A Review of the Pharmacology and Anesthetic Implications of Cannabis

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Cannabis is now legal, for medical and/or recreational use, in numerous states. Although the cultural shift in acceptance of cannabis is apparent in the public, that sentiment has not necessarily translated to healthcare professionals. As anesthesia providers, we must understand the pharmacology of cannabis and its effects on physiology to provide safe anesthetic care to patients who consume it. The purpose of this article is to describe cannabis and its pharmacologic and physiologic effects and to review the anesthetic implications of its short-term and long-term use.

Keywords: Anesthetic implications of marijuana, cannabinoids, cannabis, cannabis-drug interactions.

Objectives
At the completion of this activity, the learner will be able to:
1. Review the history of cannabis.
2. Identify the pharmacokinetics of cannabis.
3. Describe the system-wide pharmacodynamics of cannabis.
4. Explain the anesthetic implications of cannabis.
5. Detail drug interactions with cannabis of particular relevance.

Introduction
Since the first article on cannabis (marijuana) appeared in this journal in 1980, the context of cannabis use and its availability in the United States have changed drastically. The use of cannabis draws responses from healthcare providers that are often vastly different from those regarding other substances taken by patients. Our perceptions are sometimes based on emotional responses or opinions of cannabis as a recreational and/or illicit drug. Healthcare providers can be subject to allowing their personal beliefs about their patients’ drug intake to affect the care they provide, as is sometimes the case when patients have histories of alcohol or opioid misuse. Cannabis has been stigmatized through media portrayals by its perceived role in criminal activity. This, combined with law enforcement and other sociopolitical issues, makes such a change in perception difficult.

In addition to treating all patients with unbiased empathy, Certified Registered Nurse Anesthetists (CRNAs) are in an important position to evaluate patient use of all substances. They do this through the attainment of an accurate preoperative assessment and development of an anesthesia plan based on consideration of potential drug interactions and altered physiologic responses. Although the literature provides insight related to the presumed pharmacologic mechanisms and physiologic implications of cannabis, there is a dearth of information related to the anesthetic implications for patients who receive it. Because of the drugs’ Schedule I designation by the US Drug Enforcement Administration (DEA), few controlled studies exist; thus, much of the literature informing anesthetic implications comprises isolated case reports, many of which are well over a decade old. Furthermore, much of the published data is the result of research funded by third parties with a vested interest either for or against legalization of cannabis. This review of the literature describes cannabis and its pharmacologic and physiologic effects and reviews the anesthetic implications of its short-term and long-term use.

History
Cannabis administration for medicinal purposes dates back centuries, with evidence of its use appearing in early Chinese, Hindi, and Greek literature. Documentation of its application in Western medicine did not appear until 1839, when W. B. O’Shaughnessy first described the benefits of cannabis for the treatment of rabies, tetanus, cholera, infantile convulsions, and delirium tremens. Cannabis
was also widely used as a treatment of many other maladies based on its analgesic, sedative, anti-inflammatory, anti-spasmodic, and anticonvulsant properties, all of which are reemerging as therapeutic uses for the drug today.³,⁵

Cannabis was criminalized federally through the Harrison Narcotics Act in 1914. In 1970 the Controlled Substances Act classified cannabis as a Schedule I drug, like heroin or LSD, by the DEA claiming that there is “no accepted medicinal use” of cannabis.³ As a result, extreme limitations are placed on its use in controlled trial research.³,⁴ Attitudes regarding a therapeutic value for cannabis in the 1980s began to change when physicians and their seriously ill patients reported the value of cannabis in managing pain, reducing nausea and vomiting associated with chemotherapy, preserving vision in glaucoma, and reversing anorexia due to AIDS.²-⁴

Although the lack of both controlled drug trials and US Food and Drug Administration (FDA) approval of cannabis has also created skepticism among many medical professionals as to the benefits of the drug, reputable medical and nursing journals are beginning to publish articles recognizing the potential therapeutic value of cannabis (Table 1). As federal and state laws evolve, greater emphasis on informing nurse anesthetists on physiologic, pharmacologic, sociologic, and mental health implications of cannabis use will be needed.

