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Effects of Medications Used in ADHD on the Cardiovascular Risk: A Review of the Current Literature

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Abstract

Background: Attention-deficit/hyperactivity disorder (ADHD) is a common neurodevelopmental disorder frequently treated with stimulant and non-stimulant medications. Due to the increasing use of ADHD pharmacotherapy, concerns regarding cardiovascular safety have become increasingly important.

Aim: This review aimed to summarize current evidence regarding the cardiovascular effects of medications used in ADHD.

Materials and Methods: A narrative review was conducted using PubMed, Scopus, and Web of Science databases. Studies published between 2015 and 2026 evaluating cardiovascular outcomes associated with ADHD medications were included.

Results: Current evidence indicates that ADHD medications may cause modest increases in blood pressure and heart rate, mainly due to enhanced sympathetic nervous system activity. Stimulants demonstrated stronger hemodynamic effects, whereas α2-adrenergic agonists were associated with reductions in blood pressure and heart rate. Most studies did not demonstrate a substantial increase in serious cardiovascular events in the general ADHD population. However, higher cardiovascular risk may occur in patients with pre-existing cardiovascular disease, arrhythmias, or long-term cumulative medication exposure.

Conclusions: ADHD medications are generally associated with mild and manageable cardiovascular effects in most patients. Nevertheless, individualized cardiovascular risk assessment and regular monitoring remain important, particularly in high-risk populations.

Keywords: *cardiovascular risk; ADHD; ADHD management; ADHD medications*

1. Introduction

Attention-deficit/hyperactivity disorder (ADHD) is a childhood-onset neurodevelopmental disorder characterized by developmentally inappropriate levels of inattention, hyperactivity, and impulsivity that lead to functional impairment across multiple settings [1,2]. It represents a prevalent and clinically significant condition, with epidemiological studies estimating its occurrence at approximately 1.4–3.0% in European definitions and around 3.4% globally in children, with some meta-analyses suggesting prevalence as high as about 5.3% depending on diagnostic criteria [1,3]. Other reports indicate rates of approximately 5–7% among children and adolescents and 2–5% among adults, highlighting that ADHD persists beyond childhood in a substantial proportion of individuals [4]. Importantly, ADHD is associated with considerable clinical burden, including comorbid psychiatric conditions, impaired social and academic functioning, and increased long-term health risks [3]. The management of ADHD typically involves a multimodal approach, combining psychosocial interventions with pharmacotherapy, particularly in patients with more severe symptoms. Pharmacological treatment includes two main classes of medications: stimulants such as methylphenidate, dexmethylphenidate, amphetamine, dexamphetamine, lisdexamfetamine and non-stimulants including atomoxetine, guanfacine, clonidine [5–8]. Stimulant medications are considered first-line therapy and demonstrate high efficacy, with response rates exceeding 90% in some populations, whereas non-stimulant agents are used in cases of intolerance, contraindications, or specific clinical considerations [4]. In recent decades, the use of ADHD medications has increased substantially worldwide, reflecting greater recognition of the disorder as well as expanded access to pharmacological treatment [5].

This trend underscores the importance of evaluating the safety profile of these therapies, particularly their cardiovascular effects. Both stimulant and non-stimulant medications have been shown to increase heart rate and blood pressure in the short term, raising concerns about their potential long-term impact on cardiovascular health. Given that many individuals require prolonged treatment—often extending for several years or even more than five years in a significant proportion of patients—understanding these risks is of critical clinical relevance [6].

The mechanisms of action of ADHD medications are primarily related to modulation of catecholaminergic neurotransmission, especially dopamine and norepinephrine pathways, which improves attention and behavioral control but may also influence the cardiovascular

system through increased sympathetic activity [5]. Consequently, concerns have been raised regarding their potential contribution to cardiovascular disease (CVD). Recent large-scale observational studies suggest that longer cumulative exposure to ADHD medications may be associated with a modest but measurable increase in cardiovascular risk, with each additional year of use linked to approximately a 4% increase in CVD risk and higher risks observed for hypertension and arterial disease [6].

Given the widespread and often long-term use of ADHD pharmacotherapy, as well as the potential for cardiovascular effects, careful assessment of cardiovascular safety is essential. Therefore, the aim of this review is to analyze the impact of medications used in ADHD on the cardiovascular system, with particular emphasis on their mechanisms of action and long-term cardiovascular risk.

