

Cannabis (marijuana) and anesthesia

BY HUU TRAM ANH NGUYEN, MD, FRCPC

Cannabis (marijuana) has been used for a long time. In 2700 BC, the use of cannabis for malaria, constipation, and the pain of rheumatism was described. In 1839, the analgesic, antispasmodic, and anti-inflammatory properties of cannabis as a medicinal substance were promoted. However, cannabis became popular as a recreational drug only in the 1960s. Contrary to widespread belief, especially among marijuana users, cannabis is far from innocuous. Acute and chronic cannabis use has many effects, some of them major, even fatal. The problems of addiction, causing illness, accidents, and violence cannot be considered negligible, and affect approximately 30 million Americans. This issue of *Anesthesiology Rounds* deals with the pharmacology, as well as the acute and chronic effects of cannabis on humans, followed by a proposed anesthetic plan for cannabis users.

PREVALENCE

The recreational use of cannabis has risen sharply since the 1980s. In the United Kingdom,¹ > 40% of students between the ages of 15 and 16, and 60% of 18-year-old students have tried cannabis at least once. Among university students, 20% report using it at least once a week. Medical students are no exception: 41% use it, and 10% use it at least once a week. An estimated 30% of young doctors in the United Kingdom use cannabis and 10% are chronic users. Moreover, at least 1% of school-age youths smoke daily. The other groups with a high prevalence rate are alcohol and/or illicit drug abusers and psychiatric patients. In Australia,² 10% of cannabis users use it daily and 20%-30%, weekly. According to a 1999 American national report on substance abuse,³ cannabis is the most commonly used illicit drug. Approximately 18 million Americans used it in 1998, and >30% of Americans >12 years have tried it at least once.

According to a report on psychotropic drug use published by the Quebec government in November 2003, Quebecers are heavy drug users, much more so than the residents of other provinces. Due to the growing popularity of cannabis, drug use has doubled in a decade, from 6.5% of the population in 1989 to 13.5% in 1998. The rate is estimated to be as high as 39.2% in the 12- to 17-year-old age group, but drops off sharply after the age of 30-35.

PHARMACOLOGY OF CANNABIS

Sources and components of cannabis

The names "cannabis" (British) and "marijuana" (American) both refer to the *Cannabis sativa* plant. Different parts of the plant are used and the various chemical compounds it contains – called "cannabinoids" and "noncannabinoids" – have been identified (Figure 1). Among the 61 types of cannabinoids identified to date, delta-9-tetrahydrocannabinol (THC) is the most potent psychoactive compound and the prime ingredient responsible for the effects of cannabis. The synthetic cannabinoids nabilone (Cesamet®) and dronabinol (Marinol®) are both used to treat the nausea and vomiting that invariably accompany chemotherapy and may have certain beneficial effects in treating chronic neuropathic pain. This article deals solely with natural cannabis.

There are presently three main preparations of cannabis based on the parts of the female plant used, and these three types determine the concentration of THC

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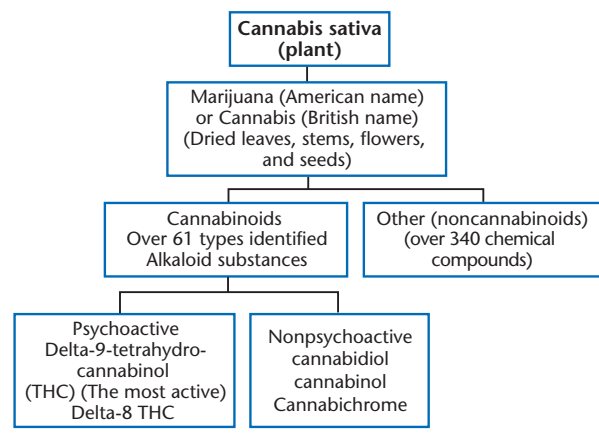
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FIGURE 1: Origins and components of cannabis

(Table 1). It is worth noting that growing cannabis has become increasingly sophisticated in order to obtain crops with a high level of THC.

Pharmacokinetics of cannabinoids

Approximately 50% of the THC and other cannabinoids in a marijuana “joint” (cigarette) enter the lungs after a single inhalation. The remaining 50% are lost to volatilization and burning.¹ The quantity absorbed depends on the style of inhalation. With experienced smokers who inhale deeply and then hold their breath before exhaling, nearly all of the cannabinoids in the smoke enter systemic circulation.

