Propofol, introduced in the United States in 1989, is a potent short-acting intravenous sedative-hypnotic agent used to induce and maintain anesthesia and to provide continuous sedation in the intensive care unit (ICU).\(^1\) Propofol has a rapid onset of action (approximately 30 seconds), a rapid rate of distribution (half-life, 2-4 minutes), a dose-related hypnotic effect, and a short elimination half-life (30-60 minutes; Table 1).\(^2\) Use of propofol in the ICU allows frequent and ongoing neurological evaluations and assessments. Furthermore, it does not accumulate in patients with renal or hepatic disease.\(^1\) These pharmacological properties make propofol an ideal agent for treatment of critically ill patients. Currently, propofol

**Propofol Infusion Syndrome**

A Rare Complication With Potentially Fatal Results

Melissa M. Zaccheo, RN, BA, MSN, ACNP-BC, FNP-BC
Donald H. Bucher, RN, MSN, ACNP-BC, CCRN, CSC, CMC

**PRIME POINTS**

- Propofol infusion syndrome (PRIS) is an adverse drug event associated with high doses and long-term use of propofol.
- Critically ill patients receiving catecholamines, glucocorticoids, or high-dose propofol are at risk for PRIS.
- Severe metabolic acidosis, rhabdomyolysis, hyperkalemia, lipemia, renal failure, hepatomegaly, and cardiovascular collapse are key features.
- To improve outcomes, recognize PRIS early and discontinue the propofol infusion.

**CE Continuing Education**

This article has been designated for CE credit. A closed-book, multiple-choice examination follows this article, which tests your knowledge of the following objectives:

1. Identify the 7 key features of propofol infusion syndrome (PRIS)
2. Describe why critically ill patients receiving catecholamines, glucocorticoids, or high-dose propofol are at risk for PRIS
3. Discuss the cardiovascular depressant effects of propofol on patients and identify the increased risk group

**Table 1** Pharmacological properties of propofol\(^3\)

<table>
<thead>
<tr>
<th>Pharmacodynamic properties</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Rapid onset of action (approximately 30 seconds)</td>
</tr>
<tr>
<td>• Decreased mean arterial pressure and heart rate with induction and maintenance of anesthesia</td>
</tr>
<tr>
<td>• Ventilatory depression</td>
</tr>
<tr>
<td>• Decreased cerebral blood flow</td>
</tr>
<tr>
<td>• Decreased intracranial pressure</td>
</tr>
<tr>
<td>• Decreased cerebral metabolism</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Pharmacokinetic properties</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Rapid rate of distribution (half-life 2-4 minutes)</td>
</tr>
<tr>
<td>• Rapid elimination (half-life 30-60 minutes)</td>
</tr>
<tr>
<td>• Extensive distribution</td>
</tr>
<tr>
<td>• Rapid total body clearance (1.5-2 L/min)</td>
</tr>
<tr>
<td>• Metabolism mainly in the liver with formation and urinary excretion of inactive conjugates and quinols</td>
</tr>
<tr>
<td>• Linear pharmacokinetics</td>
</tr>
</tbody>
</table>

\(^*\) Data derived from AstraZeneca.\(^2\)
is indicated for continuous sedation in adult patients receiving mechanical ventilation in the critical care setting.

Despite the inherent pharmacological and clinical advantages of propofol therapy in the ICU, its use is associated with adverse drug-related events (Table 2). Propofol has a cardiovascular depressant effect that may lead to hypotension and bradycardia, particularly in volume-depleted patients. Hypertriglyceridemia associated with pancreatitis, respiratory depression, pain with injection, green discoloration of urine, nausea, vomiting, and allergic complications have also been historically associated with propofol administration.

