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KRAVETS O., KLYGUNENKO O., YEKHALOV V., KOVRYHA O.

FEATURES OF ANESTHESIA IN PATIENTS WITH SPECIAL NEEDS. PART 1

Dnipro State Medical University. Dnipro, Ukraine

Everything is poison, everything is medicine;
only the dose makes the difference.
Paracelsus, 16th century

Abstract. The non-medical use of cannabis can negatively impact the morpho-functional state of almost all organs and systems of the human body. During acute intoxication and established drug dependence, it can unevenly distort the effects of drugs for local and general anesthesia, leading to life-threatening complications. Part 1 of this review presents the results of acute and chronic cannabis use on homeostasis. Cerebral vasospasm and atherosclerosis are identified as major etiological factors for cannabis-related cerebrovascular diseases. Young to middle-aged individuals (30–50 years), male gender (male-to-female ratio 3.7:1), and chronic use (86%) are significant independent predictors of neurovascular toxicity, cerebral hypoperfusion, and stroke. Cannabis smoking causes damage to the respiratory mucosa similar to tobacco. Regular cannabis smokers can expect irritation of the upper respiratory tract, swelling, obstruction, chronic cough, bronchitis, lung emphysema, and bronchospasm. Some reports advocate the use of dexamethasone during surgery to alleviate these symptoms. The detrimental effects of chronic cannabis use on liver and kidney functions are somewhat overstated. Disturbances in immune system, thermoregulation, blood clotting, and carbohydrate metabolism in the absence of anesthetic precautions can significantly complicate the patient's condition in the perioperative period. Cannabinoids can interact with drugs from various groups. Awareness of the pathophysiological and biochemical consequences of cannabis use allows anesthesiologists to anticipate and effectively manage perioperative complications in this patient category.

Keywords: cannabis, cannabinoids, cardiovascular system, respiratory system, coagulopathy, hypoglycemia.

INTRODUCTION

Cannabis is the most widely prohibited drug globally [1], holding a significant lead in prevalence in Ukraine. On December 21, 2023, the Verkhovna Rada of Ukraine adopted bill No. 7457, «On the regulation of the circulation of plants of the genus Cannabis for medical, industrial, scientific, and scientific-technical purposes». However, the recreational use of marijuana remains prohibited. The FDA (Food and Drug Administration, USA) classification of marijuana as a prohibited substance has led to research limitations [2]. Hence, there is a critical need to consolidate existing literature on cannabis to provide healthcare professionals with tools during the perioperative period to address issues arising in the treatment of patients using cannabis [3].

EVIDENCE ACQUISITION

Articles were included for investigation if they (1) were published in English, Ukrainian, German, or French; (2) reported health disorders associated with cannabis use; (3) reported the interaction of cannabis with local and general anesthesia drugs; (4) informed about the prevalence of cannabis withdrawal syndrome and its treatment methods; (5) utilized an observational study design (cohort or cross-sectional). A retrospective information search was conducted

Для кореспонденції: OLHA KRAVETS, PhD, Doctor of Medical Sciences, Professor, Head of Department of Anesthesiology, Intensive Care and Emergency Medicine, Faculty of Postgraduate Education, Dnipro State Medical University, Dnipro, Ukraine; e-mail: 602@dmu.edu.ua; https://orcid.org/0000-0003-1340-3290

from 2002 to 2023, inclusive, using a spatial-vector model of the descriptor system, based on classifiers, complemented by manual searches of reference lists of included articles. 74.5 % of the literature sources used were from the last 5 years, 94.2 % from the last 10 years, and 5.8 % from earlier publications.