### Pharmacology

Cannabis is a complex plant that contains numerous compounds which contribute to its many characteristics. Cannabinoids are the biologically active compounds responsible for the physiologic effects of the drug through their activity at cannabinoid receptors located throughout the body. Delta-9-tetrahydrocannabinol (THC) is the cannabinoid that is most frequently associated with the mood-altering effects or “high” of cannabis; however, numerous other cannabinoids are present that exhibit pharmacologic properties of physiologic benefit without producing this effect.⁴ There are more than 100 biologically active non-THC cannabinoids⁴,⁶ and hundreds of terpenes, flavonoids, and terpenoids identified in cannabis plants.⁷,⁸ Notably, cannabidiol (CBD) and cannabinol are cannabinoids that exhibit wide pharmacologic activity and lack psychoactive properties.⁶,⁹ Lipophilic, plant-derived cannabinoids, including THC, mimic numerous endogenous compounds referred to as endocannabinoids. These naturally occurring ligands, along with cannabinoid receptors and enzymes, comprise the endocannabinoid system. Identified in 1994,⁸ the endocannabinoid system is a complex homeostatic modulator involved in the functioning of most major organ systems in the body and many other functions,²,⁷,⁸,¹⁰ including the following¹¹:

- Motor control and coordination
- Cognition, memory, emotion, behavior
- Autonomic nervous system modulation
- Immunity, inflammation, and antitumor effects
- Antinociception
- Endocrine, reproduction, sleep, and temperature regulation
- Gastrointestinal motility and metabolism
- Intraocular pressure

Arachidonylethanolamine (anandamide) and 2-arachidonoylglycerol (2-AG) are the 2 primary endogenous ligands that cannabinoids such as THC and CBD share similar properties with⁷,¹¹,¹² (Figure). The G-protein ligand-receptor interaction causes a variety of ion channel activities, such as inhibition of adenylate cyclase, inhibition of calcium channels, opening of potassium channels, and decreased production of cyclic adenosine monophosphate (cAMP).⁴,¹³ Cannabinoids acting on these receptors eventually modulate release of dopamine, acetylcholine, glutamate, serotonin, and other neurotransmitters.¹¹,¹³

The presence of endogenous cannabinoids and receptors may be considered similar to the endogenous opioid complexes and receptor system. CB1 receptors are present in all body tissues with higher concentrations in cardiovascular, immunologic, adrenal, ocular, and gastrointestinal

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Table 1. Common and Less Common Therapeutic Applications of Cannabinoids²-⁴,¹⁵
Abbreviations: ADD/ADHD, attention-deficit disorder/attention-deficit/hyperactivity disorder; PTSD, posttraumatic stress disorder.
nal systems; however, they are most heavily concentrated in the central nervous system, with abundant receptors in the brain.\(^7,8\) Whereas THC exhibits an agonist effect on these brain receptors, CBD affects CB2 receptors, which are primarily present in peripheral locations, including immune cells such as macrophages and mast cells.\(^4,11,14\) This contributes to the well-established role of CBD in immunity, inflammation, and pain. CB2 receptors are highly induced and increase exponentially as a result of tissue injury or during inflammatory processes.\(^12\)

Although it has very low affinity for CB1 receptors, CBD does not impair cognitive function but has been shown to potentiate, attenuate, and modulate various effects of THC.\(^2,4,7\)

