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MEDICATIONS USED IN THE TREATMENT OF ADHD AND THEIR EFFECTS ON THE CARDIOVASCULAR SYSTEM

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ABSTRACT

Attention-deficit/hyperactivity disorder (ADHD) is a common neurodevelopmental disorder frequently requiring long-term pharmacological treatment. The medications used in ADHD management, particularly stimulant agents and non-stimulant therapies, may influence cardiovascular function through effects on sympathetic activity, heart rate, and vascular tone. This review summarizes the available evidence regarding the potential association between ADHD medications and cardiovascular outcomes, with particular emphasis on arrhythmias and changes in arterial blood pressure. We analyze findings from previously published studies evaluating the effects of pharmacological treatment on blood pressure, heart rate, and the occurrence or risk of cardiac rhythm disturbances in children, adolescents, and adults with ADHD. Current evidence suggests that ADHD medications may be associated with modest changes in blood pressure and heart rate, although the clinical significance of these effects varies among individuals and treatment modalities. The potential risk of arrhythmias remains an important consideration, particularly in patients with pre-existing cardiovascular disease or other risk factors. The review highlights the importance of individualized risk assessment, baseline cardiovascular evaluation, and appropriate monitoring during treatment. Further research is needed to clarify long-term cardiovascular outcomes and identify patients who may be particularly susceptible to adverse cardiovascular effects.

KEYWORDS

Attention-Deficit/Hyperactivity Disorder (ADHD); ADHD Medications; Cardiovascular Effects; Arrhythmias; Blood Pressure; Cardiovascular Monitoring

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I. Introduction

Attention Deficit Hyperactivity Disorder (ADHD) is a neurodevelopmental disorder that is being diagnosed with increasing frequency in both adults and children. It affects around 1 million adults in Poland. [1]

Based on data published by the National Health Fund, a sharp rise in the number of patients diagnosed with ADHD can be observed. In 2010, the figure stood at 32,035, whilst in 2024 it reached 64,090 cases, representing an increase of 100.1 per cent over 15 years. [2]

The global prevalence of ADHD among children and adolescents is estimated at 8.0% (95% CI 6.0–10%). [3] A lack of proper diagnosis can have serious consequences for the patient and their loved ones. People with untreated ADHD experience difficulties in their personal and professional lives. The accompanying emotional dysregulation, impaired self-esteem and difficulties in social functioning significantly reduce their quality of life [4]. Treatment of this disorder involves non-pharmacological interventions such as psychological support and cognitive-behavioural therapy, as well as pharmacotherapy. Medicines are used whose mechanism of action is based on modulating the availability of neurotransmitters such as noradrenaline and dopamine. These neurotransmitters are characterised by their versatile action, which means they affect not only the central nervous system but also the circulatory system.

II. Methodology

A narrative literature review was conducted to identify and synthesize available evidence regarding the potential cardiovascular effects of medications used in the treatment of attention-deficit/hyperactivity disorder (ADHD), with particular emphasis on arrhythmias and changes in arterial blood pressure. Relevant publications were identified through searches of the PubMed database and additional scientific databases and online sources. The search included publications in both English and Polish. The search strategy used combinations of keywords and phrases related to ADHD, pharmacological treatment, stimulants, non-stimulant medications, methylphenidate, amphetamine derivatives, atomoxetine, guanfacine, clonidine, cardiovascular effects, arrhythmias, tachycardia, blood pressure, hypertension, heart rate, and cardiovascular risk. The review included original research articles, systematic reviews, meta-analyses, and other relevant peer-reviewed publications addressing the cardiovascular safety and hemodynamic effects of ADHD medications. Studies were selected based on their relevance to the objectives of the review, with particular attention given to evidence concerning changes in systolic and diastolic blood pressure, heart rate, cardiac rhythm disturbances, and other clinically relevant cardiovascular outcomes. Due to the narrative nature of the review, the identified evidence was synthesized qualitatively rather than through a formal statistical meta-analysis.

III. Results:

Pharmacotherapy for ADHD

Medicines used to treat ADHD can be divided into two main categories: psychostimulants and non-stimulants. Psychostimulants include: methylphenidate and amphetamine derivatives such as: lisdexamfetamine, dextroamphetamine and amphetamine salts.

