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Review Article

Cannabis-induced cardiac arrhythmias: mechanistic insights, epidemiologic patterns, and clinical implications

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ABSTRACT

As legalization and therapeutic interest expand, the global use of cannabis has significantly increased. Nonetheless, its cardiovascular impacts-particularly the risk of heart arrhythmias-are still not well acknowledged. Recent findings suggest that cannabis, especially its psychoactive element Δ^9-tetrahydrocannabinol (THC), could have a mechanistic influence in triggering both atrial and ventricular dysrhythmias. A narrative review was performed utilizing pertinent literature released from January 2000 to May 2024. Databases explored encompassed PubMed, Scopus, and Web of Science. Studies were chosen for their relevance to cannabis-related arrhythmias, encompassing experimental, observational, and clinical findings. Focus was directed towards mechanistic understanding, epidemiological patterns, and clinical results. Cannabis use has been linked to a range of arrhythmias, from harmless palpitations to severe ventricular tachyarrhythmias and sudden cardiac arrest. THC affects cardiomyocyte ion channels, extends repolarization, and modifies autonomic control, establishing a basis for arrhythmogenesis. Long-term exposure could lead to myocardial fibrosis and oxidative stress. Increased risk is observed in young adults, regular users, and those with psychiatric or pre-existing heart issues. Even with these results, awareness and screening continue to be restricted in regular clinical practice. Cannabis presents a clinically important yet overlooked danger for cardiac arrhythmias. Healthcare professionals ought to take into account recent cannabis consumption in patients who show unexplained arrhythmias. There is a critical requirement for longitudinal research and revised clinical guidelines to guide risk evaluation, patient education, and monitoring approaches.

Keywords: Cannabis, Arrhythmia, Tetrahydrocannabinol, Sudden cardiac death, Cardiac electrophysiology, Substance use, Psychiatry, Cardiology

INTRODUCTION

In last 20 years, worldwide scenario of cannabis consumption has changed in ways that even experienced public-health experts may not have foreseen. Recent figures from U. N. indicate that close to 200 million individuals-about one in every fifteen adults aged 15 to 64-consumed cannabis in last year, marking an approximately 20 percent rise since 2010.[1-3] Over fifty countries, along with an increasing number of U.S. states, currently allow its medical use and, in many areas, its recreational use.[4]

However, this liberalization has occurred alongside a less-recognized trend: laboratory tests of seized substances show that levels of Δ^9-THC have more than doubled during the same timeframe, prompting troubling inquiries regarding dose-related toxicity and the sufficiency of current regulatory protections.[5] While proponents frequently highlight the therapeutic potential and social justice reasons, the increasing strength of modern products complicates simple narratives of advantage, emphasizing an urgent necessity for detailed risk-benefit evaluations that can match the swift changes in policy. Usage trends have changed alongside evolving legislation. Previously limited mainly to teenagers and young adults, cannabis use now includes a wider age range; recent national surveys indicate that almost ten percent of adults over 50 claim to have used the substance in the last month-a discovery that would have been hard to envision a generation earlier.[6] Reflecting this increase, emergency departments noted a 35% jump in cannabis-related visits from 2016 to 2020.[7]Particularly troubling are incidents related to synthetic cannabinoids-sold under labels like “K2” and “spice”-which seem to trigger more intense and less predictable cardiovascular issues compared to natural products.[8] Collectively, these trends suggest that increasing access has not only enlarged the user base but might also be presenting new clinical challenges that current surveillance systems are just starting to identify.