**Pharmacokinetics**

Absorption, distribution, metabolism, and elimination considerations can vary depending on the dose and mode of consumption.\(^3,14\) According to MacCallum and Russo,\(^15\) “Absorption has the most variability, and is affected by product lipophilicity, bioavailability as well as the inherent organ tissue differences (i.e., alveolar, dermal vs. gastric).” Once cannabinoids enter the systemic circulation, they are rapidly distributed to highly vascularized organs such as the brain, liver and lungs.\(^13\) Because it is highly lipophilic, cannabis can be administered via inhaled, oral, buccal, intramuscular, rectal, topical, and transdermal routes. Inhaled administration routes for cannabis include conventional inhalation, vaporization, and aerosolization of leaves, oils, and waxes. It is rapidly absorbed into the bloodstream after inhalation; peak effects occur within minutes, with rapidly declining effects over 30 minutes and a plateau lasting 2 to 4 hours.\(^4,10\) In addition, oral and buccal administration is achieved through the placement of cannabis in edible food forms. Because of prolonged gut absorption, orally ingested products can exhibit a slower peak onset of 1 hour or more, with effects that can last 5 or 6 hours.\(^10,16\) Because of delayed onset of a perceived effect, individuals may ingest additional cannabis product, potentially leading to intoxication through what can be described as stacking of dosage, a term used to define the perils of self-administering without waiting for the onset of effects. Oral cannabis products undergo extensive first-pass metabolism via liver enzymes, notably cytochrome (CYP) P450, producing active metabolites that can have more pronounced effects than the parent compound, THC.\(^10\) Many of these metabolites play a substantial role in the physiologic activity expressed by the drug consumed. Some cannabinoids may induce CYP450 enzymes, including CYP1A2, CYP2B6, CYP2C9, CYP2D6, and CYP3A4.\(^4\) Others may inhibit them; therefore, there exists a potential unpredictable effect on the metabolism.

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**Figure. Cannabinoid Receptor Interaction\(^4,7,10-13\)**

The 2 primary endogenous ligands similar to exogenous cannabinoids (CBDs) are arachidonylethanolamide (AE) and 2-arachidonoylglycerol (2-AG). Interactions at the G-protein ligand-receptors cause a variety of ion channel activities, such as inhibition of adenylate cyclase, inhibition of calcium channels, opening of potassium channels, and decreased production of cyclic adenosine monophosphate and modulation of the release of dopamine, acetylcholine, glutamate, serotonin, and other neurotransmitters. CBD has lower affinity, but its binding effects are believed to be similar to 2-AG.

Abbreviation: THC, Δ9-tetrahydrocannabinol.
of other drugs. Because cannabinoids can accumulate and sequester in fatty tissue, complete elimination from the body can take several days. Traces of cannabinoid metabolites can be found in urine for more than 30 days in daily cannabis users. Within 5 days, up to 90% of THC is eliminated, mostly as metabolites. More than 65% is eliminated through the feces, and approximately 20% is eliminated in the urine.

- **Pain and Neurologic Effects.** The interaction of THC with CB1 receptors in the brain affects memory, perception, movement, and mood, typically resulting in mood enhancement. Dysphoria is a dose-dependent negative effect of THC that can occur in susceptible individuals. All cannabinoids, including THC, exhibit a high therapeutic index. Unlike opioid receptors, there are no cannabinoid receptors in the brainstem, so respiratory suppression does not occur, even at toxic doses; however, unwanted psychotropic and cognitive effects may be exhibited. According to Aggarwal et al, in more than 4,000 years of documented use of cannabis, there has never been a reported death due to cannabis overdose.

The endocannabinoid and endogenous pain receptor systems are in close proximity and therefore influence each other. CB1 receptors are expressed throughout the central nervous system. After THC activates κ- and Δ-opioid CB1 receptors, it demonstrates antinociceptive properties. The interaction of CBDs with CB2 receptors in the dorsal horn has been shown to cause a reduction in inflammation and the modulation of pain. Cannabidiol is recognized for its anti-inflammatory properties by its interaction with macrophages and mast cells. Selective cannabinoids may have a role as adjunct therapy for refractory neuropathic pain. Cannabis also suppresses hyperalgesia and allodynia that is associated with some forms of chronic pain. The potential for a positive synergistic interaction between cannabis and gabapentin may exist to enhance analgesia, particularly for those experiencing allodynia. Commonly, cannabis is used in managing pain and other debilitating symptoms associated with inflammatory diseases such as Crohn disease and colitis. Cannabis has shown efficacy in treating pain associated with cancer, reducing overall opioid requirements, and improving nausea resulting from chemotherapy.

