Marijuana consumption is growing in the United States because of state legalization for recreational and medical use. However, many anesthesia practitioners are unaware of the potential adverse effects that may occur if marijuana is taken before the administration of an anesthetic. This review provides a history of marijuana use, the current laws and regulations, the pharmacology of marijuana, and best evidence-based practices related to anesthetic care of the marijuana user.

Keywords: Anesthesia, cannabis, drug interaction, marijuana.

Marijuana Use in the Anesthetized Patient: History, Pharmacology, and Anesthetic Considerations

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The cannabis plant, also known as marijuana, becomes a mind-altering, psychoactive drug when consumed or inhaled; it is one of the oldest documented plants with an extensive history of medicinal and recreational use.\(^1\) Despite legalization of the drug for medical and recreational use by several states; the US Department of Justice’s Drug Enforcement Administration continues to list marijuana as a Schedule I substance under the Controlled Substances Act, citing a high potential for abuse and the lack of accepted safety for use.\(^2\) In 2013, a year after the first states legalized recreational marijuana use, 19.8 million Americans were classified as current users.\(^3\) Recent broader legalization of medical marijuana recreational use has dramatically increased use of this drug from 7% of the population in 2013 to 13% in 2016.\(^4\) The most recent federal survey, released in 2015, estimates 33 million current cannabis users in the United States.\(^5\) The number of marijuana users rivals that of nicotine cigarette smokers, which is currently estimated to be 38 million.\(^5\)

As marijuana use increases in the United States, anesthesia practitioners can expect to provide care to an increased number of patients using this drug. A thorough understanding of marijuana’s pharmacodynamics and pharmacokinetics and related anesthetic implications are important for the anesthesia practitioners caring for patients who are acutely intoxicated and/or using marijuana long term. A review on cannabis and its effects on anesthesia were discussed in 1980 by Dickerson,\(^6\) but because of the substantial increase in the rate of marijuana use and advancements in our understanding of the drug’s pharmacology, an updated review on the history, pharmacology, and anesthetic implications is warranted.

History of Marijuana Use

The cannabis plant genus can be traced back millions of years and was indigenous to the Asian continent.\(^7\) The 2 main cannabis plant species are Cannabis sativa and C indica; both can be used to produce hemp fiber, but the latter is more commonly associated with the psychoactive derivatives of the plant.\(^7\) Western medicine was introduced to cannabis during the early 1800s through the writings of W. B. O’Shaughnessy, a physician working with the British in India, according to a historical perspective by Mikuriya.\(^8\) His successful experimentation with hemp preparations in animals led him to begin treating humans with cannabis extracts.\(^8\) O’Shaughnessy found it useful for the treatment of pain, muscle spasms, and, reportedly, seizures.\(^8\) In 1860 the Ohio State Medical Society published a report on C indica, which chronicled its important uses and lack of the deleterious side effects seen with opium. The authors noted that potency varied considerably among cannabis derivatives, but cannabis was not as potent as opium and, furthermore, it was devoid of the deleterious and unpleasant side effects associated with opiates.\(^8\) The latter half of the 19th century saw a decline in the use of cannabis preparations because of medical advances such as the hypodermic syringe and water-soluble opiates. Nonetheless, over-the-counter cannabis preparations were available and remained a part of the nation’s pharmacopeia until 1941.\(^7,9\)

During the early 20th century, the United States was introduced to the recreational smoking of cannabis, or “marijuana,” as it then became known, and its use quickly spread throughout the country.\(^9\) In 1924 the second International Opiates Conference declared cannabis to be a narcotic, and the states began to regulate and criminalize marijuana possession and its use. Despite
various reports extolling the value of marijuana use, by 1956 the US Narcotics Control Act ordered a first-time offense of cannabis possession punishable with 2 to 10 years of prison time and fines.9

The 1960s and 1970s popularized the recreational use of marijuana, and during this time various states’ legislative efforts attempted to decriminalize the use and possession of marijuana. Reports began to appear during the 1970s and 1980s that marijuana was helpful in relieving nausea associated with chemotherapy and the wasting syndrome that occurred in patients with AIDS.9, 10