2. Materials and Methods

A narrative literature review was conducted using databases from: PubMed, Scopus, and Web of Science. The search included studies published between 2015 and 2026. The following keywords and their combinations were applied: "ADHD," "stimulant medications," "methylphenidate," "amphetamine," "atomoxetine," "cardiovascular risk," "blood pressure," "heart rate," and "arrhythmia." Inclusion criteria included: studies conducted in humans, focusing on medications used in ADHD, assessing cardiovascular parameters, and published in English. Exclusion criteria included: animal studies and studies without reported cardiovascular outcomes.

3. Results

3.1. Mechanisms of Action of ADHD Medications and Cardiovascular Effects

The cardiovascular effects of ADHD medications appear to be closely related to their pharmacological mechanisms. Stimulants, including methylphenidate, dexmethylphenidate, amphetamine, dexamphetamine, and lisdexamfetamine, act mainly by increasing dopaminergic and noradrenergic neurotransmission. This sympathomimetic activity may increase heart rate, blood pressure, and myocardial workload, which provides a biological explanation for the hemodynamic changes observed in clinical studies [7,9,10].

Methylphenidate was the most frequently studied stimulant. Its cardiovascular effects were usually described as small increases in systolic blood pressure, diastolic blood pressure, and heart rate, although some studies also reported changes in ventricular repolarization markers or increased short-term cardiovascular risk [11–13].

Non-stimulant medications showed a more heterogeneous cardiovascular profile. Atomoxetine, a selective norepinephrine reuptake inhibitor, may also increase noradrenergic tone and was associated with increases in blood pressure and heart rate similar to stimulants in some reviews, although usually with a weaker or less consistent long-term cardiovascular signal [6,14]. In contrast, α2-adrenergic agonists such as guanfacine and clonidine reduce sympathetic outflow and were generally associated with reductions in blood pressure and heart rate [7,10,14].

3.2. Effects on Blood Pressure and Heart Rate

Across the included studies, ADHD medications were most consistently associated with modest increases in blood pressure and heart rate. For stimulants, the reported mean increase in systolic blood pressure generally ranged from approximately +1 to +8 mmHg, while diastolic blood pressure increased by approximately +1 to +14 mmHg, depending on the study population and medication type [14–17]. In children treated with methylphenidate in the ADDUCE study, systolic blood pressure increased from 108 to 113 mmHg, and diastolic blood pressure from 65 to 67 mmHg over 24 months [11]. In a community sample of children, stimulant treatment was associated with approximately +3.0 mmHg higher systolic blood pressure and +2.8–3.0 mmHg higher diastolic blood pressure compared with children without ADHD [9].

Heart rate also increased with stimulant therapy. Across studies, the reported increase ranged from approximately +1 to +12.3 beats/min [9,15,16]. In the ADDUCE study, pulse rate increased from 80 to 83 bpm in children and adolescents treated with methylphenidate [11]. In another pediatric study, children receiving methylphenidate had a significantly higher heart rate than controls, 91.6 ± 18 bpm vs 83.3 ± 14 bpm [12]. The largest reported difference was observed in a community-based pediatric sample, where children taking stimulants had a 12.3 bpm higher heart rate than controls [9].

The effect differed by drug class. Guanfacine reduced heart rate and blood pressure, whereas dexmethylphenidate increased them. Combination therapy showed an intermediate profile, with no significant change in heart rate or systolic blood pressure and only a moderate increase in diastolic blood pressure [7]. Overall, the observed hemodynamic changes were usually small and often not clinically significant in healthy individuals, but they may be relevant in patients with hypertension, congenital heart disease, arrhythmias, or other cardiovascular risk factors [10,18].

3.3. Risk of Arrhythmias and Cardiac Events

The relationship between ADHD medications and arrhythmias was inconsistent. Several large observational studies did not find a significant increase in serious arrhythmias, myocardial infarction, stroke, or sudden cardiac death in the general pediatric or adult population [19–21]. However, some studies identified specific risk signals, particularly shortly after medication initiation or in high-risk populations.

In a Japanese self-controlled case series, atomoxetine was associated with an increased risk of arrhythmia, especially during the first 7 days after initiation, with an adjusted incidence rate ratio of 6.22 and a subsequent exposure risk of 3.23. No similar risk was detected for methylphenidate in that study [22]. In children and adolescents using methylphenidate, another self-controlled case series reported an increased arrhythmia risk during exposure, RR 1.41, with the highest risk during the first 1–14 days, RR 2.17; however, increased risk was also present before exposure, suggesting possible confounding by underlying clinical factors [23].