SYNTHESIS OF EVIDENCE

Hemp contains about 60 cannabinoids, categorized into three types: phytocannabinoids (nabiximols), endocannabinoids (anandamide and 2-arachidonoylglycerol), and synthetic cannabinoids (dronabinol and nabilone). Major cannabinoids include Δ9-tetrahydrocannabinol (Δ9-THC) – the primary psychoactive component, cannabidiol (CBD), and cannabinol, along with other cannabinoids and non-cannabinoid phytochemicals. CBD lacks psychoactive properties, while cannabinol is a moderately psychoactive compound. Cannabinoids activate two cannabinoid receptors, CB1 and CB2. CB1 receptors are predominantly expressed in the brain, with high concentrations in the hippocampus, frontal cortex, neocortex, olfactory areas, basal ganglia, cerebellum, and spinal cord. CB2 receptors are more peripherally located, mainly in immune system tissues (spleen macrophages) and peripheral nerve endings [1,4-8]. The presence of endogenous ligands (endocannabinoids, primarily anandamide and 2-arachidonoylglycerol) suggests that cannabinoids play a crucial role in mediating various neurophysiological processes, including nociception [8,9]. Synthetic cannabinoids are full agonists of CB1, and their toxic effects are undoubtedly stronger than phytocannabinoids [10]. In the spinal cord, CB1 receptors are localized in several areas involved in nociceptive processing, including the superficial dorsal horn, dorso-lateral funiculus, and X plate. Activation of cannabinoid receptors inhibits adenylate cyclase activity, leading to a decrease in intracellular cyclic adenosine monophosphate (cAMP) levels, activating voltage-gated potassium channels and suppressing calcium channels. This inhibits the release of neurotransmitters, forming a potential anatomical basis for the analgesic effects of cannabinoid agonists [3]. CB1 receptor activation mediates the psychoactive properties of cannabis, affecting mood, consciousness, memory processing, and motor control. Activation of CB1 influences the release of neurotransmitters, including acetylcholine, L-glutamate, gamma-aminobutyric acid (GABA), norepinephrine, dopamine, and serotonin in the prefrontal cortex. The THC-induced dopamine release in the endocannabinoid system is considered a potential brain «reward» mechanism [3, 11].

CBD can synergistically work with Δ9-THC, contributing to its analgesic effect while simultaneously reducing psychoactive and cognitive side effects, such as sedation and memory impairment. CBD has low affinity for both CB1 and CB2 receptors. It acts as a negative allosteric modulator of the CB1 receptor and a weak inverse agonist of the CB2 receptor. It also interacts with other non-cannabinoid targets, including serotonin 1A receptors, vanilloid receptor 1 (TRPV1), and adenosine A2A receptors, all of which regulate pain perception. The inverse agonist activity at the CB2 receptor explains the anti-inflammatory action of CBD [3].

Δ9-THC has high lipophilicity and distributes in tissues with high perfusion, metabolizing primarily in the liver through the P450 system. It also binds to proteins (from 95 to 99%), mainly with lipoproteins [11]. When smoked, peak plasma concentration is reached in about 8 minutes, with psychoactive effects developing within a few minutes [4]. Orally ingested tetrahydrocannabinol leads to an unpredictable onset of psychoactive effects, ranging from 1 to 3 hours. Δ9-THC is almost entirely metabolized in the liver through microsomal hydroxylation and P450 oxidation. The half-life of Δ9-THC in tissues is approximately 7 days, and complete elimination can take up to 30 days [1,4,12]. CBD has a half-life of 27-35 hours after smoking or inhalation and 2-5 days after oral ingestion. A significant portion of CBD is excreted unchanged in urine and feces [3]. Marijuana, derived from the hemp plant, is one of the oldest documented remedies in history, possessing antiemetic, muscle relaxant, anticonvulsant, analgesic, appetite-stimulating, sedative, anxiolytic properties. It is used to reduce intraocular pressure, treat depression, epilepsy, Tourette's syndrome, AIDS-related anorexia, and certain dermatological and viral conditions [1,13-15].

As cannabis smoking is the most widespread substance abuse, anesthesiologists regularly encounter its users [1,9]. Cannabinoids can potentially have a dangerous interaction with medications used for anesthesia, posing a risk to life. Anesthesiologists need to have up-to-date knowledge about psychotropic drugs, their side effects, their interaction with widely used anesthetics, and the basics of treating complications during surgeries and anesthesia [16]. In addition to a thorough medical history, it is crucial to ask specific questions about pill consumption. Triggers for suspicion include peculiar behaviour in the clinic and symptoms such as diarrhoea or vomiting. Ultimately, the main consequence of this scenario is adequate sedation, which should be administered by the anesthesiologist regardless of the ASA assessment, especially in cases with higher cardiovascular and respiratory risks [17,18]. Informed and accessible communication with patients contributes to improved understanding and mutual trust, enhancing overall outcomes [3]. An anesthesiologist should gather information on the frequency of drug use and the time since the last consumption. There is a possibility that

social consequences may lead patients to conceal this information [12,19].