To date, 33 states and the District of Columbia have legalized marijuana for medical use. Nine of these states and the District of Columbia also allow for its recreational use.11 There appears to be strong public support for medical access, but the debate waxes on regarding full legalization. Federally, it remains an illegal substance, causing difficulty with funding for research and murky legal questions concerning the states’ implementation of legalization.10

Pharmacodynamics of Marijuana
• Receptor Pharmacology. Marijuana is extracted from the dried leaves of the plant C sativa and contains approximately 450 distinct compounds, including 60 cannabinoids.12 Cannabinoids are pharmacologically active compounds that act as agonists at the G-protein coupled receptors of the endocannabinoid system.13,14 Although the relative concentration of the 60 cannabinoids in marijuana varies depending on growing conditions and variety, Δ9-tetrahydrocannabinol (THC) is typically present in the highest concentration.15 For example, in more than 31,000 samples of marijuana confiscated by law enforcement between 1993 and 2008, the average mean concentration of THC was 4.5% compared with the next highest concentrated cannabinoid, 0.4% cannabidiol (CBD).16 Furthermore, the psychoactive effects of marijuana are primarily attributed to THC, and therefore, the potency and efficacy of the drug is directly related to the THC concentration. The ratio of THC to CBD may also be important because CBD appears to attenuate THC’s psychoactive effects.13 The National Institute on Drug Abuse reports that the potency of marijuana has increased dramatically over the last 2 decades; the average measured THC concentration increased from 3.8% in the late 1990s to 12.2% in 2014.17 More importantly, a number of illicit synthetic cannabinoids have become available with potencies up to 200-fold higher than that of THC.18

Two cannabinoid receptors have been described: cannabinoid-1 (CB1) and cannabinoid-2 (CB2).18 Although THC is considered a weak partial agonist of CB2 and CB1, some synthetic cannabinoids act as full agonists at these receptors.18 The CB1 receptors are primarily expressed presynaptically in the peripheral nerves, spinal cord, basal ganglia, cerebellum, hippocampus, and association cortices.13 However, low levels of CB1 receptor expression have been described on the heart, the blood vessels (endothelial and smooth muscle cells), peripheral autonomic neurons, and in other peripheral locations.18 Activation of CB1 results in the inhibition of glutamate, γ-aminobutyric acid (GABA), norepinephrine, dopamine, serotonin, and acetylcholine release and the indirect modulation of several neuronal receptors, including opioid and N-methyl-d-aspartate (NMDA) receptors.13,19 The CB2 receptors are expressed in cells of the immune and hematopoietic systems, where they have been shown to have inhibitory effects on inflammation, immune function, and nociception.12,13

• Effects of Marijuana. The physiologic effects of marijuana can be both excitatory and inhibitory in nature and are primarily attributed to the dose-dependent effects of THC.15 Based largely on case studies, epidemiologic data, and retrospective research findings, it appears that the central nervous, cardiovascular, and respiratory systems are most affected by marijuana use. Unfortunately, at this time, definitive evidence from prospective, randomized, double-blinded studies remain insufficient to draw solid conclusions regarding the risk of systemic disease with marijuana use.

• Central Nervous System (CNS) Effects. Subjectively, marijuana users most commonly report euphoria with antianxiety-like effects.13,15 However, periods of dysphoria, including panic, psychosis and anxiety, occur in some individuals.13,15 Other subjective effects include enhanced appetite, enhanced perception of sensory stimuli, and somnolence.13,15,19 Cognitively, learning and memory are impaired and psychomotor slowing is observed.19 Pupillary responses to light are impaired, which may complicate a neurologic examination.19 Although a clear cause and effect relationship has not been established, patients genetically predisposed to psychiatric disease, including depression, anxiety and schizophrenia, have an increased risk of mental illness with long-term use of marijuana.20 Furthermore, although the risk of addiction in patients using cannabis for medical reasons is unknown, among those who experiment with recreational use, 9% become addicted.20