Determining causality is still challenging, but the growing evidence is difficult to disregard: both epidemiological findings and experimental studies increasingly point to cannabis being linked to various cardiovascular issues.[9-12] A meta-analysis, though constrained by diverse study designs and inconsistent sampling, suggests that active users face approximately a 30% increased risk of clinically significant dysrhythmia compared to those who abstain.[13] These data alone do not demonstrate a direct toxic effect; nonetheless, they contest the frequently benign public discourse on cannabis, particularly considering increasing product strength and broader legal availability. A more detailed examination-ranging from fundamental electrophysiology to broader population surveillance-appears necessary to determine who is genuinely at risk and in which circumstances those risks become clinically significant. Initial clinical accounts-limited by small participant numbers and typical reporting biases-indicate that cannabis use may trigger a surprisingly wide range of arrhythmias. In these reports, atrial fibrillation (AF), paroxysmal supraventricular tachycardia (SVT), and even severe ventricular tachyarrhythmias have developed within hours of use, occasionally in individuals whose previous cardiac assessments were normal.[14,15]

While these individual observations do not prove causality, they bring up two urgent questions. Could a portion of users possess a hidden electrophysiological sensitivity that cannabis merely reveals? Secondly, do elements like dosage, formulation, or administration method serve as the critical point at which harmless experimentation transitions into clinically meaningful rhythm disruption? Elucidating these options will necessitate a thoughtful combination of mechanistic investigations and well-structured prospective cohorts-efforts that have only just started.

From a chemical perspective, cannabis plant showcases a complex mosaic rather than a singular, homogeneous substance. Scientists have documented more than a hundred unique phytocannabinoids; however, academic discussion remains mostly focused on Δ^9-THC and cannabidiol (CBD).[16,17] Both engage with endocannabinoid system-a widespread signaling network that regulates appetite, mood, pain perception, immune response, and inflammation processes-yet their impacts on CVS seem less distinctly defined. Activation of CB_1 and CB_2 receptors could potentially shift autonomic balance, alter vascular reactivity, and affect conductance of cardiac ion channels. Exactly how those receptor-level occurrences convert into arrhythmogenic risk is still only partially understood, creating significant space for differing interpretations and methodological concerns. At this point, any definitive causal assertion linking a specific cannabinoid to clinically significant dysrhythmias should be considered tentative, awaiting comprehensive research that connects molecular pharmacology, *in-vivo* electrophysiology, and extensive population data.

By acting as a partial agonist at CB_1 and CB_2 receptors-widespread G-protein-coupled receptors present not just in the central nervous system but also in cardiac cells, blood-vessel lining, and immune cells-Δ^9-(THC elicits numerous physiological effects.[18] Activation of CB_1 seems to shift autonomic balance in favor of sympathetic dominance while diminishing parasympathetic activity, potentially leading to increased heart rate, sudden fluctuations in blood pressure, and creating an electrophysiological setting susceptible to rhythm irregularities.[19-21] While these connections are primarily based on preclinical and brief human studies, they illustrate a biologically feasible pathway through which acute THC exposure could disrupt cardiac conduction, particularly in those with existing autonomic instability.

Laboratory findings have begun to outline a credible electrophysiological pathway connecting cannabis to arrhythmia, though much still remains uncertain. In isolated-cell and animal models, Δ^9-(THC reduces voltage-gated sodium and L-type calcium currents while concurrently inhibiting the delayed-rectifier potassium channels IKrand IKs.[22-24] The overall impact-extended repolarization coupled with an increased likelihood of early afterdepolarizations-fosters conditions for both triggered activity and re-entry phenomena. Prolonged exposure seems to introduce an additional level of susceptibility: ongoing oxidative stress, mild endothelial damage, and uneven interstitial fibrosis have been noted, alterations that might reduce the threshold for malignant tachyarrhythmias *in vivo*.[25,26]

Synthetic cannabinoids add more complexity to the situation. As complete CB_1 agonists with significantly increased receptor affinity, products sold as “K2” or “Spice” can intensify autonomic changes and ion-channel disruptions, and have been consistently associated with acute coronary syndromes, ventricular fibrillation, and even sudden cardiac death-frequently in healthy young adults.[27,28] The mechanistic basis for these effects is becoming more evident; however, translating lab findings into clinical practice is more challenging due to varying formulations, dosages, and usage patterns.