With inhalation, the psychotropic effects are perceptible within minutes or even seconds. The peak effect of cannabis is reached in 30 minutes and its activity lasts 2-3 hours. With oral ingestion, the onset of cannabis action is slower (30 to 120 minutes), its bioavailability lower (6%-20% vs. 18%-50% for inhalation), the peak plasma concentration is much lower, but the effect is longer (5-6 hours). Once in systemic circulation, cannabinoids are distributed to richly vascularized organs (brain, lungs, liver etc.). Highly liposoluble, cannabinoids accumulate in adipose tissue and are then slowly redistributed to other areas. Elimination half-life is 56 hours for chronic users and 28 hours for occasional users.⁴ Because of sequestration in adipose tissue, complete elimination of a single dose of cannabis can take 30 days and there is a risk of accumulation for chronic users. Cannabinoids are primarily (80%) metabolized by the hepatic cytochrome P 450 (more specifically the CYP2C and 3A4 subfamilies). Twenty different metabolites have been identified, some of which are psychoactive. Renal elimination accounts for 20%.

Pharmacodynamics of cannabinoids

Researchers have identified two specific types of cannabinoid receptors: CB₁ and CB₂.^{5,6} The CB₁ receptors are very heavily concentrated in the central

TABLE 1: Primary cannabis preparations¹

Form	Components	Amount of THC (approximate values, widely variable)
Marijuana “grass”	Dried stems, leaves and flowers of the female plant (cigarette/joint)	500-1000 mg of marijuana 1%-5% THC (10-50 mg)
Hashish	Dried resin secreted by the stems and flowers of the plant (Pipe/mixed with tobacco)	6%-20% THC
Hashish oil	Oil extracted from the flower stalks	30%-60% THC

THC = delta-9-tetrahydrocannabinol

nervous system (CNS). Clinically, these receptors are found in the cognitive zone, the regions related to memory, emotion, anxiety, pain, sensory perception, as well as the centres for nausea and vomiting, appetite, motor coordination, and endocrine and immune functions. The CB₁ receptors have a constitutive expression. Moreover, humans have certain endogenous ligands (or endocannabinoids) such as arachidonylethanolamide (AEA), also known as anandamide, and 2-arachidonylglycerol (2-AG). Anandamide, a derivation of arachidonic acid first described in 1992, produces effects similar to THC, but has a less potent agonist action and a shorter half-life. The selective antagonist of the CB₁ receptors has also been identified: SR 141716A.

The peripheral CB₂ receptors, on the other hand, are concentrated in the immune and lymphoid systems (spleen, tonsils, mast cells, and macrophages). Unlike CB₁, the CB₂ receptors tend to be induced by inflammation. Their endogenous ligands are 2-AG and palmitoylethanolamide (PEA), and their selective antagonist is SR 144528. CB₁ and CB₂ are G protein-coupled receptors^{1,7} and their activation inhibits adenylylase. They interact heavily with the opiate, GABA, dopaminergic, noradrenergic, serotonergic, and cholinergic receptors, the glucocorticoid axis and the prostaglandin system.

ACUTE EFFECTS OF CANNABIS

The acute effects of cannabis are located primarily in the CNS. The risk of acute toxicity is low and no death caused by direct toxicity related to cannabis has been documented. Nonetheless, a few cases of coma have been caused from accidental ingestion by children.¹ Euphoria is the most sought-after effect, the magnitude of which depends on the dose, method of administration, expectations, environment, and the user's personality. Other effects are dysphoria (anxiety, panic, paranoia, agitation, depersonalization, illusions, loss of control) and sedation, as well as flashbacks that can persist for days or

weeks after a single exposure to cannabis. After an initial period of excitation, a generalized depression of the CNS occurs, leading to lethargy and sleep. A physical state of inertia with ataxia, dysarthria, and general disequilibrium that can last for hours has also been observed. Problems with short-term memory recall, attention deficit, and speed of thought have also been documented. Altered perceptions of colour and sound have also been noted, along with distortions of visual, temporal, and spatial perception. Sometimes cannabis produces a psychotic state in users (delirium, confusion, auditory and visual hallucinations, possibly extending to paranoid or manic states).