Some studies reported increased risk of broader cardiac events. In children and young adults aged 5–30 years, ADHD medication use was associated with increased cardiac events, mainly driven by undefined arrhythmias, with aOR 2.66, while defined arrhythmias and cardiac arrest were not significantly increased [24]. In a nationwide study of out-of-hospital cardiac arrest, methylphenidate use was associated with higher odds of cardiac arrest, OR 1.78, with the highest risk among recent starters, OR 2.59 within 180 days [25].

In contrast, studies in children with congenital heart disease did not show a clear increase in severe outcomes. In one cohort of children with CHD, stimulant use was not associated with sudden cardiac death, clinically meaningful blood pressure changes, QTc prolongation, or life-threatening arrhythmias [26]. Another CHD study found only one cardiovascular adverse event among 831 children, corresponding to a prevalence of approximately 0.12–0.21%, and no cases of sudden cardiac death [27].

3.4. Differences Between Stimulant and Non-Stimulant Medications

Stimulants generally showed a stronger hemodynamic effect than non-stimulant α2-agonists. Methylphenidate, amphetamine derivatives, and lisdexamfetamine were associated with increases in blood pressure and heart rate, whereas guanfacine and clonidine tended to lower both parameters [10,14,16].

Atomoxetine occupied an intermediate position. Although classified as non-stimulant, it increases norepinephrine signaling and may raise blood pressure and heart rate. In the long-term Swedish study, atomoxetine showed only a slight increase in cardiovascular risk during the first year, whereas stimulants, especially methylphenidate and lisdexamfetamine, showed stronger and more persistent associations with cardiovascular disease risk [6]. A rare case report described atomoxetine-related Takotsubo cardiomyopathy, probably linked to excessive norepinephrine activity and interaction with fluoxetine, a CYP2D6 inhibitor [28].

Lisdexamfetamine did not show a clearly higher risk than other ADHD medications in a population-based cohort from Denmark and Sweden. The pooled incidence rate ratio for major cardiovascular and cerebrovascular events was 1.10, indicating little to no increased risk compared with previous users of other ADHD drugs [20]. In contrast, long-term cumulative stimulant exposure was associated with higher cardiovascular risk in other studies, suggesting that treatment duration and dose may be more important than drug class alone [6,29].

3.5. Cardiovascular Risk in Special Populations

The clinical significance of cardiovascular effects appears to depend strongly on baseline cardiovascular risk. In older adults, stimulants may increase blood pressure by approximately 1–5 mmHg and heart rate by 4–10 bpm, which may be clinically relevant because this population has a higher baseline prevalence of hypertension, arrhythmias, diabetes, and cardiovascular disease [30]. In adults initiating ADHD treatment, older age was an important predictor of short-term cardiovascular disease, with total incident CVD occurring in 1.7% of the cohort over 2 years [31].

Patients with pre-existing cardiovascular disease or cardiac channelopathies require caution. In patients with long-QT syndrome, ADHD medication was associated with a markedly increased risk of cardiac events: 35.4% in the treated group versus 15.6% in controls, with a long-term cumulative risk of 62% vs 28% and a multivariate hazard ratio of 3.07. The risk was especially high in males, HR 6.80, despite no significant difference in QTc interval, suggesting that sympathetic activation rather than QT prolongation itself may drive risk [32].

Comorbidities were also important predictors of adverse cardiovascular outcomes. In a large lifespan study of stimulant users, the strongest predictor was atherosclerotic heart disease, aOR 36.7, followed by hypertension, aOR 2.78, diabetes, aOR 1.56, obesity, aOR

1.45, cocaine use, aOR 1.64, depression, aOR 1.55, and anxiety, aOR 1.46. ADHD itself was not an independent cardiovascular risk factor after adjustment [18].

These findings support the need for cardiovascular screening before treatment initiation. Most sources emphasize baseline assessment of personal and family cardiac history, blood pressure, and heart rate, with ECG or cardiology consultation reserved for patients with known heart disease, symptoms, abnormal examination, family history of sudden death, or abnormal ECG findings [10,17,33].

3.6. Long-Term Cardiovascular Outcomes

Long-term data remain limited but are increasing. The most consistent long-term signal was related to hypertension and arterial disease rather than acute myocardial infarction, stroke, or sudden cardiac death. In the Swedish case-control study including individuals aged 6–64 years, longer cumulative ADHD medication use was associated with higher cardiovascular disease risk. Compared with non-use, the adjusted odds ratio increased from 1.09 after 1–2 years to 1.27 after 3–5 years and 1.23 after more than 5 years. Each additional year of medication use was associated with approximately 4% higher CVD risk. The strongest associations were observed for hypertension, with AOR 1.72 after 3–5 years and AOR 1.80 after more than 5 years, and arterial disease, with AOR 1.65 after 3–5 years [6].