In light of this context, the current review gathers existing evidence on cannabis-related arrhythmogenesis, from cellular-level mechanisms to broad-scoped epidemiological indicators, and highlights the key gaps that healthcare professionals and researchers need to tackle so that policy and practice can align with swiftly changing consumption patterns.

MECHANISTIC INSIGHTS

Emerging evidence suggests that cannabis-linked rhythm disturbances arise from an interplay of acute cardiovascular strain, rapid shifts in autonomic balance, and direct modulation of myocardial ion channels-processes that may commence within mere minutes of THC exposure. Yet precisely how these elements converge, and in whom they cross the threshold from transient perturbation to clinically significant arrhythmia, remains only partially understood.

Acute vascular effects

Inhaled Δ^9-THC seems to elicit a quick, biphasic response in blood pressure-a sharp increase in systolic pressure, a temporary decrease, and then a subsequent rise-that occurs within minutes of inhalation. Simultaneously, experimental and imaging research indicates a temporary narrowing of coronary vessels paired with endothelial oxidative stress, alterations that could diminish myocardial perfusion and increase platelet reactivity.[10] Short instances of subendocardial hypoxia might, theoretically, establish specific areas of ischemia that act as origins for premature ventricular complexes, especially when sympathetic activity is at its highest. Although these vascular disturbances have mainly been observed in acute-challenge scenarios, their clinical importance likely varies based on individual factors such as initial cardiovascular capacity, simultaneous stimulants, and the overall level of autonomic activation.

Autonomic imbalance

Accumulating experimental data suggests that activation of CB_1 receptors by Δ^9-THC skews autonomic equilibrium in favor of sympathetic dominance while reducing vagal effects.[16] Microneurography in humans has shown this adrenergic shift in real time, while supporting animal studies indicate that the same disruption decreases baroreflex sensitivity, thereby reducing the threshold for arrhythmia onset.[18] In practical terms, the sympathetic response speeds up heart rate, reduces heart-rate variability, and quickens intracellular calcium cycling in cardiomyocytes. In these circumstances, even slight ectopic activity can develop into persistent tachyarrhythmias, such as catecholaminergic polymorphic ventricular tachycardia (CPVT).[19] Even though these results primarily come from acute-exposure models, they highlight a potential mechanistic pathway through which THC may transform short-lived electrical instability into clinically relevant rhythm disturbances.

Ion-channel disruption

Recent data suggest that THC may directly influence cardiac ion channels. Cannabis consumption has been associated with prolonged QT interval, primarily as it reduces the rapid and slow delayed-rectifier potassium currents IKr and IKs.[22] Extra in vitro research using human-derived cardiomyocytes indicates that THC also reduces peak sodium influx and L-type calcium currents.[24] Reducing these currents extends action-potential propagation and leads to uneven recovery throughout the myocardium, laying the groundwork for re-entry circuits. In line with this mechanism, case reports indicate that torsade's de pointes can arise within an hour after cannabis consumption-even in patients without structural heart conditions or identified channelopathies.[29]

Individual vulnerability

For many individuals, the ion-channel perturbations induced by THC translate into nothing more than transient palpitations; for a susceptible minority, however, the same physiology may unmask latent vulnerabilities. Brugada-like ECG patterns can emerge, and a marginally prolonged QT interval can slip into a hazardous range when additional stressors-concomitant QT-prolonging drugs or electrolyte deficits such as hypokalaemia or hypomagnesaemia-are present. In such scenarios, what begins as a minor autonomic flutter may escalate into a clinically significant arrhythmia, underscoring the need for careful risk stratification in users with any pre-existing cardiac or metabolic fragility.[16,29]

Chronic changes

With continued exposure, the transient disturbances seen after a single dose begin to solidify into more durable structural and autonomic alterations. Chronic THC signalling appears to foster low-grade myocardial scarring, disrupt intercellular coupling, and blunt baroreflex sensitivity, at least in preclinical models.[18] Prolonged cannabinoid activity has also been shown to heighten mitochondrial oxidative stress, compromise ATP production, and stimulate progressive fibrosis within cardiac tissue.[30] In studies extending twelve weeks or longer, sections of ventricular myocardium were gradually replaced by collagen, generating pockets of electrical heterogeneity that lower the threshold for re-entrant circuits and other arrhythmogenic mechanisms.[31] Thus, what starts as a fleeting electrophysiological perturbation may, over time, evolve into a fixed substrate capable of sustaining malignant rhythms.