Propofol has been administered to millions of patients worldwide, in various acute care clinical settings, and has an acceptable safety record for sedation of adult ICU patients. Propofol was compared with benzodiazepines and/or opioids in 14 clinical trials involving a total of 550 ICU patients. Of these, 302 patients received propofol and comprise the overall safety database for ICU sedation. Six of these studies were done in the United States and Canada and provide the basis for dosage recommendations and the safety profile of the drug.

In 1992, Parke et al. published a landmark article that suggested a link between propofol infusions and mortality in children. In 2006, Corbett et al. documented a total of 15 adult cases of propofol infusion syndrome published in the English-language literature. Propofol infusion syndrome (PRIS) is an adverse drug event associated with high doses (>4 mg/kg per hour or >67 μg/kg per minute) and long-term (>48 hours) use of propofol. PRIS is characterized by severe metabolic acidosis, rhabdomyolysis, hyperkalemia, lipemia, renal failure, hepatomegaly, and cardiovascular collapse. The true incidence of PRIS is unknown; however, Crozier, on the basis of pooled clinical data, calculated with a 95% probability that the incidence of PRIS is 1 in 270 patients (0.37%) or lower.

We present 2 case reviews of PRIS and review the pathophysiology, clinical manifestations, and management associated with this potentially fatal clinical condition.

### CASE 1

A 33-year-old woman came to the emergency department via ambulance after a motor vehicle crash. She had been unresponsive at the scene, with a score of 6 on the Glasgow Coma Scale. She did not open her eyes spontaneously, to voice, or to pain. Paramedics noted that she was making incomprehensible sounds with flexion posturing to deep stimulation. She was subsequently intubated to obtain definitive protection of the airway. Computed tomography of the head showed a right temporal subdural hematoma and a subarachnoid hemorrhage. A ventriculostomy drain was placed, and intracranial pressure (ICP) monitoring was started. Her initial ICP was 40 mm Hg.

An infusion of propofol was started at 20 μg/kg per minute to achieve adequate sedation, in conjunction with a continuous morphine infusion at 1 mg/h for analgesia. In addition, the attending physician ordered propofol to be titrated to manage intracranial hypertension. The patient’s ICP

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### Table 2 Adverse drug effects of propofol

<table>
<thead>
<tr>
<th>Effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Local pain on induction</td>
</tr>
<tr>
<td>Hypotension</td>
</tr>
<tr>
<td>Bradycardia</td>
</tr>
<tr>
<td>Transient apnea during induction</td>
</tr>
<tr>
<td>Nausea and vomiting</td>
</tr>
<tr>
<td>Headache</td>
</tr>
<tr>
<td>Thrombosis and phlebitis</td>
</tr>
<tr>
<td>Epileptiform movements</td>
</tr>
<tr>
<td>Rhabdomyolysis</td>
</tr>
<tr>
<td>Pancreatitis</td>
</tr>
<tr>
<td>Postoperative fever</td>
</tr>
<tr>
<td>Discoloration of urine</td>
</tr>
<tr>
<td>Anaphylaxis</td>
</tr>
<tr>
<td>Sexual disinhibition</td>
</tr>
<tr>
<td>Pulmonary edema</td>
</tr>
</tbody>
</table>

*Data derived from AstraZeneca.*

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**Authors**

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ranged from 20 to 35 mm Hg, necessitating an increase in the propofol infusion an average of 20 μg/kg per minute every hour to 100 μg/kg per minute. The goal for the patient, with regard to the propofol titration, was to decrease her ICP to maintain a cerebral perfusion pressure greater than 70 mm Hg. During the patient’s second day in the ICU, the average dose of propofol was 120 μg/kg per minute. The goal cerebral perfusion pressure was not achieved; therefore, nor-epinephrine was started at 0.05 μg/kg per minute. The patient’s cerebral hypertension did not change appropriately with the additional therapy, and continuous nondepolarizing neuromuscular blockade was subsequently started. A pancuronium infusion was begun at 0.1 mg/kg per hour, and a train-of-4 muscular contraction examination indicated 1 muscle contraction per 4 stimuli.