- **Cognitive Effects.** Effects on the central nervous system mediated by THC can include fatigue, sedation, and vertigo; however, these are typically avoided when dosing begins low and is titrated slowly. Tolerance to psychoactive effects eventually occur. In higher concentrations THC may cause psychoactive effects, referred to as the “high” associated with use of the drug. Although this may be a desired effect, due to varying concentrations of THC, the high can be unwanted and stress inducing, especially if it is accompanied by panic, paranoia, and anxiety of which some individuals are predisposed to. The potential for adverse cognitive effects have been suggested and can differ between short-term and long-term use. Acute use effects may include acute impairment of memory, mood, coordination, and judgment. Potential effects of long-term use, such as cannabis use disorder and cognitive impairment have been reported. Adverse health effects, including dependency and addiction, are also addressed extensively by Volkow et al from the National Institute on Drug Abuse of the National Institutes of Health.

- **Respiratory Effects.** Inhaled cannabinoids quickly enter the bloodstream from the lungs along with other noncannabinoid chemicals just as those associated with tobacco smoking do, with the exception of nicotine. These chemicals can act as bronchial irritants, similar to cigarette smoke, causing coughing, hoarseness, wheezing, dyspnea, and sputum production. If the patient smokes both cannabis and cigarettes, these symptoms can be more pronounced. The literature frequently cites the difficulty differentiating the impact of inhaled cannabis from tobacco as the incidence of using both substances by individuals is noted to be high and is often uncontrolled for in data analyses. In addition to bronchial irritability caused by cannabis inhalation, the concern for the potential development of pneumonia, pneumomediastinum, and pneumothorax has been discussed in literature. Unlike tobacco use, a causal relationship has not been established conclusively between cannabis smoking and the development of lung cancer or chronic obstructive pulmonary disease.

Although it has been demonstrated that inhaled cannabis causes bronchodilation that persists for up to 1 hour, conventional thinking has suggested that long-term use of inhaled cannabis can result in changes in pulmonary function similar to those seen in tobacco users, including decreased forced expiratory volume in the first second of expiration (FEV1) and forced vital capacity (FVC) as well as decreased diffusion capacity. These findings, however, have been disputed. Ware and Ziemianski conducted a 1-year prospective study of 215 participants and, after adjusting for tobacco smoking and other covariates, did not find a significant change in pulmonary function parameters of functional residual capacity or total lung capacity among the cannabis users. They did report reductions in residual volume and a mean decline of 54 mL in FEV1 and a mean decrease of 0.78% in the FEV1/FVC ratio but no change observed in FVC. In a 20-year longitudinal study, Pletcher et al compared pulmonary function between tobacco smokers, marijuana smokers and nonsmokers. The researchers followed up 5,115 participants over a 20-year period, evaluating pulmonary function at 2-, 5-, 10-, and 20-year marks. The authors concluded that occasional and low cumulative marijuana use was not associated with adverse effects on pulmonary function.

In 1996, Mallat et al reported an isolated incident of
uvular edema, which the authors theorized to be a result of recent inhaled cannabis in a 17-year-old patient. Other individual case reports of uvular edema have also been reported. Edible forms of cannabis avoid the respiratory effects yet introduce risks such as hallucinations, nausea and vomiting, and renal failure (isolated reports in 6 patients) from overmedication.