Together, these short-term triggers and long-term structural changes help explain why cannabis can cause anything from simple sinus tachycardia to severe arrhythmias like ventricular fibrillation-and why regular, heavy use may gradually raise risk of life-threatening events.

Table 1: Electrophysiological effects of cannabis and cannabinoids.

Mechanism	THC Action	Clinical Consequences	Reference
Sodium channel block	↓ Na^+ influx	Slowed conduction, increased QRS	24
L-type calcium block	↓ Ca^{2+} influx	Reduced contractility, prolonged repolarization	24
I_Kr and I_Ks inhibition	↓ K^+ efflux	QT prolongation, torsades de pointes risk	22 and 24
CB_1 receptor activation	↑ Sympathetic, ↓ Vagal tone	AF, SVT, VT	18, 19 and 21
Chronic oxidative stress	Fibrosis, mitochondrial injury	Re-entry circuits, ventricular arrhythmias	30 and 31

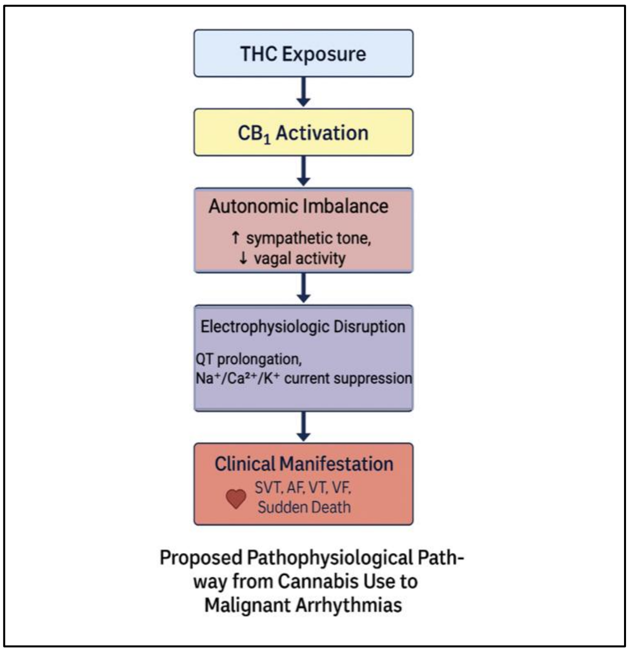

Figure 1: Pathophysiological pathway from cannabis use to malignant arrhythmias.

EPIDEMIOLOGIC PATTERNS

Observational studies derived from large inpatient registries and emergency department data increasingly associate cannabis use with a wide range of rhythm disorders.[25,26] Among the supraventricular types, AF appears most reliably. Cannabis seemingly shifts autonomic balance by increasing sympathetic activity and reducing vagal tone, circumstances that promote early atrial depolarizations. Consistent high exposure, on the other hand, might lead to atrial enlargement and persistent fibrosis-structural alterations that create a conducive environment for re-entry and, as a result, AF.[8,9] In one of the most extensive studies conducted so far, which examined over 23 million hospital admissions throughout California, those indicating cannabis use demonstrated a 35% greater occurrence of new-onset AF compared to non-users.[28] In addition to these population-level findings, case reports highlight healthy young adults who experienced sudden, symptomatic AF within hours of cannabis use, emphasizing that the risk of arrhythmia is not limited to older individuals or those with comorbidities.[8]