These are first-line treatments which are also used successfully in children. They are characterised by good efficacy and a rapid onset of action. However, it should be borne in mind that there is a risk of dependence.

The group of non-stimulant medicines includes: memantine, atomoxetine, modafinil, clonidine, bupropion and guanfacine. They have a lower potential for dependence and are often effective in patients who cannot tolerate stimulants. A summary of the information on pharmacotherapy is presented in Table 1. [5]

Table1. Pharmacotherapy for ADHD

Type of medication	Medication / Active substance	Mechanism of action
Stimulant	Methylphenidate	Inhibits the reuptake of dopamine and norepinephrine by blocking the dopamine and norepinephrine transporters, thereby increasing the concentration of these neurotransmitters in the synaptic cleft.
Stimulant	Lisdexamfetamine	A prodrug of dexamfetamine that increases the availability of norepinephrine and dopamine in the central nervous system, primarily by promoting their release and inhibiting their reuptake.
Stimulant	Dexamfetamine (Dextroamphetamine)	A sympathomimetic amine and the dextro enantiomer of amphetamine. It increases central dopaminergic and noradrenergic neurotransmission, primarily by promoting the release of dopamine and norepinephrine and inhibiting their reuptake.
Non-stimulant	Memantine	An uncompetitive antagonist of N-methyl-D-aspartate (NMDA) receptors, modulating glutamatergic neurotransmission.
Non-stimulant	Atomoxetine	A selective norepinephrine reuptake inhibitor that increases norepinephrine concentrations in the synaptic cleft, particularly in the prefrontal cortex.
Non-stimulant	Modafinil	Binds to the norepinephrine transporter and inhibits norepinephrine reuptake; it also affects several other neurotransmitter systems involved in wakefulness and cognitive function.
Non-stimulant	Clonidine	An α2-adrenergic receptor agonist acting in the central nervous system. It reduces sympathetic outflow and modulates norepinephrine release, including in the prefrontal cortex.
Non-stimulant	Bupropion	Inhibits the reuptake of dopamine and norepinephrine, thereby increasing their concentrations in the synaptic cleft.
Non-stimulant	Guanfacine	A selective α2A-adrenergic receptor agonist that modulates noradrenergic signaling in the prefrontal cortex and basal ganglia through direct effects on α2A-adrenergic receptors.

Cardiac Arrhythmias

Epidemiology and Proarrhythmic Risk Assessment

Evaluating the cardiovascular safety of ADHD pharmacotherapy heavily relies on tracking cardiac arrhythmias as a primary endpoint. Current epidemiological data link methylphenidate directly to increased proarrhythmic risk, though the absolute incidence remains highly dependent on individual patient vulnerability. In the general population, the adjusted incidence rate ratio (IRR) for arrhythmia during methylphenidate treatment is 1.61 (95% CI: 1.48–1.74).

This risk demonstrates a highly distinct chronological pattern, peaking sharply within the first three days of starting the drug (IRR: 2.01; 95% CI: 1.74–2.31) (Shin et al., 2016). This acute phase spike suggests a profound initial autonomic shock before the cardiovascular system develops homeostatic tolerance. Following this initial window, the risk gradually declines. Long-term exposure exceeding 56 days does not appear to trigger any further statistically significant increase in risk compared to non-exposure periods.

However, baseline cardiac anatomy fundamentally changes this risk equation. Congenital heart defects remain the main factor compounding this danger. The IRR climbs to 3.49 (95% CI: 2.33–5.22) in patients with structural anomalies, compared to just 1.34 (95% CI: 1.23–1.46) in those without them. This stark divergence emphasizes that while the healthy myocardium tolerates psychostimulant-induced stress relatively well, a heart with compromised structural or electrical architecture possesses a significantly lower threshold for tachyarrhythmias. [6]

Pathomechanism of Arrhythmia Induction

Enhanced sympathetic neurotransmission drives arrhythmia induction during ADHD therapy. Most prescribed agents elevate dopamine and norepinephrine levels in the synaptic cleft by blocking their respective

reuptake transporters (DAT and NET) or triggering presynaptic vesicle release. Beyond central nervous system effects, this peripheral sympathetic surge stimulates cardiac beta-1 and beta-2 receptors, as well as vascular alpha-1 receptors.