• Cardiovascular System Effects. Several lines of evidence suggest that marijuana use may be associated with an increased risk of cardiovascular and cerebrovascular events.18,21,22 Potential mechanisms contributing to this risk include altered autonomic nervous system function, endothelial and myocardial cell damage, atherogenic effects, oxidative stress, alteration in flow through the coronary microcirculation, promotion of a hypercoagulable state, and increased carboxyhemoglobin levels by the drug.18,22 Extensive preclinical research demonstrates that both CB1 antagonists and CB2 agonists improve short-term hemodynamic alterations and long-term outcomes in several animal models of circulatory
Respiratory Effects. These results suggest that excessive signaling through the CB1 receptor may lead to cardiovascular pathology, whereas signaling through the CB2 receptor may offer a cardiac and vascular protective effect. Thus, it is likely that exogenous modulators of these receptors, such as THC found in marijuana, may also have substantial short-term and long-term cardiovascular function. However, because THC acts as a weak partial agonist of both CB1 and CB2 receptors, the cumulative effects of marijuana use on cardiovascular health (harmful vs protective) remain unclear, and further research in this area is warranted.

Hemodynamic effects of marijuana that have been determined experimentally include both vasodilation and vasoconstriction, reduced cardiac contractility, and both bradycardia and tachycardia. Tachycardia and orthostatic hypotension are most consistently observed in healthy volunteers under the influence of the drug. Tachycardia is presumed to result from inhibition of acetylcholine signaling at the vagal nerve as well as enhanced sympathetic nervous system tone. These increases in heart rate have been reported to last up to 3 hours after marijuana use. Electrophysiologic changes associated with marijuana intoxication include a reduction in p-wave amplitude, ST-segment abnormalities, and sinoatrial node and atrioventricular node conduction time. Furthermore, several case reports have suggested an association between marijuana use and the development of arrhythmias such as atrial fibrillation, atrioventricular block, and ventricular tachycardia. However, no randomized controlled studies have been conducted to confirm these findings.

Individuals appear to be at particularly high risk of adverse cardiovascular and cerebrovascular effects while acutely intoxicated with marijuana; therefore, it may be prudent to avoid anesthesia during this period. For example, Mittleman and colleagues reported that marijuana users have a 5-fold increased risk of myocardial infarction in the first hour after use of the drug. Of note, this risk did not appear to extend beyond the first hour following drug use. Furthermore, several reports of acute coronary syndrome reported after marijuana use occurred in the context of strenuous exercise. Although these results have not been confirmed in controlled studies, it is possible that acute intoxication with marijuana may make a patient particularly sensitive in contexts such as anesthesia in which sympathetic nervous system activity would be increased. Another disturbing trend of particular interest to anesthesia providers is a report of serious cardiovascular complications in relatively young patients reported to be otherwise healthy.

- Respiratory Effects. Although animal research suggests that marijuana smoke may induce hyperreactive airways, inflammation, and advanced obstructive lung disease at rates higher than with tobacco smoke, there is insufficient evidence to make any definitive conclusions regarding the impact of marijuana use and pulmonary function and/or disease in humans. Evidence suggests long-term marijuana use induces tobacco-like pulmonary complications. For example, there appears to be an association between marijuana inhalation and the development of lung cancer, bullous emphysema, and chronic obstructive pulmonary disease. Furthermore, several investigations suggest that patients who smoke marijuana may present with a higher incidence of uveal edema, bronchoconstriction, excessive pulmonary secretions, and altered results of pulmonary function tests, and may require deeper levels of anesthesia during airway instrumentation.

- Other Systemic Effects. Other anticholinergic-type adverse effects associated with marijuana use include blurred vision, dryness of the eyes and mouth, and difficulty voiding. Of particular interest to anesthesia providers, THC administration greatly slows gastric emptying in human volunteers from an average of 30 to 120 minutes.

- Potential Medical Indications. The US Food and Drug Administration (FDA) has approved 3 synthetic cannabinoids for use in the United States. Dronabinol and nabilone have been approved for use in treating chemotherapy-induced nausea and vomiting and the wasting syndrome in patients with cancer and AIDS, and the Epidiolex brand of cannabidiol was recently approved for 2 specific forms of epilepsy: Lennox-Gastaut syndrome and Dravet syndrome. Although marijuana remains illegal federally, many states have approved marijuana and other cannabinoid-containing compounds for the treatment of glaucoma, chronic pain, inflammatory conditions (asthma, Crohn disease), sleep disorders, anxiety disorders, movement disorders (Tourette syndrome, dystonia, Parkinson disease), posttraumatic stress disorder, generalized seizure disorders, and spasticity induced by multiple sclerosis and spinal cord injury. In general, the quality of evidence supporting these proposed (non-FDA approved) indications is highly variable. In some cases, the recommendations are based on results of animal studies, preliminary research, and/or poorly controlled studies in humans. Therefore, additional research is required to firmly establish efficacy.