Routine arterial blood gas (ABG) analysis on the morning of the second day showed no metabolic derangements: pH 7.45, PCO₂ 32 mm Hg, PO₂ 97 mm Hg, base excess 5 mEq/L, bicarbonate 27 mEq/L.

On days 3 and 4, the patient’s mental status progressively declined and cerebral hypertension continued despite a propofol infusion rate of 180 μg/kg per minute and continued use of paralytic agents and vaspressors. The patient’s ICP was 30 to 39 mm Hg, and her cerebral perfusion pressure was 60 to 69 mm Hg. Serum triglyceride level was 7910 mg/dL (to convert to millimoles per liter, multiply by 0.0113) on day 4. Attempts were made to decrease the propofol infusion; however, these attempts were unsuccessful because of ongoing refractory intracranial hypertension.

Figure 1 Progression of propofol infusion syndrome on electrocardiograms (ECGs). A, ECG obtained in the morning of day 6 shows normal sinus rhythm with a first-degree atrioventricular block, left anterior hemiblock, and right bundle branch block, all of which were of new onset since admission. B, ECG obtained after the arrest phase shows ventricular tachycardia. C, ECG obtained after the first cardiac arrest shows second-degree atrioventricular block.
On day 6, the ICP increased to 40 to 45 mm Hg. Concurrent physical examination revealed anisocoria. This clinical change in the patient’s condition required further increase in the propofol infusion to 190 μg/kg per minute. Subsequent ABG analysis revealed pH 7.39, Pco2 26.0 mm Hg, Po2 85 mm Hg, base excess -7.2 mEq/L, and bicarbonate 15.3 mEq/L. A 12-lead electrocardiogram revealed normal sinus rhythm with a first-degree atrioventricular block, left anterior hemiblock, and right bundle branch block (Figure 1A).

Several hours later, a wide-complex ventricular rhythm developed (Figure 1B) along with profound hypotension with a systolic blood pressure of 70 mm Hg. The hypotension did not improve after administration of norepinephrine and fluid replacement. ABG analysis revealed a worsening metabolic acidosis: pH 7.17, Pco2 46 mm Hg, Po2 131 mm Hg, base excess -14 mEq/L, and bicarbonate 10 mEq/L. A bolus of sodium bicarbonate followed by a continuous intravenous infusion was administered to treat the acidosis. Tachycardic and bradycardic rhythms (Figure 1C) continued, and a temporary transvenous pacemaker was emergently placed. One hour after the bicarbonate infusion was started, ABG analysis revealed a refractory metabolic acidosis: pH 7.18, Pco2 24 mm Hg, Po2 224 mm Hg, base excess -17.6 mEq/L, and bicarbonate 8.9 mEq/L. Additional laboratory evaluation indicated the following serum levels: lactic acid 12.5 mg/dL (to convert to millimoles per liter, multiply by 0.111), total creatine kinase 18333 U/L (to convert to microkatal, multiply by 0.0167), myoglobin 18470 ng/mL (to convert to nanomoles per liter, multiply by 0.0571), and triglycerides 11420 mg/dL.

After traumatic motor vehicle accidents, elevations in the serum level of creatine kinase are to be expected, depending on the severity of the trauma. However, the patient’s entire constellation of signs and symptoms could not be explained by other possible causes in the differential diagnosis, such as trauma, acute coronary syndrome, sepsis, or seizures. The propofol was discontinued, additional boluses of sodium bicarbonate were given, and a continuous infusion of epinephrine was begun. The patient’s clinical condition rapidly declined, despite maximal supportive therapies, and she progressed to a cardiac arrest with pulseless electrical activity. Advanced Cardiac Life Support was ineffective, and the patient died.