- **Cardiovascular Effects.** Cannabis containing THC can induce a dose-dependent increase in heart rate from 20% to 50%, with peak elevation occurring within 15 to 20 minutes of inhalation. Heart rate increases may also be a result of parasympathetic nervous system inhibition, reflex tachycardia resulting from vasodilation and THC action on CB1 endocannabinoid receptors. A resultant increase in myocardial oxygen demand occurs. Orthostatic hypotension due to a decrease in peripheral vascular resistance is also a potential side effect that has resulted in syncope in some individuals. This is particularly evident when high doses of cannabis are administered. In such cases, inhibition of sympathetic activity results in unopposed parasympathetic activity, which may lead to bradycardia, and hypotension. Tachycardia is associated more with short-term dosing of cannabis as opposed to bradycardia, which predominates with long-term use. It has been established that tolerance to the cardiovascular effects of THC occurs within several days to a few weeks.

A large retrospective study evaluating recreational use of inhaled cannabis suggested that the lifetime odds of an acute myocardial infarction are increased, but the overall odds of mortality due to an acute myocardial infarction were not increased significantly. Kattoor and Mehta identified that cannabis exhibits both proatherogenic and antiatherogenic effects on the vasculature by its interaction with CB1 and CB2 receptors, and they acknowledge that inconclusive evidence exists showing a causal relationship of atherosclerosis and cannabis.

**Anesthetic Implications**

Although there is a lack of published guidelines and limited scientific research directly exploring anesthetic implications of cannabis use, the aspects of the drug’s pharmacology and physiologic effects previously discussed can guide the anesthesia practitioner. An electronic search of the literature for this review was completed using the following databases: National Library of Medicine’s PubMed/MEDLINE, Cochrane Library, Ovid MEDLINE, Cumulative Index to Nursing & Allied Health Literature (CINAHL), and ProQuest Nursing and Allied Health Database. The following subject headings and their combinations were used without date restriction: cannabis, cannabinoind, marijuana, anesthesis implications, and pharmacology. Articles were reviewed for relevance. Most articles reviewed and ultimately included in the review were not directed solely at anesthesia implications or application to anesthesia practice. Of the articles referenced, only 8 directly address anesthesia and/or perioperative application or were published in anesthesia journals, and 2 articles address pain management.

Articles directed at perioperative implications were reviewed for the strength of their evidenced as described in the *Johns Hopkins Nursing Evidence-Based Practice Evidence Level and Quality Guide*. Use of this tool guided the authors to develop observations and considerations for anesthetic implications (Table 2). Of the articles reviewed, none were found to be evidence level 1 or 2. The remainder were designated level 3 reviews of literature. Because of the paucity of specific anesthetic care guidelines in caring for the patient who uses cannabis, in the present article, 2 experienced clinicians offer their review of the literature and personal observations in an effort to establish safe clinical anesthesia practices.

**Preoperative Assessment**

Ideally, preanesthetic patient education regarding cannabis use before surgery should occur. Just as it is desirable for patients who smoke cigarettes to abstain for at least 72 hours before surgery and anesthesia, theoretically it would also be advantageous for users of inhaled cannabis to abstain. However, if patients are using cannabis for medicinal reasons, it may be unrealistic if not undesirable for a patient to stop taking his or her regularly scheduled dose. Just as it is essential to obtain a detailed history of prescribed drugs, it is equally crucial to use the same diligence in evaluating use of recreational drugs and alcohol. The anesthesia practitioner must establish a trusting relationship with the patient to obtain information regarding use or misuse of alcohol or illicit drugs. Although the stigma of cannabis may be subsiding in some settings, reluctance on the part of the patient to discuss his or her use of the drug may be of concern, so the CRNA must impart a nonjudgmental empathetic approach to the interview. This should always include inquiry as to when the drug was last taken. Although dose information of cannabis may be difficult to obtain, the route and frequency of use should be documented. Because inhaled cannabis peaks in minutes, with effects lasting up to 2 to 4 hours, an accurate assessment of the patient’s last administration as well as frequency and duration of use is warranted.

Ascertaining between long-term and occasional use as well as the presence of acute intoxication is imperative because of the differences in systemic effects previously noted and further explained later. Considerations for potential additive sedative properties must be taken for the patient currently sedated by recent use of cannabis. Conversely, long-term users of cannabis have demonstrated higher sedative dose requirements.