With the increasing prevalence of cannabis use among adults, it is crucial for anesthesia specialists to recognize its potential impact on the central nervous, cardiovascular, and gastrointestinal systems when providing perioperative care to patients with specific needs [1,9,11]. When assisting individuals dependent on cannabis, physicians should be mindful of the potential for aggressive behaviour (panic attacks or confrontational attitudes) [19]. Combining cannabis with additional anticholinergic medications often leads to paradoxical excitation [20]. When pills are consumed before the age of 18, the risk of developing dependence increases sixfold [3].

The action of non-medical cannabis on the body unfolds in two phases: the first phase involves motor and speech excitement, increased pulse rate, ringing in the ears, hallucinations accompanied by euphoria and silliness, sometimes accompanied by crying, fear, suspiciousness (paranoid behaviour), and aggression; the second phase includes a drowsy state, sleep with vivid dreams, clouding of consciousness, slowed pulse, hypotension, hypoglycaemia (pathological bulimia), hypothermia, dryness of the mucous membranes of the mouth and throat, nausea, vomiting, and diarrhoea [19]. The psychological manifestations are diverse, often characterized by relaxation, dizziness, laughter, and increased appetite. Toxicity leads to decreased coordination, muscle strength, and smoothness of hand movements, lethargy, sedative effects, orthostatic hypotension, inability to concentrate, unclear speech, and slowed reactions. Dependent individuals may be distrustful, dysphoric, experiencing fear, and even transient psychotic episodes [4]. Well-documented effects of cannabis on humans include impaired short-term memory, analgesic, and antiemetic properties. The impact on memory and nausea is mainly mediated through CB1 receptors, while antinociceptive effects involve other receptors [8]. Cerebral vasospasm and atherosclerosis have been identified as major etiological factors for cannabis-related cerebrovascular disorders, with young and middle-aged individuals (30–50 years old), males (male-to-female ratio 3.7:1), and chronic use (86%) being recognized as significant independent predictors of neurovascular toxicity, cerebral hypoperfusion, and stroke [10, 21]. Cannabinoids exhibit both anticonvulsant and proconvulsant effects. They may induce various psychological consequences (anxiety, tachycardia) [22, 23]. The impact of exogenous cannabinoids on the heart varies with dosage [24]. Low doses lead to sympathetic stimulation with tachycardia and mild hypertension [25]. Clinical studies show that the initial tachycardia observed in these patients may be mediated by the β-adrenergic effect of adrenaline (adrenal gland stimulation) together with suppression of the parasympathetic nervous system. This principle is supported by research indicating that prior treatment with propranolol effectively blocked Δ9-THC-mediated heart rate increase [10]. Similarly, an increase in heart rate, left ventricular contractility, and cardiac output has been reported in regular users after daily marijuana consumption (Δ9-THC ≥ 10 mg). These positive chronotropic and inotropic effects are enhanced when CB-R agonists (synthetic cannabinoids) are used and suppressed by the administration of the CB1 antagonist rimonabant. As cannabinoid dosage increases, a strong parasympathetic response accounts for orthostatic hypotension and bradycardia in the supine and postural positions [10]. Prolonged sympathetic stimulation may be insufficient to compensate for these postural hemodynamic changes, resulting in peripheral vasodilation and baroreflex dysregulation, leading to orthostatic hypotension. Anandamide (endogenous cannabinoid receptor ligand) is responsible for vasodilation in other vascular beds (mesenteric) mediated by the activation of expressed sensory nerve terminals of vanilloid receptors (TRPV1-R) with subsequent release of calcitonin gene-related peptide (CGRP). After controlled administration of high doses of Δ9-THC in regular users, a decrease in heart rate and tolerance to orthostatic hypotension have been reported [10]. For lower doses, a biphasic response is observed. The first phase is characterized by tachycardia (a 20-100 % increase in heart rate) lasting up to several hours, along with an increase in cardiac work. Cannabis use has been associated with an increase in systolic, but not diastolic blood pressure [21, 24]. The effects of cannabis on the cardiovascular system involve a complex interplay of sympathetic and parasympathetic nervous system activity. After the initial phase of increased sympathetic activity, characterized by tachycardia and elevated cardiac output, a subsequent phase follows with bradycardia and hypotension at higher cannabis doses. It's noteworthy that reflex bradycardia during the Valsalva maneuver expiration phase is suppressed during this phase [24]. In patients who consumed small or moderate cannabis doses, there is an elevation in sympathetic activity and a reduction in parasympathetic activity, resulting in tachycardia with increased cardiac output [4, 12, 26-28]. At low or moderate doses, marijuana increases sympathetic activity, decreases parasympathetic activity, and induces an increase in heart rate, cardiac output, and blood pressure. However, with higher doses, the parasympathetic system takes precedence, leading to bradycardia and hypotension. Animal studies suggest that the suppression of the sympathetic nerve occurs due to the influence of a biologically active component of cannabis on CB1 receptors [1, 23, 25]. Long-term intense cannabis use has been independently associated with an increased