Case 2

A 64-year-old man with a history of chronic alcoholism, hypertension, diabetes mellitus, and chronic obstructive pulmonary disease came to the emergency department with lethargy, confusion, aphasia, and hemiparesis. In his home hours earlier, he had had 2 witnessed tonic clonic seizures. In the emergency department, he had a witnessed 2-minute tonic clonic seizure that was relieved with lorazepam. Computed tomography and magnetic resonance imaging of the head showed no acute organic intracranial abnormality. Results of blood cultures, serological studies, and lumbar puncture also were unremarkable. Additionally, an emergent electroencephalogram displayed no ongoing epileptiform discharges.

The patient was admitted to a telemetry monitored, neurology nursing unit with a presumed diagnosis of alcohol withdrawal seizures. At the discretion of the attending neurologist, the patient was treated supportively with phenytoin and zonisamide.

During the following 48 hours, the patient had worsening encephalopathy, with a decrease in level of consciousness without clinical seizure activity. Seventy-two hours after admission, the patient became unable to protect his airway because of his decreasing mental status, a situation that resulted in hypoxia. He was emergently intubated. An electroencephalogram indicated that he was in status epilepticus. He was treated with intravenous
fosphenytoin, but the seizure activity continued. He was then given a bolus dose of propofol in the amount of 1 mg/kg, and an infusion of 120 to 140 µg/kg per minute was started to achieve burst suppression of the seizure activity. Intravenous fluids and norepinephrine were started because of hypotension in the setting of a continuous infusion of high-dose propofol. With crystalloid volume replacement, his exogenous catecholamine requirements decreased during the next several hours.

Approximately 48 hours after the continuous infusion of propofol was started, acute, profound hypotension developed, associated with a lactic acidosis and acute oliguric renal failure. The diagnosis of PRIS was considered, and appropriate evaluation ensued. Serum triglyceride level was 628 mg/dL; creatine kinase level was 11,268 U/L; ABG analysis indicated pH 7.34, Pco₂ 27.1 mm Hg, Po₂ 181.4 mm Hg, and bicarbonate level 14.1 mEq/L; arterial lactate level was 8.4 mg/dL; and myoglobin level was 117,769 ng/mL. Propofol was immediately discontinued, and an arterial catheter was placed emergently. Rapid resuscitation was begun with the reintroduction of vasopressor support (norepinephrine 0.4 µg/kg per minute) and crystalloid/colloid administration because of profound shock. Because of ongoing oliguric acute renal failure complicated by hyperkalemia, continuous renal replacement therapy was started.

Subsequent serological evaluation (Table 3) revealed a triglyceride level of 310 mg/dL; ABG analysis indicated pH 7.35, Pco₂ 32.6 mm Hg, Po₂ 91.5 mm Hg, and bicarbonate level 17.7 mEq/L; arterial lactate level was 7.7 mg/dL; and creatine kinase level was 69,175 U/L. Neurological examination revealed brainstem reflexes only, consistent with anoxic brain injury. The patient was maintained on maximal supportive therapies at that time.

Ninety-six hours after admission, the patient had a strikingly elevated myoglobin level of 210,030 ng/mL. His clinical status remained unchanged, with ongoing shock, metabolic acidosis, oliguric renal failure, lactic acidosis, rhabdomyolysis, and dyslipidemia. His family decided on withdrawal of care and provision of comfort measures only. The patient died approximately 10 minutes after discontinuation of all supportive measures.

