary causes of AI do not result in hypoaldosteronism because aldosterone production is primarily regulated by the renin–angiotensin system. Electrolyte disturbances are typically not seen in either secondary or tertiary AI.
Generally, patients with Addison’s disease receive routine replacement doses of hydrocortisone ranging from 20 to 30 mg per day in divided doses, primarily administered in the morning. These patients may still develop symptomatic AI when exposed to stressful states such as surgery and the perioperative period due to their inability to sufficiently increase cortisol levels proportionate to the stress.  

Addisonian Crisis

Addisonian crisis, also referred to as acute AI, is a life-threatening emergency. It occurs most often in patients with primary AI, but may also occur in patients with secondary or tertiary AI. A thorough history and physical examination plus a high index of suspicion for Addison’s disease are the most important tools for effective diagnosis of the condition. Addisonian crisis typically is precipitated by the onset of a stressful event in an individual who is unable to mount an appropriate cortisol stress response. Precipitating factors can include surgery, trauma, infection, alcohol withdrawal, and excessive loss of fluid and sodium through sweating or diarrhea.

Glucocorticoids are not stored and must be synthesized by the body on demand. In a surgical patient with a normal functioning HPA axis, prompt elevated production and secretion of cortisol occurs at the onset of surgery. This secretion of cortisol remains elevated for approximately 3 days postoperatively. However, in patients with HPA axis suppression or those who are unresponsive to stress, cortisol secretion fails to occur. Acute failure of cortisol secretion leads to Addisonian crisis. In the perioperative period, Addisonian crisis may present as circulatory collapse and hypotension. In the postoperative period, the diagnosis may be missed or delayed due to nonspecific symptomatic complaints accompanied by a rapid, nonspecific deterioration lacking an obvious underlying etiology. Symptoms include lethargy, weakness, weight loss, fever, diarrhea, nausea, and vomiting, which may be intractable. Confusion, stupor, or coma may occur. In patients with primary AI, electrolyte disturbances, cutaneous and/or mucosal hyperpigmentation and volume depletion secondary to aldosterone deficiency are likely to occur. Hypotension may vary from mild orthostasis to shock. The patient also likely will present with abdominal pain of severity that may be confused with an acute abdomen.

Considerations for Assessment and Management

Preoperative Assessment

Preoperative screening aims to identify patients who are at increased risk for HPA axis suppression, including those who are being treated for diagnosed AI while maintaining a high index of suspicion for patients with undiagnosed or suspected AI. For example, a high index of suspicion should be maintained for patients taking 20 mg or more of prednisone (or the equivalent) for 5 or more days, or patients who exhibit laboratory findings of hyponatremia, hyperkalemia, unexplained hypotension, or eosinophilia. Similarly, a high index of suspicion should be held for patients undergoing chronic corticosteroid therapies for underlying medical conditions such as uncontrolled persistent asthma or severe rheumatoid arthritis. These patients have an increased risk for suppression of the HPA axis and may require additional corticosteroid coverage perioperatively in order to meet stress demands. Patients with undiagnosed primary AI may present with fatigue, weight loss, nausea, vomiting, diarrhea, and hyperpigmentation. Basic or routine laboratory examination may reveal hypoglycemia, electrolyte abnormalities, and eosinophilia. Diagnostic testing of the HPA axis can be performed in the
medically stable patient if one suspects undiagnosed primary AI, but investigation for adrenal suppression is rarely done preoperatively. Furthermore, emergent cases may not permit the time necessary to conduct diagnostic testing.

The anesthesiologist, again, must rely on and maintain a high index of suspicion based on the patient’s preoperative clinical presentation and medical history. Tests to detect perioperative adrenal suppression or to identify patients who will respond to supplemental glucocorticoids have been neither sensitive nor specific. However, the short ACTH stimulation test has been found to reliably assess adrenocortical function. Kohl et al reported that preoperative abnormalities in the short ACTH stimulation test justify supplemental perioperative glucocorticoid administration and that a systematic approach should be taken to determine the necessity of steroid supplementation for those at risk for perioperative adrenal suppression. If the clinician suspects the presence or development of AI and the surgical procedure is emergent, steroids should be administered. However, if there is less urgency and time allows, the findings of Kohl et al suggest conducting an ACTH stimulation test to see if the adrenal gland responds appropriately to supraphysiologic doses of ACTH. In patients who are predetermined to have a high risk for developing perioperative AI, but results from the preoperative ACTH stimulation test are normal, steroids should be administered per guidelines (Tables 1-3) in a stress dose consistent with the level of injury if these patients present preoperatively with unexplained hypotension refractory to volume repletion. These guidelines have been developed to assist in the determination of an appropriate steroid stress dose based on the severity of the surgical procedure. For example, if a patient with presumed or previously diagnosed AI undergoes a minor surgical procedure such as an inguinal hernia repair, guidelines suggest giving a steroid stress dose of 25 mg hydrocortisone IV at induction of anesthesia or the morning of the procedure.