frequency of cardiovascular events in men aged 40–60 [12, 21 ,29 ,30]. The interaction with both receptors inhibits adenylate cyclase and stimulates the opening of potassium channels [4]. Non-medical cannabis use leads to myocardial depression, exacerbating existing tachycardia, and causing supraventricular or ventricular ectopic activity [1, 27]. Acute cannabis use may result in orthostatic hypotension, while chronic use may be associated with a moderate increase in systolic blood pressure [31]. Major cardiovascular changes include an increase in heart rate (HR) and a decrease in vascular resistance (these changes last about 2-3 hours). However, repeated intake may lead to a decrease in HR and blood pressure (BP) [4]. Chronic cannabis use can lead to bradycardia, as Δ9-THC inhibits calcium-dependent ATPase in the cardiac muscle [12]. Since cannabis consumption increases the myocardium's oxygen demand and decreases its supply, patients with pre-existing ischemic disease may experience increased angina symptoms, especially during marijuana smoking. Cannabis use is recognized as an independent predictor of heart failure [21]. There is a risk of a fivefold increase in the likelihood of myocardial infarction within the first hour after smoking due to increased cardiac output, myocardial oxygen demand, catecholamine levels, carboxyhaemoglobin levels, and postural hypotension [1, 9]. The heightened risk of myocardial infarction associated with cannabis use is attributed to the combination of tachycardia and peripheral vasodilation, leading to compensatory orthostatic hypotension, and increased myocardial oxygen demand [32]. The endocannabinoid system partially acts as a neuromodulator of the cardiovascular system [24].

The electrocardiogram (ECG) visualizes increased ectopic activity of supraventricular and ventricular tachyarrhythmias, ST segment elevation, as well as changes in the T wave [4, 27, 28]. Supraventricular or ventricular ectopic activity has been reported during cannabis use, along with abnormalities in the ST segment and T wave. The ECG of cannabis-dependent individuals often shows premature ventricular contractions, reversible changes in the ST and T wave, or a decrease in P-wave voltage [12]. Δ9-THC potentiates the action of antiarrhythmic agents (amiodarone, dronedarone, flecainide, propafenone), β-adrenergic blockers (carvedilol, metoprolol), and dihydropyridine calcium channel blockers (amlodipine, felodipine) [21]. The additive effect of marijuana and potent inhaled anesthetics can lead to significant myocardial depression during general anesthesia [12, 27, 33].

The use of atropine and adrenaline in such patients may induce pronounced tachycardia [27, 28], but there are reports suggesting that adrenaline does not have a synergistic effect with marijuana. When it comes to cardiovascular effects, further research is needed to explore the potential interaction between marijuana and perioperative medications [23]. Patients with a history of acute marijuana use should avoid all medications affecting heart rate, but bradycardia and hypotension (resulting from high doses of marijuana) raise doubts about the necessary amount of atropine and vasopressors [23].

It's known that cannabinoids reduce salivation (xerostomia/dry mouth), and cannabinoid receptors CB1 and CB2 have been identified in the submandibular glands [34]. In patients intoxicated with cannabis during procedures where propofol was administered as a general anesthetic, there is often a paradoxical increase in saliva secretion [34, 35]. Chronic cannabis use can lead to stomatitis, glossitis [19], tongue swelling, and obstruction of the airways [10, 12, 23].