### Table 3 Comparison of laboratory data for case 1 and case 2

<table>
<thead>
<tr>
<th>Case</th>
<th>Day</th>
<th>Triglyceride, mg/dL</th>
<th>Creatine kinase, U/L</th>
<th>pH</th>
<th>Pco₂, mm Hg</th>
<th>Po₂, mm Hg</th>
<th>Bicarbonate, mEq/L</th>
<th>Arterial lactate, mg/dL</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>4</td>
<td>7910</td>
<td></td>
<td>7.39</td>
<td>26</td>
<td>85</td>
<td>15.2</td>
<td></td>
</tr>
<tr>
<td></td>
<td>5, 4 AM</td>
<td>11,420</td>
<td>18,333</td>
<td>7.17</td>
<td>46</td>
<td>131</td>
<td>10</td>
<td>12.5</td>
</tr>
<tr>
<td>2</td>
<td>2</td>
<td>628</td>
<td>11,268</td>
<td>7.33</td>
<td>27.1</td>
<td>181.4</td>
<td>14.1</td>
<td>8.4</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>310</td>
<td>69,175</td>
<td>7.35</td>
<td>32.6</td>
<td>91.5</td>
<td>17.7</td>
<td>7.7</td>
</tr>
</tbody>
</table>

SI conversion factors: To convert triglycerides to mmol/L, multiply by 0.0113; to convert creatine kinase to µkat/L, multiply by 0.0167; to convert lactate to mmol/L, multiply by 0.111.

### Pathophysiology

PRIS is thought to be related to impaired utilization of fatty acid in the mitochondria. Researchers have focused on impaired respiratory chain functions in the mitochondria. The etiology of the mitochondrial defect is undetermined. Hypotheses have included metabolite-mediated defects and an underlying neurovascular defect. Propofol increases the activity of malonyl coenzyme A, thus inhibiting carnitine palmitoyl transferase I, so that long-chain free fatty acids cannot enter the mitochondria. β-Spiral oxidation and the respiratory chain are uncoupled at complex II; therefore, neither medium- nor short-chain free fatty acids can be used, a situation that leads to myocytolysis. The imbalance...
between energy supply and demand may lead to cardiac and peripheral muscle necrosis. Additionally, the accumulation of free fatty acids may contribute to arrhythmias.

The clinical conditions that comprise PRIS are cardiac failure, rhabdomyolysis, severe metabolic acidosis, and renal failure. Multiple factors must be present for PRIS to develop. Vasile et al identified critical illness as the “priming factor.” Critical illness is a prerequisite for the development of this syndrome. Concomitant “triggering factors” must be present to allow the pathological cascade of events to occur and ultimately result in the clinical syndrome known as PRIS.

Triggering factors in critical care patients include high-dose propofol, catecholamines, and glucocorticoids (Figure 2).

Critical illness leads to tissue damage and the release of proinflammatory cytokines, anti-inflammatory glucocorticoids, and endogenous catecholamines. If the endogenous glucocorticoid response is inadequate, as often occurs in prolonged and/or severe illness, susceptibility to inflammation is enhanced by the continued release of catecholamines and cytokines. In this persistent inflammatory state, increased catabolism leads to progressive cardiac and skeletal muscle dysfunction and breakdown. According to Kam and Cardone, the imbalance between cellular energy production and utilization in muscle, both cardiac and peripheral, is the key pathogenic mechanism that causes muscle necrosis.

Propofol impairs utilization of free fatty acids (the fuel for cardiac and skeletal muscle) and mitochondrial activity. The body’s metabolic demands increase with critical illness, but with the inability to generate fuel, catabolism occurs, leading to cardiac and skeletal muscle necrosis with accumulation of free fatty acids. Clinically, this accumulation is evidenced by elevated levels of serum creatine kinase, troponin I, and myoglobin.

Figure 2 Pathophysiology of propofol infusion syndrome. Priming factors are an essential prerequisite for the syndrome to develop. Critical illness with inadequate stress hormone responses creates a proinflammatory state with hypercatabolism, causing progressive organ dysfunction (priming). High-dose propofol, catecholamines, and glucocorticoids are the triggering factors associated with propofol infusion syndrome.8
Propofol has a negative inotropic effect due to the decreased sympathetic tone and antagonism of β-adrenoreceptors and calcium channels. This negative inotropic effect results in increased catecholamine requirements to maintain hemodynamic stability. As the catecholamine surge increases, propofol infusions must be increased to provide the same sedative effect. Thus, a vicious cycle is created. Propofol and catecholamines drive each other, resulting in increased catecholamine production that leads to a hypercatabolic state and breakdown of skeletal and cardiac muscle. As skeletal and cardiac muscles break down, rhabdomyolysis, metabolic acidosis, acute renal failure, and, ultimately, cardiac failure shortly ensue.