Instances of SVT have also been recorded shortly following cannabis use. Individuals affected frequently describe a rapid, intense heartbeat-rates surpassing 180 beats per minute-sometimes accompanied by feelings of dizziness or near-fainting. The dominant hypothesis mirrors that of AF: increased sympathetic activity combined with reduced vagal control promotes atrioventricular-nodal re-entry, a frequent basis for SVT.[14,15] Emergency department studies consistently indicate that these incidents often occur in patients without underlying structural heart conditions and-comfortingly-typically resolve with basic vagal maneuvers or a single dose of adenosine.[14,15]

Ventricular rhythm abnormalities present much greater risk than those originating from the supraventricular region. Examinations of national inpatient data indicate that users of recreational cannabis are approximately two to three times more prone to admission due to ventricular tachycardia or fibrillation-this increased risk appears to rise further when QT-prolonging medications or electrolyte deficiencies like hypokalaemia or hypomagnesaemia are also present.[19] The danger is heightened by synthetic cannabinoids (e. g., K2, Spice), which attach to cannabinoid receptors with much greater strength than natural THC. Numerous case series now document occurrences of out-of-hospital ventricular fibrillation and sudden cardiac arrest in otherwise healthy young adults shortly after using these products.[27,28] While the exact frequency is unclear, the seriousness of these occurrences calls for increased clinical attention, especially when synthetic formulations or other pro-arrhythmic elements are involved.

One of the most disturbing indicators relates to unexpected cardiac death, a situation that seems to affect younger users more significantly. Multiple forensic studies report lethal arrests in individuals who had recent cannabis exposure yet show no angiographic or histopathologic signs of coronary disease or structural cardiomyopathy-results indicating a primary electrical mechanism instead of an ischemic cause.[32] In a population-focused study conducted in Canada, the chance of experiencing an arrhythmia-related incident was reported to be over two times the baseline within the initial four hours following use.[17] Additionally, a sudden-death registry from France recorded that about three percent of all deaths in individuals under thirty-five happened within hours of confirmed cannabis use, even when toxicology tests ruled out other drugs and autopsies showed no structural heart issues.[33]

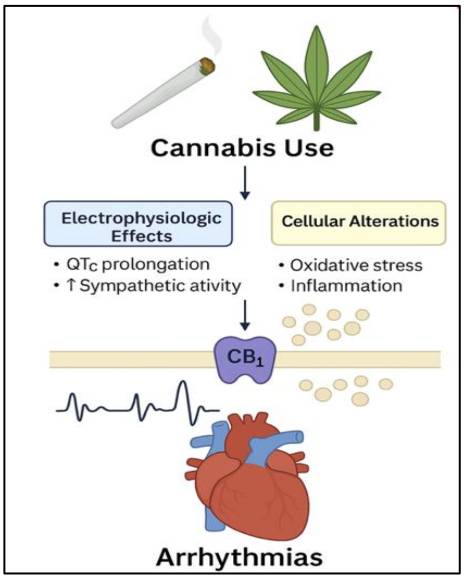

Figure 2: Mechanistic overview of cannabis-induced arrhythmogenesis.

Table 2: Observational and forensic evidence linking cannabis to cardiac arrhythmias.

Study/dataset	Population/setting	Key findings	Reference
Jouanjus et al	National pharmacovigilance system (France)	Cannabis implicated in serious cardiovascular events; 35% were arrhythmias	11
Halpin et al	Meta-analysis of 9 studies	Cannabis use associated with ~30% increased arrhythmia risk	13
Nguyen et al	Multicenter autopsy series	Cannabis-related sudden cardiac death without structural heart disease	32
Dupont et al	French national sudden death registry	3% of deaths in <35 y/o occurred within hours of cannabis use	33
Arora et al	Population registry	2× increase in arrhythmia-related events within 4 hours post-use	17

CLINICAL IMPLICATIONS

Synthetic cannabinoids require special attention in the context of cannabis-related arrhythmias. Promoted under benign labels like "herbal incense" or "legal highs," substances such as K2 and spice interact with the endocannabinoid system in significantly different ways than THC from plants. While THC functions as a partial CB_1 agonist, these lab-generated compounds attach to the receptor with much higher affinity and act as full agonists, thus enhancing autonomic surges and disrupting cardiac electrophysiology.