### Perioperative Management

Patients with Addison’s disease require glucocorticoid supplementation perioperatively in order to prevent Addisonian crisis. The dose of corticosteroid supplementation with hydrocortisone or an equivalent should be determined on an individualized basis. This dose should be based on the acuity of the operation and anticipated severity of the surgical procedure.

The literature shows some variation in dosing guidelines. For example, most guidelines support adding 100 to 150 mg hydrocortisone IV for major procedures (Tables 2 and 3). However, Axelrod suggests using methylprednisolone 10 mg IV during surgery and every 8 hours afterward, tapering to baseline
dose in 2 to 3 days for procedures with major operative stress. The use of 10 mg IV methylprednisolone was favored over the higher dose of hydrocortisone because a previous report documented that hydrocortisone at doses greater than 100 mg per day should be avoided due to its significant mineralocorticoid action and high risk for fluid retention.

Overall, supplementation guidelines are similar and maintain the need for additional glucocorticoid coverage tailored to the patient’s need and anticipated surgical severity. In the event that supplementation was not given and Addisonian crisis ensued, Frederick et al provide therapeutic management. The mainstay of emergency treatment of Addisonian crisis is hydrocortisone, which has mineralocorticoid and glucocorticoid effects. An initial dose of 100 mg IV is given and repeated every 6 hours for 24 hours. If after 24 hours progress is satisfactory, the dose is reduced to 50 mg every 6 hours on the second day and then tapered as an oral dose thereafter. Supportive measures are performed, including insertion of large-bore IV catheters and subsequent replacement of volume. Volume replacement initially can be approached by giving 5% dextrose in normal saline at a rate of 500 mL per hour for the first 4 hours. Monitoring fluid volume and predicting fluid responsiveness noninvasively in the mechanically ventilated patient can be successfully done by using the pleth variability index. A blood sample should be drawn to check levels of serum glucose, cortisol, and electrolytes. Hypoglycemia and/or derangements in electrolyte levels need to be treated accordingly.

Other considerations for perioperative management include the effects of certain drugs in patients with known or suspected AI. Etomidate, an anesthetic-sedative drug, is frequently used as an induction agent and also can be used as a sedative by continuous infusion. For patients who are hemodynamically unstable, etomidate is a particularly favorable option. However, etomidate inhibits adrenal mitochondrial hydroxylase activity and cortisol biosynthesis, decreasing steroidogenesis, which may precipitate acute AI or Addisonian crisis.

Etomidate is best avoided in patients with known or suspected AI. In recent years, carboetomidate and methoxycarbonyletoimdate, drugs that are analogues of etomidate but do not cause suppression of adrenocortical function, have been developed for possible future use. Addison’s disease also has a propensity to cause electrolyte disturbances, including hyperkalemia. This effect may be highly important in patients receiving succinylcholine, a depolarizing neuromuscular blocking agent with a side effect of inducing hyperkalemia.
Postoperative Management

During the postoperative period, steroids should be continued until the stress response diminishes.3-5,14 Typically, the supplemental steroid dose is rapidly tapered to the patient’s regular glucocorticoid replacement dose over 24 to 48 hours.3-5,14 Rapid tapering prevents adverse effects such as postoperative infection, gastrointestinal hemorrhage, and delayed wound healing that may result from receiving excessively high corticosteroid dosages.