A thorough history and physical examination with particular attention to respiratory, cardiac, and cognitive function is crucial. The standard airway assessment

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should include notice of the presence of oropharyngeal and uvular inflammation and edema because it has been reported following recent cannabis inhalation. The need for further advanced testing such as for pulmonary function or cardiovascular status should be guided by the history and physical findings, as would occur with all patients, since published recommendations specific to long-term cannabis use are not available.

**Airway and Respiratory Considerations**

Recent preoperative inhaled use might result in a more reactive upper airway as well as bronchial airway mucosal irritation. This concern is magnified in patients with asthma or other reactive airway issues, as well as those who also smoke tobacco. A heightened level of concern should exist, particularly when anesthetizing these patients undergoing procedures involving the airway, such as bronchoscopy or gastroscopy in which airway stimulation occurs. Although cannabis use is often associated with dry mouth, administration of glycopyrrolate preoperatively may be helpful in reducing airway secretions that can contribute to irritability in patients who regularly smoke tobacco. It has been suggested that long-term inhalation of cannabis is associated with increased sputum, so it may follow that glycopyrrolate may be useful for these patients as well. Other pharmacologic interventions such as the administration of intravenous lidocaine or nebulized bronchodilator administration may help mitigate airway sensitivity. Supraglottic airway devices may be warranted for airway maintenance. Unless contraindicated, deep extubation may be appropriate for patients in which the potential for an irritable airway is suspected. Although cannabis does not produce respiratory depression, dose-dependent sedative and psychomotor properties of formulations containing THC should be considered, and additive effects with conventional anesthetic and sedative drugs may be of concern.

**Cardiac Considerations**

Since the cardiovascular effects of cannabis can vary based on dose and frequency and duration of use, these implications on anesthesia can also vary and may be difficult to predict. Because of the anticholinergic effects, the CRNA must be cognizant of the potential for increased sympathetic or sympathomimetic activity with resultant tachycardia and increased blood pressure following recent or high-dose consumption, particularly in the cannabis-naïve patient. Avoidance of anesthetics and adjuncts that also increase heart rate or blood pressure such as ketamine, epinephrine, or atropine may be warranted. Hypotension as a result of vasodilation may respond to fluid resuscitation or vasopressor therapy. Conversely, if parasympathetic activity prevails because of higher dose ingestion, bradycardia may require pharmacologic management.

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| **Respiratory**  | Short- or long-term     | Bronchial irritation and reactive airway; increased airway secretions | • Give preoperative antisialagogue  
• Administer preoperative aerosol treatment if there is active congestion or wheezing  
• Consider use of a supraglottic airway over endotracheal intubation  
• Consider deep extubation |
| Route: inhalation | Short-term              | Sympathetic effects of tachycardia and hypotension (note: tolerance to these effects occurs with long-term use) | • Avoid agents that cause tachycardia  
• Provide vasopressor support as needed  
• Treat symptomatic bradycardia |
| Cardiovascular   | Short- or long-term     | Potential for sympathetic blockade and bradycardia with high doses and long-term use | • Give rapid sequence induction  
• Give aspiration prophylaxis |
| Route: inhalation, oral, other methods | Short-term | Aspiration potential if solid food containing cannabis was ingested | • Carefully titrate enzyme-dependent anesthetic agents  
• Patient may require higher doses of induction agents |
| **Gastrointestinal** | Short-term | Enzyme inhibition or induction with unpredictable metabolism of anesthetic agents | • Carefully titrate sedatives and anesthetic agents  
• Patient may require higher doses of induction agents |
| Route: oral      | Long-term               | Unpredictable additive or inhibitory interaction with sedatives | • Carefully titrate sedatives and anesthetic agents  
• Patient may require higher doses of induction agents |
| **Liver and Renal** | Long-term               | Enzyme inhibition or induction with unpredictable metabolism of anesthetic agents | • Carefully titrate enzyme-dependent anesthetic agents  
• Patient may require higher doses of induction agents |
| Route: inhalation, oral, other methods | Short-term | Psychoactive activity | • Consider obtaining informed consent from a proxy |