Clinical Manifestations

Table 4 lists the clinical manifestation of PRIS. Unexplained metabolic acidosis (no indications of shock) may be one of the earliest warning signs. Three case reports have suggested that early onset of lactic acidosis after the start of propofol infusions, in the absence of other causes, may be an early marker of PRIS. ST-segment elevation in the right precordial leads (V1-V3) may be the first indication of cardiac instability commonly associated with PRIS. In the absence of known causes of rhabdomyolysis (seizures, anti-dopaminergic medication, depolarizing paralytic agents, hyperthermia, or extensive trauma), PRIS should be expected. An enlarged liver due to fatty infiltration is common but is often an underappreciated finding on physical examination. In 1 case review, 82% of patients with PRIS had a clinically enlarged liver.

Management

Prevention of PRIS is paramount. Although propofol has been approved by the Food and Drug Administration for use in providing sedation in the ICU, clinicians have a responsibility to use this drug safely and within the recommended guidelines. High doses of propofol greater than 67 μg/kg per minute or 4 mg/kg per hour for longer than 48 hours should be avoided. Additionally, ICU nurses should recognize those patients with the priming factor (critical illness) and concomitant triggering factors (catecholamines, glucocorticoids, and high-dose propofol infusion) as patients at risk for PRIS.

Once PRIS is suspected and/or diagnosed, management consists solely of supportive treatments addressing the clinical manifestations and the end organ damage associated with this syndrome. The propofol infusion should be discontinued immediately, and an alternative sedative should be started. Intravenous crystalloid and colloid replacement and vasopressor and/or inotropic support are usually required to treat the hypotension and refractory shock. Cardiac pacing may be used for symptomatic bradycardia, and, ultimately, hemodialysis or continuous renal replacement therapy will be required to treat the acute renal failure and metabolic acidosis associated with PRIS.

Early recognition of the syndrome in the appropriate clinical setting and discontinuation of the propofol infusion are of paramount importance for decreasing untoward morbidity and mortality. The Society of Critical Care Medicine recommends that patients receiving propofol have serum triglyceride levels measured after 2 days.

Table 4 Clinical manifestations of propofol infusion syndrome

- Unexplained metabolic acidosis
- Hypertriglyceridemia
- Hypotension
- Lactic acidosis
- Arrhythmia: sudden onset of marked bradycardia resistant to treatment
- Rhabdomyolysis
- Acute renal failure
- Hepatomegaly

Table 5 Recommendations for the clinical care of patients who are receiving propofol infusions or have early markers of propofol infusion syndrome

- Continuous electrocardiographic monitoring
- Serum triglyceride levels: initial measurement after 2 days of continuous infusion (per guidelines of the Society of Critical Care Medicine)
- Awareness of priming factor (critical illness) and triggering factors (glucocorticoids, catecholamines, high-dose propofol)
- Creatine kinase levels
- Early adequate carbohydrate intake
- Markers of propofol infusion syndrome to be aware of: unexplained metabolic acidosis, elevated serum lactate, elevated creatine kinase and myoglobin levels, hyperlipidemia
Because hypertriglyceridemia can be an early marker in the development of PRIS, triglyceride levels should be monitored in all patients receiving propofol.1 The Society of Critical Care Medicine also recommends that alternative sedative agents be considered for patients with escalating vasopressor or inotropic requirements3 (Table 5).