In adults, higher-intensity ADHD treatment was associated with modestly increased 10-year cardiovascular risk. Compared with prior users, individuals receiving ≥1 defined daily dose per day had a higher standardized 10-year absolute risk of stroke, 2.1% vs 1.7%, heart failure, 1.2% vs 0.7%, and composite cardiovascular outcome, 3.9% vs 3.0%. The corresponding risk ratios were 1.2 for stroke, 1.7 for heart failure, and 1.3 for the composite outcome. No clear association was found for acute coronary syndrome [29].

In contrast, pediatric methylphenidate studies generally did not show increased major cardiovascular events or mortality. A large Taiwanese pediatric cohort found no increased risk of major cardiovascular events, arrhythmia, acute coronary syndrome, stroke, cardiogenic shock, or death among methylphenidate users [19]. The ADDUCE study found no serious cardiovascular adverse events over 2 years, although methylphenidate was associated with increases in blood pressure and pulse rate, supporting continued monitoring rather than avoidance of treatment [11].

Overall, the evidence suggests that ADHD medications commonly cause small increases in blood pressure and heart rate. Serious cardiovascular events appear rare in the general population, especially in children and adolescents, but risk may increase with higher

cumulative exposure, higher doses, older age, pre-existing cardiovascular disease, hypertension, long-QT syndrome, and other comorbid risk factors. Therefore, the available data do not provide unequivocal evidence of increased cardiovascular mortality in the general ADHD population, but they support individualized cardiovascular risk assessment and regular monitoring during treatment.

4. Discussion

4.1. Pathophysiological Basis of Cardiovascular Effects

The cardiovascular effects of ADHD medications are primarily associated with modulation of catecholaminergic neurotransmission and activation of the sympathetic nervous system. Stimulant medications, including methylphenidate, dexmethylphenidate, amphetamine, dexamphetamine, and lisdexamfetamine, increase synaptic concentrations of dopamine and norepinephrine through inhibition of reuptake transporters, while amphetamine derivatives additionally promote catecholamine release. Increased adrenergic stimulation subsequently leads to elevated heart rate, peripheral vasoconstriction, and increased blood pressure, findings that were consistently reported across both clinical trials and observational studies [9,14,15].

The reviewed literature also suggests that enhanced sympathetic activation may contribute to arrhythmogenesis. Topriceanu et al. described the potential pro-arrhythmogenic effect of stimulants and atomoxetine through β-adrenergic stimulation and increased myocardial excitability. Similar conclusions were presented in broader reviews of psychiatric medications, where stimulants were linked to tachycardia, QT-related abnormalities, and increased cardiovascular stress. ECG-based studies further support this mechanism. In children treated with methylphenidate, ventricular repolarization markers such as QT dispersion, QTc dispersion, and TpTe-related indices were significantly prolonged compared with controls, suggesting possible subclinical proarrhythmogenic effects even in the absence of overt arrhythmias [12].

Atomoxetine demonstrates a somewhat different but partially overlapping mechanism. As a selective norepinephrine reuptake inhibitor, it increases sympathetic activity indirectly and may therefore produce similar cardiovascular effects to stimulants [10,14]. This neurohormonal mechanism may explain rare reports of severe catecholamine-mediated complications, including atomoxetine-associated Takotsubo cardiomyopathy accompanied by markedly elevated norepinephrine concentrations [28].

In contrast, centrally acting α2-adrenergic agonists such as guanfacine and clonidine reduce sympathetic outflow from the central nervous system and are generally associated with reductions in blood pressure and heart rate [7,14]. Their cardiovascular profile therefore differs substantially from that of stimulants and atomoxetine and may be advantageous in selected high-risk patients.

4.2. Clinical Significance of Observed Changes

One of the most consistent findings across the reviewed studies was the presence of modest but measurable increases in blood pressure and heart rate during stimulant treatment. Mean increases in systolic blood pressure generally ranged from approximately 1–8 mmHg, while heart rate increases ranged from approximately 1–12 bpm depending on age group, medication type, and study design [9,15,16]. The ADDUCE study additionally demonstrated that these hemodynamic effects may persist for at least two years during methylphenidate treatment, although no serious cardiovascular adverse events were reported [11].