Potential hyperreactivity of the airways during acute cannabis use positively correlates with respiratory symptoms related to chronic bronchitis, such as coughing, sputum production, and wheezing (odds ratio = 2.98) [24, 36]. Cases of oropharyngitis, acute upper airway edema, and obstruction have been described in patients after general anesthesia [4, 23]. Cannabis smoking causes similar damage to the respiratory mucosa as tobacco. Potential irritation of the upper airways, swelling, obstruction, chronic cough, bronchitis, lung emphysema, and bronchospasm are all expected in regular cannabis smokers. In some reports, the use of dexamethasone during surgery is even advocated to reduce these symptoms [1, 4, 12, 23, 25, 27]. This recommendation seems reasonable, given the life-threatening bronchospasm leading to asphyxia, brain damage, or death in case of unsuccessful tracheal intubation in patients with airway obstruction. Steroids should help with inhalation-induced uvulitis, as they increase endotracheal permeability, reduce mucosal swelling, and stabilize lysosomal membranes, thereby decreasing the inflammatory response. Prospective randomized double-blind studies have shown that adding methylprednisolone to salbutamol in patients with partially reversible airway obstruction helped reduce reflex bronchoconstriction caused by tracheal intubation. Some authors recommend the prophylactic use of dexamethasone [10, 12, 23]. The increased risk of airway hyperactivity during inhalation or ingestion leads to decreased airway resistance and increased airway permeability [4, 11]. Bronchial dilation is short-lived, but obstruction can develop, especially in those who use cannabinoids for an extended period [2, 25]. Cannabis smokers have 144 % more sputum production than non-smokers, significantly increasing the likelihood of pneumonia [12, 23, 37]. CB2 receptors are mainly found in peripheral tissues with immune functions (spleen macrophages) and peripheral nerve endings [1, 4, 8]. The immunosuppressive effect of Δ9-THC causes harmful structural changes in the large airways of marijuana smokers [37]. Signs of stridor

caused by upper airway edema should be monitored [25]. Anesthesiologists should be prepared to manage airway hyperactivity during surgery if patients do not have secure airways due to potential irritation from preoperative cannabis use [11]. Preoperative cannabis smoking is associated with postoperative airway obstruction characterized by swelling of the oropharynx and tongue. For this reason, it is recommended to postpone the surgery if the patient smoked marijuana shortly before the planned operation. Nevertheless, some practicing physicians may consider prescribing steroids to reduce the risk of tongue swelling or uvulitis [10]. The use of cannabinoids significantly increases the risk of developing laryngospasm after extubation [22, 23]. There are reports of pneumothorax from frequent Valsalva manoeuvres. Patients with chronic cough are at risk of wound dehiscence after surgery [25].

The physiological basis of dry eye symptoms involves the activation of CB1 neuronal receptors in the lacrimal gland, leading to reduced tear production, conjunctival injection, and decreased intraocular pressure [4,14]. This may increase the risk of perioperative corneal damage [2]. Risk factors include older age, female gender, antidepressant use, and certain autoimmune diseases [38].

Several cases of potential liver damage during simultaneous use of cannabis and acetaminophen have been described [2]. In high doses, cannabis can elevate liver enzyme levels [31]. Large epidemiological studies have repeatedly linked cannabis use to liver pathologies, but all results were retrospective and uncontrolled, especially regarding the presence of other potential causes of chronic liver injury [14].

Synthetic cannabinoids are potent CB1 agonists initially developed as research compounds but later emerged on the market as popular and potentially dangerous recreational drugs under names like «spice» or «K2.» Specifically, the synthetic cannabinoid XLR-11 was identified as a nephrotoxic compound associated with its impact on the function of mitochondria in proximal tubular cells. Kidney biopsy often demonstrates acute tubular necrosis with occasional cases of acute interstitial nephritis, while CB1 inhibition prevents kidney fibrosis development and reduces proteinuria [31, 39].

A recent retrospective cohort study revealed an association between chronic cannabinoid use and a 20 % increased frequency of anesthesia-related postoperative nausea and vomiting [3, 12, 29, 30, 40]. Mechanisms may involve reduced CB1 receptor regulation and inhibition of gastric emptying [21, 24]. In the case of recent drug use, considering such a patient as having a full stomach is advisable [4]. Prolonged (6-7 years) daily and weekly cannabis use is associated with hyperemesis, characterized by periodic episodes of severe nausea, vomiting, abdominal pain, and diarrhoea [2, 14, 31, 32, 41].