Psychomotor performance after a single dose of cannabis can become hazardous. Cannabis is the drug most frequently involved in road accidents: contributing to 10%-20% of traffic fatalities in the United Kingdom, 40% in the United States, and 60% in Norway. In 80% of those cases, no alcohol was found. After a pilot had consumed cannabis a few hours before take-off, his airplane crashed in 1991.¹ Two fatal train accidents have been attributed to the use of cannabis. Studies on automobile driving and aircraft flying under real conditions or in a simulator have established numerous harmful effects of cannabis, such as slower response time, poor detection of light stimuli, poor oculomotor activity, errors between braking and accelerating, difficulty with speed control, poor judgment, etc. In addition, the potential for violence and crime may be higher among certain predisposed individuals (the words "assassin" and "hashish" have the same etymology). A study in an American prison found that 30% of 73 inmates convicted of homicide had used cannabis in the 24-hour period prior to committing their crime.¹

CHRONIC EFFECTS OF CANNABIS

Addiction and withdrawal reaction

Like all drug abusers, cannabis users run the risk of addiction and the cannabis withdrawal syndrome clinically resembles that of opiates and alcohol. In fact, the prevalence of the cannabis withdrawal syndrome is 16%-29%. An estimated 10,000 Americans a year seek treatment for cannabis addiction.¹

Long-term cognitive impairment

Although structural anomalies, especially in the hippocampus, amygdala, and septal areas have been found in the brains of Rhesus monkeys exposed to doses comparable to those of humans, no gross anatomical anomaly has been documented in humans. Yet, the attention deficit persists with only partial recovery among abstainers.¹

Effects on the cardiovascular system

The effect on the autonomic nervous system depends on the dose. A small dose produces tachycardia with little or no change in blood pressure.

Oxygen consumption can increase by up to 30%. In large doses, orthostatic hypotension and even low blood pressure, syncope, bradycardia, and a drop in body temperature occur.^{1,8} These effects are generally quite easily tolerated by healthy youths. Yet a few cases of ischemia, myocardial infarction, and transitory cerebral ischemia related to cannabis consumption have been reported in young men between the ages of 20 and 30.^{9,10} Myocardial ischemia can occur as a result of cannabis use among patients at risk due to tachycardia and the increase in oxygen consumption it causes.^{3,8}

The cardiovascular risk among users increases in the long run because of the carbon monoxide (CO) absorbed with the cannabis smoke. In fact, marijuana smoke contains approximately the same quantity of CO as tobacco smoke, but because of deep and slow inhalation, the concentration of carboxyhemoglobin (COHb) is 5-times higher than with tobacco smoke.¹¹ A high level of COHb is considered a major factor in the atherosclerosis associated with cigarette smoke.

Effects on the respiratory system

Cannabis smoke contains the same chemical compounds as tobacco smoke except for nicotine in tobacco and cannabinoids in cannabis.⁶ Some compounds have been identified as bronchial irritants, mutagens, and carcinogens. In cannabis, the concentration of benzantracene and benzapyrene, both of which are carcinogens, is twice that in tobacco smoke. In addition, these compounds are insoluble particles that remain deposited in the lungs. Contrary to the belief of chronic marijuana users, cannabis is more toxic than tobacco smoke.

Wu et al. studied 15 patients who chronically consumed cannabis and tobacco for over 5 years.¹¹ The cannabis users inhaled a larger volume than did the tobacco users and held in the smoke 4 times as long. Thus, after smoking a single joint, three times as many particles entered the lungs of marijuana smokers. Moreover, the concentration of COHb was 5 times as high. The authors of this interesting study concluded that cannabis smoke puts a much heavier burden on the lungs than does smoke from tobacco.

The same group of researchers also found that smoking 3-4 marijuana joints a day is equivalent to 20 tobacco cigarettes a day in terms of the incidence of bronchitis and damage to the bronchial epithelium.^{12,13}

According to a study that followed 1,037 persons born in 1972-73,¹⁴ 10% were addicted to cannabis at age 21 according to DSM-III-R criteria and 28.1% smoked tobacco. After control for tobacco use, the following respiratory symptoms were clearly associated with the cannabis addiction: wheezing without URTI (upper respiratory tract infection), exertional dyspnea, nocturnal awakening with tightness in the

TABLE 2: Increase in the incidence of different respiratory symptoms in regular cannabis users who do not smoke tobacco compared with a control group¹⁴

Symptoms	Compared with a group including occasional cannabis users and nonsmokers	Compared with group of nonsmokers only
Wheezing without URTI	61%	89%
Exertional dyspnea	65%	76%
Nocturnal awakening with tightness in the chest	72%	86%
Early morning cough	144%	348%
	P<0.01-0.05	P<0.01-0.05