**Pharmacokinetics**

The onset of effect of marijuana or synthetic formulations of THC depends on the route of administration. The peak concentration is achieved most rapidly with rectal administration (15 minutes), followed by inhalational (smoking/vaporization) (22 minutes), sublingual (30 minutes), oral (1-2 hours), and transdermal (2 hours). THC is approximately 97% protein bound, highly lipophilic (average volume of distribution = 236 L) and undergoes first-pass metabolism by various cytochrome P-450 enzymes, including CYP2C9, CYP3A4, and CYP2B6. Although the liver is the major site for metabolism, a significant portion is also metabolized in the lungs.
of metabolism, the heart and lungs also contribute. Approximate... intoxicated with marijuana, so one should consider delaying all nonemergent cases during this period. As noted in the Table, the hepatic effects of cannabis may inhibit the metabolism of warfarin, thereby increasing its therapeutic effects leading to bleeding. The potential for increased surgical blood loss should be noted and the necessary preparations implemented. Before initiating neuraxial and regional anesthesia blocks, laboratory verification of correction of international normalized ratio and/or prothrombin time might be considered.

**Intraoperative Considerations.** Several concerns with marijuana use exist during the intraoperative period.

- **Airway and Pulmonary Concerns.** If possible, elective procedures should be delayed 72 hours following marijuana use, to reduce the risk of airway hyperresponsiveness with airway instrumentation. Research has shown there is an association between inhalational marijuana and spontaneous pneumothorax and bullous emphysema. Caution should be taken when one is using positive pressure ventilation because increases in pressure in bulla will increase the risk of rupture leading to tension pneumothorax. Increases in peak airway pressure, desaturation, and tachycardia should alert the provider to possible lung collapse. Data from studies conflict on whether inhaled marijuana actually alters pulmonary function, with studies showing both decreases and increases in airway conductance. Inhaled smoke from a cannabis cigarette contains many of the same components as tobacco smoke—elements that lead to bronchial irritation. For the long-term marijuana smoker, this creates the possibility of airway hyperreactivity similar to that of the tobacco smoker. A review of 34 studies on cannabis smoking and pulmonary function found no association between chronic use and airflow obstruction; however, it did find that long-term cannabis smoking leads to chronic cough, airway irritability, wheezing, bronchospasm, and bronchitis. Hyperreactive airways are a major cause of intraoperative complications. Chronic cough also puts a patient at risk of surgical wound dehiscence. Caution should be exercised with airway instrumentation and emergence from anesthesia, since any foreign substance in the airway can trigger reactivity leading to subsequent hypercarbia and/or hypoxia. Histamine-releasing agents should be avoided, and dexamethasone at oral or intravenous doses of 4 to 8 mg should be considered as prophylaxis if the patient is undergoing general anesthesia. Increased mucus production and the loss of cilia function from smoking can lead to a decreased capacity to clear airways. Albuterol, corticosteroids, and laryngeal topical anesthesia may all help to suppress cough and reactivity. If volatile anesthetics are used during induction, sevoflurane may offer the best conditions to prevent coughing because of its least pungent odor. However, all volatile anesthetics bronchodilate the airways, which make them useful to blunt reactive airway responses.
Although the literature suggests that cannabinoids inhibit inflammation and immune function, case reports of upper airway edema related to a swollen uvula (uvulitis or uvular angioedema) during general anesthesia have been described.34 Anesthesia practitioners should remain vigilant, monitoring for signs of airway edema. Stridor, decreasing oxygen saturation, hoarseness, dysphagia, and progressive dyspnea should alert the practitioner to potential edema. Again, prophylactic use of dexamethasone should be considered.