Management of the Case Presented

A review of the past medical history provided no additional information other than the 3-year history of Addison’s disease. Physical examination demonstrated a Mallampati 1 class airway, clear lungs to auscultation, and a regular heart rate and rhythm. Vital signs were all within normal limits, except for a low-grade fever. An IV route was secured in the emergency room. Risks and benefits were discussed with the patient, including the use of regional or general anesthesia. The patient opted for general

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Table 3. Guidelines for Glucocorticoid Supplementation in Patients With Adrenal Insufficiency

<table>
<thead>
<tr>
<th>Surgical Stress</th>
<th>Glucocorticoid Dosagea</th>
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<tbody>
<tr>
<td>Minimal</td>
<td>Usual replacement dose, 15-30 mg hydrocortisone/d.</td>
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<tr>
<td>• &lt;1 h under local anesthesia</td>
<td></td>
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<tr>
<td>(eg, routine dental work, skin biopsy)</td>
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<tr>
<td>Minor</td>
<td></td>
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<tr>
<td>• Inguinal hernia repair</td>
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<tr>
<td>• Colonoscopy</td>
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<tr>
<td>• Laparoscopic appendectomy</td>
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<tr>
<td>• Dental procedure requiring &lt;1 h under local anesthesia</td>
<td>Double the daily dose of glucocorticoid on day of procedure (eg, 40 mg oral hydrocortisone). Usual replacement dose next day.</td>
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<tr>
<td>(eg, multiple extractions, periodontal surgery)</td>
<td></td>
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<tr>
<td>Moderate</td>
<td></td>
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<tr>
<td>• Open cholecystectomy</td>
<td></td>
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<tr>
<td>• Segmental colon resection</td>
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<tr>
<td>• Lower limb revascularization</td>
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<tr>
<td>• Total joint replacement</td>
<td></td>
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<tr>
<td>• Abdominal hysterectomy</td>
<td></td>
</tr>
<tr>
<td>• Exploratory laparotomy</td>
<td></td>
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<tr>
<td>Severe</td>
<td></td>
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<tr>
<td>• Cardiothoracic surgery</td>
<td></td>
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<tr>
<td>• Whipple procedure</td>
<td></td>
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<tr>
<td>• Esophagectomy, total proctocolectomy</td>
<td></td>
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<tr>
<td>• Liver resection</td>
<td></td>
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<tr>
<td>• Pituitary adenomectomy, severe facial trauma</td>
<td></td>
</tr>
<tr>
<td>• Dental procedure under general anesthesia, severe facial trauma</td>
<td></td>
</tr>
<tr>
<td>Critical illness/intensive care</td>
<td></td>
</tr>
<tr>
<td>• Major trauma</td>
<td></td>
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<tr>
<td>• Life-threatening complication</td>
<td></td>
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<tr>
<td>IV hydrocortisone, 200 mg/d maximum</td>
<td></td>
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<tr>
<td>(eg, 50 mg every 6 h, or by continuous infusion)</td>
<td></td>
</tr>
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</table>

*a Give parenteral if testing. Modified from reference 3.*
anesthesia. The surgeon discussed the possibility of needing to open and to not be able to perform the procedure via a laparoscopic technique. The patient stated that he had taken all of his medications before the start of his nausea and vomiting. He was administered 50 mg of hydrocortisone by IV as a stress dose. Standard monitors were placed before induction. A rapid sequence induction was facilitated with propofol, succinylcholine, lidocaine, and fentanyl. The patient was maintained under anesthesia with cis-atracurium and desflurane. The procedure was successfully completed by a laparoscopic technique. Normothermia was maintained. A total of 2,300 mL of lactated Ringer’s solution was delivered to account for prior fluid deficit and intraoperative fluid maintenance. Intraoperative blood loss was less than 50 mL. The patient was brought to the recovery room in stable condition, and additional hydrocortisone at a dose of 25 mg IV every 8 hours was administered during the next 2 days. Hydromorphone by patient-controlled analgesia was used for postoperative pain management and to minimize pain-mediated stress. The patient was discharged home without issues on postoperative day 2.

**Conclusion**

Addison’s disease is a complex disorder with many features and various degrees of severity. Patients who are not well maintained on a hormone regimen and require surgery can be significantly dehydrated, and have electrolyte abnormalities. For patients who are newly diagnosed or for those who are on stable regimens, surgery imposes additional stress that requires supplemental steroid dosing. The anesthesiologist must be aware of appropriate preoperative evaluation and correct dosing and administration of IV hydrocortisone perioperatively. Whenever appropriate, consultation with an endocrinologist may be beneficial to minimize morbidity and mortality.
REFERENCES

Visit [www.mssm.procampus.net](http://www.mssm.procampus.net) today for instant online processing of your CME post-test and evaluation form. There is a registration fee of $15 for this non–industry-supported activity. For assistance with technical problems, including questions about navigating the Web site, call toll-free customer service at (888) 345-6788 or send an e-mail to Customer.Support@ProCEO.com. For inquiries about course content only, send an e-mail to ram.roth@mssm.edu. Ram Roth, MD, is director of PreAnesthetic Assessment Online and assistant professor of anesthesiology at The Icahn School of Medicine at Mount Sinai, New York, NY.