URTI = upper respiratory tract infection

chest and early morning cough (Table 2). In addition, an FEV₁/FVC (forced expiratory volume in 1 second/forced vital capacity) of <80% has been documented in 36% of cannabis-dependent subjects, but in only 20% of nonsmokers (p<0.04), regardless of asthma. Even after using cannabis for a limited time with no respiratory symptoms and normal respiratory function test results, histopathological changes have been found in bronchial biopsies.^{1,14} These microscopic changes are consistent with respiratory tract inflammation. Moreover, these findings resemble the results of smoking 20 to 30 tobacco cigarettes a day. A precancerous state (squamous metaplasia) of the tracheobronchial epithelium has also been found in the presence of cannabis smoke.¹⁵ Finally, there have been many reported cases of oropharyngeal cancer (oropharynx and tongue, nasal epithelium and sinus, larynx) in young patients <40-years-old, who only used cannabis.²

Effects on the reproductive system

Cannabis is anti-androgenic and cannabinoids (including THC) bind to androgen receptors. In men, smoking marijuana is associated with abnormal sperm morphology, a lower sperm count, and reduced sperm mobility. However, given the lack of properly controlled studies that take other substances such as tobacco and alcohol use into account, the real impact on male fertility is not yet known.¹ In women, a suppression of ovulation and lower prolactin concentration is found with acute consumption. On the other hand, with chronic use the prolactin concentration rises, causing galactorrhea in women and gynecomastia in men.

Although cannabis crosses the placenta, there is no clear evidence of teratogenicity. A few cases of congenital abnormalities (hydrocephalus, cleft palate, ectopic pancreas, corneal

opacification) as well as male dysdifferentiation (related to lower fetal androgen production) have been reported. Some cognitive disorders in newborns have also been found, but the clinical significance of these effects is not clear, at present. A 10-times greater risk of nonlymphoblastic leukemia has been documented in children born to mothers who used cannabis during or immediately before pregnancy.¹⁶ A high risk of astrocytoma and rhabdomyosarcoma has also been reported in children whose mothers used cannabis during pregnancy.²

An association between smoking cannabis during pregnancy and premature labour, as well as intrauterine growth problems, has also been observed.¹ The effect on low birthweight may be due to the fetal hypoxia caused by an increase in COHb.¹ A meta-analysis of 10 studies on the use of cannabis concluded that the clinical evidence in support of the harmful effect of cannabis on birthweight is inadequate.¹⁷ Following this meta-analysis, Fergusson et al prospectively studied the prevalence of cannabis use among 12,000 British women in their 18th to 20th week of pregnancy.¹⁸ The women received a self-evaluation questionnaire before and during pregnancy. The study found that 5% of the patients smoked cannabis before and/or during pregnancy. They were younger, better educated, had lower parity, and also more often consumed alcohol, cigarettes, coffee, tea and hard drugs. No association was found between cannabis use during pregnancy and perinatal death or the need for special care to the newborn. However, the children born to mothers who used cannabis at least once a week before and during pregnancy had a lower birthweight (216 g) and length (1 cm), as well as smaller heads. Only the difference in weight remained statistically significant after adjustment for confounding factors. The study's main weakness was the use of a self-evaluation questionnaire that may have underestimated the real prevalence of cannabis consumption in pregnant women.

Effects on cellular, genetic, and immune functions

At the cellular level, chronic cannabis consumption reduces cell proliferation as well as protein synthesis. Mitosis is also abnormal and DNA synthesis is disrupted. Depending on dose, cellular immunity, macrophages, and cytotoxic T-cells are affected. At the same time, humoral immunity and cytokine (TNF, IL-1, IL-2) release are reduced.⁶

ANESTHETIC CONSIDERATIONS

Given the widespread prevalence of cannabis use in the general population, it can be expected

that many patients requiring anesthesia are occasional or regular users. It should be noted, however, that the current literature on the interaction between anesthesia and the use of cannabis is still poorly documented.

Cannabis can potentiate the hypnotic and sedative effects of substances that depress the CNS such as alcohol, barbiturates, opiates, benzodiazepines, and phenothiazines. As mentioned, cannabis interferes significantly with respiratory function. Problems with ventilation and/or oxygenation under general anesthesia are to be expected and, if possible, local anesthesia is preferable. Smoking cannabis can cause oropharyngitis and uvular edema, leading to airway obstruction under general anesthesia. A few cases of this phenomenon have been reported in the literature and are disturbing. Their authors recommend delaying any elective surgery for patients who have recently smoked cannabis.