**Aspiration Risk.** As stated previously, THC administration significantly slows gastric emptying in human volunteers from an average of 30 to 120 minutes,29 creating an aspiration risk for the patient. Preoperative aspiration prophylaxis regimens of H2-histamine antagonists, proton pump inhibitors, and a nonparticulate antacid should be considered. General anesthesia should be instituted with a rapid-sequence induction with cricoid pressure followed by securing the airway with an endotracheal tube.35

**Interactions With Anesthesia Drugs.** Because THC is a CNS depressant, any use of substances that also depress the CNS may lead to synergistic effects. Alcohol, benzodiazepines, narcotics, or other sedative hypnotic drugs, all of which are CNS depressants, could interact with THC to further CNS depression28 (see Table). Increased sleeping times of barbiturates by cannabinoids have been reported.9 Although no studies were found establishing this synergistic relationship on CNS depression, the additive effects of these drugs when used with THC may warrant caution during anesthesia.

The effects of drugs causing respiratory or cardiac depression may be augmented by cannabis.28 In theory, drugs that induce tachycardia such as ketamine and anticholinergic medications should be used with caution in patients acutely intoxicated with cannabis to avoid any additive effects. Furthermore, cannabis has synergistic effects with inhalational anesthetics, which sensitize the myocardium to catecholamines, leading to a more profound response.9

Habitual marijuana users may require increased amounts of induction drugs to achieve adequate anesthesia, reflecting a cross-tolerance with these drugs. For example, propofol and volatile anesthetics require substantially higher doses for the patient who routinely uses marijuana. In a study by Flisberg et al,36 the authors concluded that patients with a history of regular cannabis use demanded significantly more propofol for induction of general anesthesia and showed a more inconsistent response to propofol during induction of general anesthesia. A similar case report described a patient with a history of regular cannabis use who required much larger-than-expected doses of propofol, thiopental, isoflurane, and sevoflurane (all of which were used) to maintain baseline hemodynamics during an anesthetic.37

One mechanistic reason for the increased requirement

<table>
<thead>
<tr>
<th>Drug or drug class</th>
<th>Reported interaction when used with marijuana (cannabinoids)</th>
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<td>Benzodiazepines</td>
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</tr>
<tr>
<td>Protease inhibitors</td>
<td>Reduced effectiveness related to decreased plasma drug levels (possible CYP3A-4 interaction)</td>
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<td>Selective serotonin-reuptake inhibitors</td>
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<tr>
<td>Sildenafil</td>
<td>Myocardial infarction (possible CYP3A-4 and CYP2C9 interaction)</td>
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<td>Theophylline</td>
<td>Reduced effectiveness related to decreased plasma drug levels (increased clearance)</td>
</tr>
<tr>
<td>Warfarin</td>
<td>Increased plasma concentrations (possible CYP2C9 interaction and/or altered protein binding)</td>
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<td>Anticholinergics</td>
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<td>Naltrexone</td>
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<td>Lithium</td>
<td>Increased plasma concentrations</td>
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<td>Neuroleptic antipsychotics</td>
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<tr>
<td>Corticosteroids</td>
<td>Enhanced immunosuppression</td>
</tr>
<tr>
<td>Propofol</td>
<td>Diminished effectiveness</td>
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Table. Drug Interactions With Marijuana14,17,27
Abbreviation: CNS, central nervous system.
for anesthetic agents in marijuana users is the impact of cannabis on the cytochrome P-450 system. Cytochrome P-450 2B6 is predominantly involved in the oxidation of propofol by human liver microsomes.\textsuperscript{13,28} Therefore, upregulation of cytochrome P-450 enzymatic induction caused by long-term marijuana smoking leads to a significant decrease in the patient’s response to propofol.

Additionally, THC possesses muscle relaxant properties.\textsuperscript{38} Therefore, potentiation of nondepolarizing muscle relaxants may occur. To control for these effects, slow, cautious administration of nondepolarizing muscle relaxants with peripheral nerve stimulator monitoring is recommended.