Critical care nurses must be trained to recognize patients at risk for PRIS. Prevention and subsequent recognition of this complex syndrome are essential to improve patients’ outcomes. Nationally, hospitals are developing policies for the use of propofol in the ICU, including recommended dosages, indications for use approved by the Food and Drug Administration, recommended durations of use, and serial laboratory studies. Hospitals are also supporting the role of nurses in early identification of PRIS. CCRN

Financial Disclosures
None reported.

References

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Propofol Infusion Syndrome: A Rare Complication With Potentially Fatal Results

**Facts**

- Propofol infusion syndrome (PRIS) is an adverse drug event associated with high doses and long-term use of propofol.
- Critically ill patients receiving catecholamines, glucocorticoids, or high-dose propofol are at risk for PRIS.
- PRIS is characterized by severe metabolic acidosis, rhabdomyolysis, hyperkalemia, lipemia, renal failure, hepatomegaly, and cardiovascular collapse.
- Keys to improving outcomes are recognizing PRIS early and discontinuing the propofol infusion.


This article and an online version of the CE test may be found online at http://ccn.aacnjournals.org.
The rapid onset of action for propofol is approximately:
- a. 30 seconds
- b. 15 seconds
- c. 20 seconds
- d. 45 seconds

2. The cardiovascular depressant effects of propofol include:
- a. Bradycardia and respiratory depression
- b. Bradycardia and hypotension
- c. Respiratory depression and hypotension
- d. Hypotension and low central venous pressure

3. Which of the following doses of propofol infusion are associated with propofol infusion syndrome (PRIS)?
- a. >4mg/kg per hour and >48 hours use
- b. >3mg/kg per hour and >48 hours use
- c. >3mg/kg per hour and >72 hours use
- d. >3mg/kg per hour and >72 hours use

4. Which of the following are triggering factors for PRIS in critical care patients?
- a. Elevated blood sugars, high-dose propofol, and glucocorticoids
- b. High-dose propofol, glucocorticoids, and vasopressor therapy
- c. Glucocorticoids, high-dose propofol, and negative inotropes
- d. High-dose propofol, catecholamines, and glucocorticoids

5. Which of the following imbalances is the key pathogenic mechanism that causes muscle necrosis?
- a. Between energy supply and utilization in skeletal and cardiac muscle
- b. Between energy supply and utilization in cardiac and renal muscle
- c. Between energy supply and utilization in skeletal and renal muscle
- d. Between energy supply and utilization in cardiac and peripheral muscle

6. Propofol impairs utilization of free fatty acids, which are the fuel used for which of the following?
- a. Cellular metabolism
- b. Hemodynamic stability
- c. Cardiac and skeletal muscle
- d. Cardiac and peripheral muscle

7. Propofol has a negative inotropic effect that results in which of the following to maintain hemodynamic stability?
- a. Increased catecholamine requirements
- b. Volume replacement
- c. Pacemaker support
- d. Continuous monitoring of cardiac output

8. Propofol and catecholamines drive each other resulting in increased catecholamine production that leads to a hypercatabolic state and a breakdown of which of the following?
- a. Renal cells resulting in renal failure
- b. Skeletal muscle and brain cells
- c. Skeletal and cardiac muscle
- d. Cardiac muscle and central metabolism

9. What may be the first indication of cardiac instability commonly associated with PRIS?
- a. ST-segment elevation in right V1-V3
- b. ST-segment depression in right V1-V3
- c. ST-segment elevation in right V4-V6
- d. ST-segment depression in right V4-V6

10. What is a common underappreciated finding on physical examination related to fatty infiltrate in PRIS?
- a. Green urine
- b. Bradycardia
- c. Enlarged pancreas
- d. Enlarged liver

11. According to the Society of Critical Care Medicine, when should the initial serum triglyceride levels be checked?
- a. Daily
- b. After 2 days
- c. After 3 days
- d. After 5 days

12. PRIS is not characterized by which of the following?
- a. Respiratory failure
- b. Hyperkalemia
- c. Renal failure
- d. Rhabdomyolysis
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