Clinical anecdotes-some featuring seemingly healthy young individuals-report sudden ventricular fibrillation and even cardiac arrest occurring within minutes of use.[20,27]

People with psychiatric disorders seem to exist at a notably precarious crossroads of cannabis exposure and the risk of arrhythmias. Epidemiological research shows that anxiety, major depression, bipolar disorder, and schizophrenia are all associated with elevated rates of cannabis use-frequently described by patients as a means of informal self-medication.[33,34] Medications often prescribed for these disorders-selective serotonin reuptake inhibitors, tricyclic antidepressants, and various antipsychotics-may each extend the QT interval. When this baseline repolarisation liability aligns with THC-induced autonomic changes and ion-channel blockage, the combined impact could push a borderline QT into a truly proarrhythmic zone, increasing the risk of torsades de pointes or other dangerous ventricular rhythms.[35] This stratified risk profile indicates that healthcare providers ought to consider cannabis use more thoroughly in medication guidance and evaluate specific ECG monitoring in patients whose psychiatric care already positions them close to the electrophysiological threshold.

Acute intoxication can transiently exacerbate psychotic features and appears to provoke a surge of catecholamines and cortisol, both of which may further destabilize cardiac electrophysiology.[34,36] The hemodynamic and autonomic load grows still heavier when cannabis is taken alongside other stimulants. Concurrent use of cocaine, methamphetamine, or prescription amphetamines effectively delivers a second sympathetic jolt: heart rate climbs, arterial pressure rises, myocardial oxygen demand increases, and the milieu becomes even more permissive for malignant arrhythmias.[36,37] Although systematic data remain sparse, this “double-hit” scenario underscores the importance of screening for polysubstance use whenever patients present with otherwise unexplained rhythm disturbances.

Given these layered vulnerabilities, a baseline electrocardiogram is a prudent starting point for several groups: patients with psychiatric disorders who acknowledge recent cannabis or stimulant use; individuals presenting with syncope or a history of arrhythmia; and anyone with a family record of sudden cardiac death. A brief tracing can unmask subtle red flags-prolonged QT, Brugada-like patterns, early repolarisation shifts-well before they evolve into a clinically catastrophic rhythm disturbance.[38]

Professional societies have begun to translate these observational signals into practical bedside advice. In a 2021 clinical update, the American college of cardiology advised obtaining a baseline electrocardiogram and offering tailored risk counselling to patients who consume cannabis regularly or in high doses-particularly when concomitant QT-prolonging medication is on board.[38] Public-health bodies such as the CDC, along with guideline groups like the Institute for Clinical Systems Improvement, now similarly urge clinicians to factor cannabis’s cardiovascular effects into routine assessment and follow-up, signalling a gradual but discernible shift from passive recognition to proactive risk mitigation.[39,40]

Harnessing health-information technology may furnish the pragmatic infrastructure that emerging guidelines now imply. Ideally, electronic medical-record systems would not simply document cannabis use but would also cross-reference it, in real time, with ECG findings and medication lists. A chart that records both habitual cannabis consumption and a borderline or prolonged QT interval, for example, could trigger a decision-support alert urging the clinician to revisit electrophysiologic risk and review any QT-stretching drugs. By flagging such constellations early, these digital prompts might intercept latent danger before it crystallises into a missed or mislabelled arrhythmia-an approach that dovetails with the most recent recommendations for proactive risk mitigation.[41]