Although such changes are usually described as small, their clinical interpretation remains complex. In healthy children and adults without cardiovascular disease, these alterations are often considered clinically insignificant and rarely require treatment discontinuation. This interpretation is supported by several large cohort studies and systematic reviews that failed to demonstrate a clear increase in myocardial infarction, stroke, or sudden cardiac death in the general ADHD population [19–21].

However, the distinction between statistical significance and clinical significance is particularly important in cardiovascular medicine. Even small persistent increases in blood pressure and heart rate may contribute to long-term vascular remodeling, endothelial dysfunction, arterial stiffness, and cumulative cardiovascular burden. Longitudinal observational studies support this possibility. Zhang et al. demonstrated that cumulative ADHD medication exposure was associated with increased long-term risk of hypertension and arterial disease, with risk increasing progressively with longer treatment duration [6]. Similarly, Holt et al. reported increased long-term risk of stroke, heart failure, and composite cardiovascular outcomes in adults receiving higher-intensity ADHD treatment [29].

Another important issue is substantial interindividual variability. Most patients experience only mild hemodynamic changes, whereas some individuals may develop clinically significant hypertension, tachycardia, palpitations, or ECG abnormalities. This variability may explain why randomized trials generally report reassuring safety findings while case reports and pharmacovigilance studies occasionally describe severe cardiovascular events, including

ventricular arrhythmias, cardiac arrest, Takotsubo cardiomyopathy, or sudden cardiac death [25,28].

4.3. Safety Profile and Risk Stratification

Overall, the available evidence suggests that ADHD medications have a relatively favorable cardiovascular safety profile in most children, adolescents, and adults without major cardiovascular disease. Multiple large observational studies did not demonstrate a substantial increase in serious cardiovascular outcomes such as myocardial infarction, stroke, sudden cardiac death, or cardiovascular hospitalization [19–21].

Nevertheless, the literature consistently indicates that cardiovascular risk is not uniform across patient populations. Several reviews emphasized that patients with congenital heart disease, cardiomyopathy, inherited arrhythmia syndromes, hypertension, or established cardiovascular disease may represent higher-risk groups requiring individualized assessment. In patients with long-QT syndrome, ADHD medication was associated with markedly increased risk of cardiac events, particularly among males, despite the absence of major QT prolongation itself [32]. These findings suggest that sympathetic stimulation rather than direct QT prolongation may be the primary mechanism underlying increased arrhythmic risk in predisposed individuals.

Age also appears to influence cardiovascular vulnerability. Older adults may be particularly susceptible because of higher baseline prevalence of hypertension, atherosclerosis, diabetes, and structural heart disease. Kooij et al. highlighted that even small increases in blood pressure and heart rate may become clinically important in elderly patients with multiple cardiovascular comorbidities [30]. Similarly, Holt et al. found that cardiovascular risk among stimulant users was strongly driven by traditional cardiovascular risk factors such as hypertension, diabetes, obesity, atherosclerotic disease, depression, anxiety, and substance use rather than ADHD itself [18].

Importantly, studies involving congenital heart disease populations provided somewhat reassuring findings. Both pediatric CHD studies demonstrated extremely low rates of severe cardiovascular events during stimulant treatment and did not identify increased sudden cardiac death risk [26,27]. These results suggest that stimulant therapy may be feasible even in selected cardiac populations when careful monitoring is provided.

Taken together, these findings support an individualized risk stratification approach rather than universal avoidance of ADHD medications. For many patients, the benefits of effective ADHD treatment likely outweigh the relatively small cardiovascular risks, particularly

considering the functional impairment and psychosocial burden associated with untreated ADHD.

4.4. Monitoring and Clinical Recommendations

Most reviewed studies and expert recommendations emphasize the importance of baseline cardiovascular assessment before initiating ADHD pharmacotherapy. Recommended evaluation includes measurement of blood pressure and heart rate, detailed cardiovascular history, and assessment of family history of arrhythmias, cardiomyopathy, congenital heart disease, or sudden cardiac death. Similar recommendations were repeated across several reviews and consensus papers [15,17].

Routine monitoring during treatment is also considered essential because cardiovascular effects may persist throughout therapy. The ADDUCE study demonstrated sustained increases in blood pressure and pulse rate over two years of methylphenidate exposure, supporting the need for long-term monitoring even in otherwise healthy children [11]. In older adults and patients with pre-existing cardiovascular disease, closer follow-up may be particularly important because small hemodynamic changes may become clinically meaningful over time [30].