- **Cardiovascular Effects.** Sympathetic stimulation with concomitant increases in heart rate, often by as much as 30%, and mild to moderate hypertension can occur with small doses of cannabis.\textsuperscript{39} This can increase cardiac work and oxygen demand and can decrease oxygen delivery.\textsuperscript{7} Myocardial depression can also occur. Therefore, in the patient who has recently used marijuana, ketamine, pancuronium, atropine and epinephrine should perhaps be avoided because they increase heart rate. In this setting, β-blockers can be beneficial. However, caution should be exercised because high doses of cannabis can inhibit sympathetic activity with unopposed parasympathetic activity, leading to bradycardia and hypotension.\textsuperscript{39}

Although the mechanism is unclear, cannabis may interact with sildenafil-containing medications to cause myocardial ischemia and/or infarction.\textsuperscript{14,28} These patients need to be screened preoperatively for signs of ischemia: angina, new-onset shortness of breath, and ECG changes. Intraoperatively, continuous ECG ST-analysis aids in the detection of ischemia. Avoidance of tachycardia and maintenance of blood pressure within 20% of baseline is prudent.

- **Postoperative Considerations.** Anesthesia providers should be aware of the implications of cannabis use as an analgesic, as well as other postoperative concerns, such as withdrawal from long-term marijuana use.

- **Pain Control.** Cannabinoids have been shown to be efficacious in the treatment of chronic neuropathic pain.\textsuperscript{40-42} With the current national opioid crisis mandating a need to provide nonopioid choices, the addition of cannabinoids to the anesthesia care plan may reduce total opioid doses necessary for pain management in patients with chronic pain conditions.\textsuperscript{43} However, recent studies in which investigators evaluated adding cannabinoids to multimodal pain regimens for patients with chronic joint pain after total hip and knee arthroplasty surgery did not find a benefit to the addition of cannabinoids to the regimen. If one is adding cannabis to a treatment plan, frequent reassessment of pain scores and necessary adjustment will be important.\textsuperscript{44}

Although long-term cannabinoid use has been found to be effective as an additional analgesic for patients with cancer-related pain and chronic neuropathic pain, marijuana use appears to lower the pain threshold in surgical patients with acute pain, resulting in increased pain medication requirements. In some cases, high-dose cannabinoid use is associated with increased postoperative pain, additional doses of analgesia medications, and higher pain scores in the immediate postoperative period compared with nonusers.\textsuperscript{44} A case report described a patient with a history of long-term cannabis use who required nearly double the opioid dose of an average patient of the same body weight and height in the first 24 hours postoperatively.\textsuperscript{45} In a systematic review of the literature on the efficacy of cannabis in the management of acute pain, Stevens and Higgins\textsuperscript{46} concluded that cannabis was ineffective in the management of acute pain.

- **Other Concerns.** Long-term marijuana users can experience withdrawal symptoms when they abruptly discontinue use. Withdrawal symptoms include anxiety, irritability, fatigue, despondency, and lack of appetite.\textsuperscript{47} Monitoring for withdrawal may be challenging because of short postoperative stays and confounding factors such as generally not feeling well because of pain or nausea and vomiting. It may be prudent to discuss with the patient during postoperative discharge the symptoms of withdrawal and when to seek medical assistance.

It is known that the affinity of carbon monoxide for hemoglobin is greater than that of oxygen, resulting in decreased oxygen-carrying capacity and a left shift of the oxyhemoglobin dissociation curve. With carboxyhemoglobin levels in marijuana smokers 5 times that of tobacco smokers,\textsuperscript{27} it can be reasoned that, as seen in tobacco smokers, wound healing would be inhibited. Special precautions should be exercised to monitor wound health and treatment of complications in a timely manner.

In the obstetric population, cannabis intake is associated with preterm labor. Premature neonates exposed in utero may be stillborn and/or have cerebral palsy. Cannabis use by the mother may lead to an increase in neonatal infections due to immune suppression. Prenatal exposure to cannabinoids does not seem to affect fetal growth but is potentially associated with neurologic morbidity.\textsuperscript{48}

**Recommendations**

Surgical patients should be advised to avoid consuming or inhaling cannabis for as long as possible before surgery because cannabis can be found in the bloodstream more than 30 hours after consumption and interactions with anesthetic drugs and adjuvant drugs administered during an anesthetic are not reliably predictable. If consumption has occurred, anesthetic care should be planned to treat or avoid side effects.

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