FUTURE DIRECTIONS

Substantial uncertainty still exists regarding cannabis’s long-term electrophysiological impact, primarily because the evidence is largely made up of case reports and retrospective chart reviews. Comprehensive, forward-looking cohort studies are now crucial-preferably those that gather detailed information on dosage, frequency, method of administration (smoking, vaping, edibles), and the THC : CBD ratio-allowing for a more accurate correlation of specific exposure patterns with arrhythmia risk.[42] Randomized, placebo-controlled studies that focus on individual cannabinoids are also crucial; analyzing THC and CBD individually (and together) would help determine if their cardiovascular impacts are additive, antagonistic, or completely separate.[43]

Genetic diversity could also clarify the remarkable differences among individuals in susceptibility to arrhythmias. Variations in ion-channel genes like KCNH2 and SCN5A may potentially enhance the pro-arrhythmic effects of cannabis in vulnerable individuals.[8,36] Subsequent protocols may thus integrate specific genetic panels-or, on a larger scale, genome-wide association studies utilizing extensive population biobanks-to investigate gene-environment interactions with greater precision.[44] This comprehensive approach could ultimately enhance risk classification, advancing clinical practice from generic counseling to genuinely individualized advice on cannabis consumption.[44]

Digital technology now offers a complementary lens through which to examine cannabis-related arrhythmia risk. Smartphone apps that let users log each episode of cannabis consumption-especially when synchronized with wearable ECG devices-could capture fleeting rhythm changes that seldom reach clinical attention.[45] Aggregated and anonymized, such time-stamped data might populate public-health dashboards, revealing local “hot spots,” pinpointing problematic product types, or uncovering temporal associations between specific formulations and arrhythmic episodes.[45]

In the clinical workflow, electronic health-record platforms continue to be an underutilized asset. Decision-support modules might, for instance, automatically highlight charts where recorded cannabis use coincides with QT prolongation or a background of ventricular tachyarrhythmia, encouraging clinicians to reassess risk stratification and medication regimens.[41,46] When combined with focused provider training and point-of-care protocols, these integrated alerts can convert new research into practical prevention-closing the known gap between evidence creation and routine cardiovascular treatment.[47]

Inequalities in both exposure and outcome deserve the same level of focus. Social factors-such as limited access to regular healthcare, insecure housing, and inconsistent enforcement of cannabis regulations-determine who consumes cannabis, the frequency of use, and the circumstances under which care is eventually pursued. These identical structural factors can similarly influence arrhythmia risk, either by postponing diagnosis or by heightening co-factors like untreated hypertension and electrolyte imbalance. Subsequent research should thus chart cannabis-related cardiac incidents across racial, ethnic, and socioeconomic groups, making certain that marginalized communities-frequently the most severely punished yet least examined-do not remain overlooked in the developing body of evidence.[48]

CONCLUSION

As cannabis weaves its way into mainstream medicine and leisure, its capacity to provoke unexpected heart rhythm disturbances demands our full attention. The startling emergence of AF in young users, rapid-onset SVTs in previously healthy individuals, and rare yet dramatic episodes of ventricular fibrillation conjure a portrait of THC as a double-edged sword. Clinical intrigue grows as even modest exposures can tip the delicate autonomic scales and unmask hidden electrical vulnerabilities that lie dormant until provoked by cannabis.

In the consult room, a new level of detective work is required. Clinicians must learn to ask the right questions about cannabis habits with genuine curiosity rather than assumption. Incorporating targeted ECG screening for patients who use cannabis regularly or who take medications known to affect cardiac conduction can reveal early red flags. Collaborative conversations that explore personal motives for use and weigh individual risk factors create a therapeutic alliance where safety concerns are addressed without judgment.

Bridging knowledge gaps will call for bold partnerships across cardiology, psychiatry, primary care, and public health. Imagine rapid-response registries that capture real-time arrhythmia events linked to cannabis, or culturally tailored educational campaigns that resonate in dispensaries and clinics alike. By marrying vigilant bedside practice with innovative research frameworks, we can transform fragmented signals into clear guidelines. The challenge is to harness cannabis's promise while preventing its hidden perils, ensuring that each patient journey is both informed and safeguarded.

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