The role of routine ECG screening remains controversial. Most publications concluded that universal ECG screening before stimulant initiation is not supported by current evidence because clinically significant ECG abnormalities and severe cardiovascular events remain rare in the general ADHD population [15,17]. However, cardiology consultation and ECG evaluation are recommended in patients with syncope, abnormal cardiac examination findings, known structural heart disease, inherited arrhythmia syndromes, or family history of sudden cardiac death.

Several studies additionally suggest that the early phase after treatment initiation may represent a period of increased cardiovascular vulnerability. Increased arrhythmia risk shortly after starting methylphenidate or atomoxetine was observed in multiple self-controlled case series studies [22,23]. Similarly, the risk of out-of-hospital cardiac arrest was highest among recent methylphenidate starters [25]. These findings suggest that closer monitoring may be particularly important during treatment initiation and dose escalation.

4.5. Limitations of Current Evidence

Despite the growing number of studies investigating cardiovascular safety of ADHD medications, the current evidence base remains limited in several important aspects. First, long-term prospective randomized studies are scarce. Most randomized controlled trials lasted

only weeks or months and were not powered to detect rare cardiovascular outcomes such as sudden cardiac death or serious ventricular arrhythmias [34,35]. Consequently, most data regarding severe cardiovascular outcomes derive from observational studies, registry analyses, and pharmacovigilance reports.

Second, substantial heterogeneity exists across studies. Included populations varied markedly with respect to age, cardiovascular risk profile, medication type, dosage, treatment duration, and presence of psychiatric or cardiovascular comorbidities. Some studies focused exclusively on pediatric populations, whereas others included adults, elderly individuals, or patients with congenital heart disease and inherited arrhythmia syndromes [26,30]. This heterogeneity complicates direct comparison of findings and limits generalizability.

Another important limitation is incomplete physiological characterization. Many large observational studies evaluated only clinical cardiovascular diagnoses without direct measurements of blood pressure, heart rate, ECG changes, or arrhythmia subtype analysis [6,29]. Lifestyle-related confounders such as smoking, obesity, physical activity, socioeconomic status, and medication adherence were frequently unavailable or incompletely controlled.

Potential detection bias must also be considered. Patients receiving ADHD medications are often monitored more intensively than untreated individuals, increasing the likelihood of identifying mild arrhythmias or cardiovascular abnormalities [24]. Furthermore, several self-controlled studies demonstrated elevated cardiovascular risk even during pre-exposure periods, suggesting that underlying patient characteristics or prodromal cardiovascular symptoms may partially explain some associations [23].

Finally, publication bias may influence current understanding of cardiovascular safety. Rare and severe adverse events are more likely to be reported in case reports and pharmacovigilance databases, whereas studies demonstrating neutral findings may be underrepresented. Consequently, interpretation of isolated reports of serious complications should be performed cautiously and in the context of larger population-based studies.

Despite these limitations, the available literature consistently indicates that ADHD medications are associated with measurable cardiovascular effects, predominantly involving modest increases in blood pressure and heart rate. Serious cardiovascular complications appear uncommon in the general population, although specific high-risk groups may require individualized assessment and closer cardiovascular monitoring.

5. Conclusion

Medications used in ADHD have measurable effects on the cardiovascular system, mainly through increased sympathetic nervous system activity. Both stimulant and non-stimulant drugs may increase heart rate and blood pressure, although these changes are usually mild and clinically insignificant in most patients. Stimulants generally produce stronger hemodynamic effects, while α2-adrenergic agonists such as guanfacine and clonidine may lower blood pressure and heart rate. Current evidence suggests that serious cardiovascular events, including myocardial infarction, stroke, sudden cardiac death, and severe arrhythmias, are uncommon in the general ADHD population. However, some studies indicate that cardiovascular risk may increase with long-term treatment duration, higher cumulative exposure, and in patients with pre-existing cardiovascular disease or other risk factors. These findings highlight the importance of individualized cardiovascular risk assessment before treatment initiation and regular monitoring during therapy, particularly in high-risk populations. Blood pressure, heart rate, and cardiovascular history should be routinely evaluated, while cardiology consultation may be necessary in selected patients with structural heart disease, arrhythmias, or family history of sudden cardiac death. Although the overall cardiovascular safety profile of ADHD medications appears favorable, further long-term prospective studies are needed, especially in high-risk populations, to better understand the long-term cardiovascular consequences of chronic ADHD pharmacotherapy.

Disclosure

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