The influence of cannabis or cannabinoids is linked to a decrease in body temperature. Perioperative hypothermia is defined as a temperature < 36 °C with a shivering threshold typically set at 35.5 °C in patients without anesthesia. Hypothermia and shivering are often observed in cannabis users during the postoperative period [10]. Increased heart rate, hypoxemia, restricted oxygen delivery and consumption, myocardial ischemia, and acidosis are well-known physiological effects of shivering thermogenesis. Postoperative hypothermia and shivering are believed to be mediated by CB1 receptor activation, and it's not suspected that they result from withdrawal symptoms. Cannabinoid-induced hypothermia is mediated by CB1 receptor activation and can be reversed using the antagonist Acomplia® (SR141716A, Zimulti, rimonabant) 20 mg orally [10, 11].

The growing body of evidence demonstrates coagulation abnormalities associated with cannabinoid use, indicating the involvement of the endocannabinoid system (ECS) in modulating the blood clotting system. Partial primary and complete secondary inhibition of human platelet aggregation induced by adrenaline, as well as anticoagulant effects observed in plant-derived cannabinoids, have been observed. Diffuse alveolar hemorrhage and hemoptysis have also been associated with cannabis smoking [23, 42, 43]. Endocannabinoid receptor agonists reduce platelet activation and aggregation [44]. This anticoagulant effect opposes hemostasis and limits surgical visualization [23]. Intravenous cannabis injection leads to a significant reduction in platelet count, corresponding to the belief in anticoagulation; marijuana components cause the release of ADP from erythrocytes, leading to platelet aggregation and increased susceptibility to myocardial infarction and arterial diseases [10, 11, 23]. Increased platelet aggregation is likely associated with the presence of CB1 and CB2 receptors on platelet membranes at high doses of Δ9-THC [10, 11]. Inflammatory reaction of the arterial wall and increased oxidative stress, platelet activation, and excessive activation of factor VII have been proposed as the main mechanisms of Δ9-THC-induced platelet aggregation. Additionally, cannabinoids may reduce the availability of nitric oxide in blood vessels, leading to endothelial dysfunction and platelet activation [10]. Immune thrombocytopenia with synthetic cannabis is rarely encountered [3]. Δ9-THC and CBD may competitively inhibit CYP2C9 and the metabolism of S-warfarin isomer, resulting in supratherapeutic international normalized ratio levels [3, 45]. Warfarin, metabolized by CYP3A4, has also demonstrated a potential interaction with cannabinoids, as evidenced by a patient report actively using cannabis during warfarin intake, leading to supratherapeutic international normalized ratio (INR) levels and significant bleeding complications [36]. Warfarin and cannabinoids are

metabolized by cytochrome P450. Δ9-THC strongly binds to lipoproteins, while cannabidiol is a potent CYP3A4 inhibitor and a weak CYP2C9 inhibitor. These two factors mainly contributed to the increase in plasma warfarin levels in this patient and thus the occurrence of warfarin-related side effects [10, 11, 42, 46]. Therefore, the anticoagulant effect of warfarin may potentially be enhanced by cannabinoids, some of which exhibit anticoagulant action by inhibiting thrombin activity [11, 42]. CBD and, possibly, Δ9-THC may increase clopidogrel levels in the blood through competitive inhibition of CYP2C19, the isoenzyme responsible for transforming clopidogrel into its active thiol metabolite. This interaction may lead to the establishment of subtherapeutic levels of the active metabolite and a potential increase in the risk of stroke [3, 46].

Δ9-THC may modulate the blood clotting cascade and contribute to hypercoagulation by disrupting platelet function through an unfavorable increase in platelet activation. Chronic hypocoagulation appears to protect against this elevated risk. Geriatric cannabis users with injuries are significantly prone to deep vein thrombosis, pulmonary embolism, stroke, myocardial infarction, and higher mortality. Early detection and appropriate thromboprophylaxis can reduce the risk of venous thrombosis and thromboembolic complications [47, 48]. There are no known interactions with heparin/fondaparinux, as these agents are processed by endothelial and renal cells and not metabolized by cytochrome P450 (CYP), uridine 5'-diphosphoglucuronosyltransferase (UGT), or P-glycoprotein [3].