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**Post-test**

1. **Which region of the adrenal gland produces glucocorticoids?**
   
   a. Medulla  
   b. Zona reticularis  
   c. Zona glomerulosa  
   d. Zona fasciculata

2. **Corticotrophin-releasing hormone from the _________ stimulates release of _________ from the _________ pituitary gland, which in turn stimulates release of _________ from the _________.
   
   a. hypothalamus; adrenocorticotropic hormone; posterior; cortisol; adrenal cortex  
   b. hypothalamus; adrenocorticotropic hormone; anterior; cortisol; adrenal medulla  
   c. hypothalamus; cortisol; posterior; cortisol; adrenal cortex  
   d. hypothalamus; adrenocorticotropic hormone; anterior; cortisol; adrenal cortex

3. **Presenting signs and symptoms of adrenal insufficiency (AI) occur when _________.**
   
   a. >80%-90% of the adrenal gland is destroyed  
   b. ≥70% of the adrenal gland is destroyed  
   c. between 45% and 60% of the adrenal gland is destroyed  
   d. ≥25% of the adrenal gland is destroyed

4. **What is the most common cause of primary AI today?**
   
   a. Tuberculosis  
   b. Autoimmune adrenalitis  
   c. Histoplasmosis  
   d. HIV  
   e. Bilateral adrenal hemorrhage

5. **Primary AI differs from secondary and tertiary AI in that _________.**
   
   a. primary AI causes only mineralocorticoid deficiency  
   b. primary AI causes only glucocorticoid deficiency  
   c. primary AI causes both mineralocorticoid and glucocorticoid deficiency  
   d. primary, secondary, and tertiary AI all cause the same effects on the adrenal gland
6. A patient who has received ≥20 mg of prednisone per day for 8 days is at __________.
   a. decreased risk for suppression of the hypothalamic–pituitary–adrenal (HPA) axis
   b. increased risk for activation of the HPA axis
   c. increased risk for suppression of the HPA axis
   d. no risk for change in suppression of the HPA axis

7. A patient who has received 3 mg of prednisone per day for the past 5 weeks and does not exhibit signs and symptoms of AI is scheduled to undergo a moderately stressful surgical procedure (ie, open cholecystectomy). Of the following options, which is the best glucocorticoid supplementation for this patient?
   a. Give 25 mg of hydrocortisone IV on day of procedure only.
   b. Give the patient’s usual daily dosage of 3 mg of prednisone only.
   c. Give 50 to 75 mg of hydrocortisone IV on day of procedure or intraoperatively, and taper rapidly over next 1 to 2 days to the patient’s usual dose.
   d. Give 100 to 150 mg of hydrocortisone IV on day of procedure or intraoperatively, and taper rapidly over next 1 to 2 days to patient’s usual dose.

8. A patient who has a history of persistent asthma and uses high-dose corticosteroids regularly is scheduled for a Whipple procedure. Of the following options, which is the best glucocorticoid supplementation for this patient?
   a. Give 25 mg of hydrocortisone IV on day of procedure only.
   b. Give the patient’s usual daily dosage of 3 mg of prednisone only.
   c. Give 50 to 75 mg of hydrocortisone IV on day of procedure or intraoperatively, and taper rapidly over next 1 to 2 days to the patient’s usual dose.
   d. Give 100 to 150 mg of hydrocortisone IV on day of procedure or intraoperatively, and taper rapidly over next 1 to 2 days to patient’s usual dose.

9. A patient with a history of moderate to severe rheumatoid arthritis is scheduled for a total knee arthroplasty. Which glucocorticoid supplementation therapy should be considered for this patient?
   a. Give 25 mg of hydrocortisone IV on day of procedure only.
   b. Give the patient’s usual daily dosage of 3 mg of prednisone only.
   c. Give 50 to 75 mg of hydrocortisone IV on day of procedure or intraoperatively and taper rapidly over next 1 to 2 days to the patient’s usual dose.
   d. Give 100 to 150 mg of hydrocortisone IV on day of procedure or intraoperatively, and taper rapidly over next 1 to 2 days to the patient’s usual dose.

10. Which of the following drugs should be avoided perioperatively to prevent triggering acute AI in a surgical patient with known primary AI or at risk for adrenal suppression?
    a. Thiopental
    b. Propofol
    c. Etomidate
    d. Ketamine