Table 2. Anesthetic Considerations of Cannabis Use

13,29
Drug Interactions and Other Considerations

Drug interactions may be the result of pharmacokinetic or pharmacodynamic mechanisms. Because THC and its numerous metabolites are metabolized in the liver through CYP450 systems, the possibility exists of pharmacokinetic drug interactions through either the inhibition or induction of these enzymes. Regardless of the route, long-term use of cannabis may result in enzyme induction and may produce shorter durations of action or increased dosing needs of other drugs relying on CYP450 systems for metabolism. Conversely, inhibition of metabolizing enzymes may also occur. As a result, prolonged duration or enhanced effects of anesthetic drugs may be seen in some patients. In essence, unpredictability and a wide variation of responses to induction agents can be seen.

It is well known that many of the anesthetic and adjunct drugs administered in the perioperative setting also rely on CYP450 for metabolism. Although there is a scarcity of literature documenting specific drug interactions with THC, the inhibition of metabolism of the drug clobazam, a benzodiazepine used in the treatment of epilepsy, has been shown. It may follow that similar responses are possible with other benzodiazepines.

The potential pharmacodynamic drug interactions with cannabis may be more predictable. Other central nervous system depressant drugs, including propofol, benzodiazepines, opioids, and volatile agents, may potentiate the sedative effects of THC if the patient has recently administered cannabis. If the patient frequently uses cannabis, higher doses of induction agents may be warranted. Other effects that may be mediated through cholinergic, adrenergic, and endocannabinoid system responses can include changes in heart rate, peripheral vasodilation, dry mouth, decreased intraocular pressure, and conjunctival vascular congestion.

There may exist a potential for alterations in coagulation associated with cannabis use. Multiple studies cite inhibition of platelet aggregation, whereas others suggest an association with an increased tendency toward venous thrombosis formation. As a result, the anesthetist should have a heightened awareness for an increased risk of surgical and postoperative bleeding.

Cannabis use in elderly individuals has been well documented. Use by elderly patients may include recreational or medicinal for multiple physical or psychological conditions. The use of THC and CBD for managing agitation from dementia has shown promise. Age-related physiologic and pharmacokinetic issues and their associated anesthetic implications should be considered. The potential for previously noted cardiovascular, respiratory, and cognitive issues may be magnified in the elderly. The intuitive concerns for the presence of impaired liver or renal function in the elderly or any population may affect metabolism and elimination of cannabis.

Opportunities for Future Research

Further research is needed, including controlled, prospective studies, into specific anesthetic outcomes based on technique and specific drugs administered to patients who ingest cannabis. Just as anesthetics providers are becoming more familiar with multimodal pain therapy, it is possible that cannabis, particularly selective cannabinoids, may have a role in treating postoperative pain. Beaulieu et al suggest that there may be a utility for THC and CBD oral mucosa sprays for postoperative pain control in patients who cannot ingest oral pain medications, such as after abdominal surgery. Because cannabis has long been advocated for treating chemotherapy-induced nausea and vomiting, it stands to reason that there may be opportunities for its use to manage postoperative nausea and vomiting.

Discussion

Regardless of the intended use of cannabis, as with all foreign substance ingestion, we must consider the possibility of anesthetic implications. The pharmacology and physiologic effects of cannabis are varied and complex and, although often considered benign, it is imperative for anesthesia practitioners to be informed while planning the perioperative care of their patients. Many healthcare professionals, including nurse anesthetists, may not appreciate the pharmacologic properties of the drug nor recognize the potential impact, or lack of impact, on anesthetics.

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DISCLOSURES
The authors have declared no financial relationships with any commercial entity related to the content of this article. The authors did not discuss off-label use within the article.