Cannabinoid derivatives inhibit gluconeogenesis in experimental animals. In the second phase of cannabis action, users often experience significant hypoglycemia accompanied by pathological bulimia [19, 49, 50]. Human Langerhans islets express cannabinoid receptors CB1 and CB2; in vitro experiments have demonstrated that CB1 stimulates insulin and glucagon secretion, while CB2 agonism reduces glucose-dependent insulin secretion. Δ9-THC and CBD act as partial agonists and antagonists of CB1 and CB2, thus capable of exerting antagonistic effects on cannabinoid receptors without a clear dose-response relationship. Patients with frequent cannabis use episodes generally have lower fasting blood insulin and glucose levels [51].

CONCLUSIONS:

1. Acute and chronic cannabis use can lead to significant pathological changes in the organs and systems of the body, resulting in a substantial increase in anesthetic-surgical risk.
2. The central nervous, cardiovascular, and respiratory systems are most affected.
3. The likelihood of liver and kidney dysfunction in cannabis users remains a contentious issue.
4. Disorders of the immune system, thermoregulation, blood clotting, and carbohydrate metabolism, in the absence of anesthetic vigilance, can significantly complicate the patient's condition in the perioperative period.
5. Awareness of the pathophysiological and biochemical consequences of cannabis use will enable anesthesiologists to anticipate and effectively manage perioperative complications in this patient category.

The choice of preoperative preparation and safe anesthesia withdrawal options, as well as cannabis withdrawal syndrome, will be discussed in Part 2 of the literature review.

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КРАВЕЦЬ О.В., КЛИГУНЕНКО О.М., ЄХАЛОВ В.В., КОВРИГА О.В

ОСОБЛИВОСТІ АНЕСТЕЗІЇ В ПАЦІЄНТІВ ЗІ СВОЄРІДНИМИ ПОТРЕБАМИ. ЧАСТИНА 1

Дніпровський державний медичний університет. м. Дніпро, Україна

Абстракт. Вживання канабісу з немедичною метою здатне негативно впливати на морфо-функціональний стан практично всіх органів та систем організму людини, в період гострої інтоксикації та при сформованій наркозалежності неоднаково спотворювати дію препаратів для локальної та загальної анестезії із розвитком життєво небезпечних ускладнень. В Частині 1 даного огляду наведені результати впливу гострого та хронічного вживання канабісу на гомеостаз. Церебральний вазоспазм і атеросклероз визначені як основні етіологічні фактори цереброваскулярних захворювань, пов'язаних із канабіноїдами, тоді як молодий та середній вік (30–50 років), чоловіча стать (співвідношення до жінок 3,7:1) і хронічне вживання (86 %) є основними незалежними предикторами нервово-судинної токсичності, церебральної гіпоперфузії та інсульту. Куріння канабісу завдає такої ж шкоди слизовій оболонці дихальних шляхів, як і тютюн. Потенційна іритація (подразнення) верхніх дихальних шляхів, набряк і обструкція, хронічний кашель, бронхіт, емфізема легенів і бронхоспазм – усе це можна очікувати в регулярного курця канабісу. В кількох звітах пропагується застосування дексаметазону під час операції, щоб зменшити ці симптоми. Ушкоджуюча дія хронічного вживання канабісу на функції печінки та нирок дещо переоцінена. Розлади систем імунітету, терморегуляції, згортання крові та вуглеводного обміну при відсутності анестезіологічної настороги здатні значно обтяжити стан пацієнта в періопераційному періоді. Канабіноїди здатні взаємодіяти з медичними препаратами різних груп. Обізнаність в патофізіологічних та біохімічних наслідках вживання канабісу дозволить лікарям-анестезіологам передбачати та ефективно усувати періопераційні ускладнення у даної категорії пацієнтів.

Ключові слова: канабіс, канабіноїди, серцево-судинна система, дихальна система, коагулопатія, гіпоглікемія.

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O. Kravets, O. Kligunenko – conceptualization, **V. Yekhalov –** original text writing; **O. Kovryha –** translation.