Mallat et al describe the case of a 17-year-old male in good health who was to undergo tympanomastoidectomy.¹⁹ He had smoked marijuana once a week for 8 months. The day of the operation, the boy seemed unusually calm when the intravenous catheter was inserted, and the use of cannabis that same morning was suspected. But the patient was not specifically asked about this use because his mother was there. The pre-op examination of the airway was normal except for redness and a larger uvula and tonsils. At intubation (considered easy at the time), the anesthetist noted large red tonsils, a red pharynx, as well as a 3-cm long uvula. Despite these observations, maintenance of the anesthesia and extubation took place without incident. However, oxygen saturation plummeted post-operatively. Moreover, the patient complained of an uncomfortable sensation impossible to alleviate by coughing. Examination of the airway revealed a reddish, edematous uvula so long that the tip could not be seen. An otolaryngologist using a flexible fibre-optic laryngoscope found the tip of the uvula resting directly on the glottal opening, although vocal cord movement was entirely normal. The size of the uvula was estimated at 10-12 cm. The consulting physician confirmed the diagnosis of acute uvulitis and suggested that the excessive temperature of marijuana smoke was the cause. Treatment with intravenous dexamethasone (10 mg) was started, as well as admission for observation and oxygen supplementation. The patient was discharged the next day with a substantial reduction in the intensity of symptoms and a smaller uvula (4-5 cm). When leaving, the boy finally admitted

to having smoked marijuana 4-6 hours before the operation.

Other cases of isolated uvulitis have also been reported, especially in the emergency room where cannabis users have presented with upper airway pain, fever, hypersalivation, dyspnea, as well as respiratory distress.^{20,21} These cases respond to intravenous dexamethasone at a dose of 12 mg every 8 hours or 6 mg every 6 hours. The association between cannabis and laryngospasm under general anesthesia was described by White in 2002.²² The case involved a patient who underwent surgery for a broken jaw. Heavily intoxicated with alcohol at the time of the injury, the patient did not seek medical attention immediately. He only turned up the next day after taking paracetamol and smoking a huge marijuana joint to reduce the pain. After the surgery, laryngoscopy was performed before extubation under deep general anesthesia and confirmed the absence of any debris. In the 5-10 seconds after extubation, the patient developed heavy stridor leading to complete obstruction of the airway. This laryngospasm failed to respond to continuous positive pressure ventilation with pure oxygen, so the patient had to be reintubated. Direct laryngoscopy done with the reintubation again confirmed the complete absence of debris.

Another study²³ describes brief convulsions during induction and emergence in a 34-year-old patient anesthetized for a tooth extraction. According to the pre-operative questionnaire, the patient smoked only cigarettes. After anesthesia in which fentanyl, succinylcholine, propofol, sevoflurane, and isoflurane were administered, the patient admitted having smoked cannabis the day before surgery.

When administering anesthesia to cannabis users, one can expect dose-dependent cardiovascular changes. With low or moderate doses, there is an increase in sympathetic activity accompanied by a drop in parasympathetic activity, leading to tachycardia and increased cardiac output. With high doses, sympathetic activity is inhibited and parasympathetic activity increases, followed by bradycardia, as well as hypotension.⁸ Hypotension secondary to the use of cannabis responds well to fluids. On ECG, reversible changes in P and T waves, as well as ST segment changes, have been described. Yet, it is not clear if these modifications in the ECG are due to the cannabis itself or rather to reflex tachycardia. Despite the presence of ectopic supraventricular and ventricular beats, no fatal arrhythmias have been documented.

In cases of acute intoxication, avoiding any medication likely to increase the heart rate

such as ketamine, pancuronium, atropine, or epinephrine is recommended.²⁴ After acute consumption, more anesthetic is required due to greater catecholamine release, yet, less is required in cases of chronic use because of catecholamine depletion. Moreover, one should expect the possibility of psychiatric side-effects or withdrawal symptoms in patients during both induction and emergence. It is therefore essential to question patients about cannabis use and maintain a high index of suspicion.

CONCLUSION

Cannabis is increasingly used for recreational purposes despite the numerous harmful effects associated with both acute and chronic use. These effects extend to various systems, primarily the central nervous, respiratory, and cardiovascular systems. Because of the numerous effects on these systems, anesthesia can be hazardous for cannabis users. This makes it important for the anesthetist to be well informed about the drug. A high index of suspicion and systematic pre-operative questioning on cannabis use are highly recommended and it is prudent to delay any elective surgery in cases of acute cannabis use.

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