The resulting clinical fallout includes enhanced automaticity of the cardiac conduction system, an accelerated heart rate (positive chronotropic effect), and increased myocardial contractility (positive inotropic effect). Concurrently, peripheral vasoconstriction increases systemic vascular resistance, elevating cardiac afterload and myocardial oxygen demand. These electrophysiological shifts manifest as sinus tachycardia, premature supraventricular or ventricular beats, and complex tachyarrhythmias, particularly in patients with an underlying arrhythmic substrate. [7]

At the cellular level, excessive stimulation of myocardial beta-1 adrenergic receptors triggers a G-protein-coupled cascade that activates adenylyl cyclase, raising intracellular cyclic adenosine monophosphate (cAMP) levels. This increase activates protein kinase A (PKA), which phosphorylates L-type calcium channels and ryanodine receptors (RyR2) in the sarcoplasmic reticulum. The resulting intracellular calcium overload accelerates phase 4 spontaneous depolarization in pacemaker cells, drastically increasing automaticity.

Furthermore, this altered calcium handling promotes delayed afterdepolarizations (DADs), which can trigger ventricular premature beats. Under conditions of patchy, non-uniform repolarization induced by chronic norepinephrine exposure, these premature beats can easily initiate functional reentry circuits.

While mild sinus tachycardia and premature beats dominate the clinical picture, literature also documents rarer, severe events like paroxysmal atrial fibrillation or drug-induced QTc interval prolongation, which can precipitate life-threatening ventricular tachyarrhythmias. Chronic sympathetic overstimulation can also induce subtle myocardial focal ischemia and subsequent fibrotic remodeling. This structural alteration creates a permanent anatomical substrate for lethal reentrant pathways, occasionally presenting as amphetamine-dextroamphetamine-induced cardiomyopathy and secondary heart failure in otherwise healthy young adult populations [8].

Evidence from Large-Scale Cohort Studies

Recent large-scale population studies offer strong evidence regarding these risks. Shin et al. (2016) tracked a cohort of 1,224 children and adolescents experiencing cardiovascular complications and found that arrhythmias were the most frequent presentation of methylphenidate-induced cardiotoxicity (n = 864), occurring far more often than general cardiac conditions (n = 396), myocardial infarction (n = 52), ischemic stroke (n = 67), or heart failure (n = 44). Subgroup analyses revealed no significant difference in arrhythmia rates between low and high doses, indicating that individual biological susceptibility matters more than the actual dosage. This lack of a clear dose-response relationship strongly suggests an idiosyncratic or threshold-based mechanism rather than a predictable, cumulative toxic effect. [6]

Data from a Swedish case-control study of over 112,000 cardiovascular cases confirmed that active ADHD therapy increases the overall risk of cardiac events (aOR: 1.63; 95% CI: 1.47–1.81). The adjusted odds ratio for a composite endpoint of sudden cardiac arrest and arrhythmias reached 2.54 (95% CI: 2.17–2.96), holding true even after adjusting for somatic and psychiatric comorbidities. This study notably highlighted a cumulative risk profile: patients undergoing uninterrupted, multi-year stimulant therapy demonstrated a progressive increase in cardiovascular risk, suggesting that long-term sympathetic stress may cause insidious vascular or myocardial wear. [9]

The drug elimination (washout) period also yields critical insights. Shin et al. (2016) noted that the risk of acute coronary events spiked transiently right after discontinuing methylphenidate compared to non-exposure periods. Abrupt dosage changes or sudden withdrawal may induce a "rebound" effect and secondary autonomic instability, further predisposing the patient to rhythm disturbances. This rebound phenomenon likely stems from the sudden up-regulation and hypersensitivity of peripheral adrenergic receptors during chronic blockade evasion, making the myocardium temporarily hyper-reactive to endogenous catecholamine fluctuations upon drug cessation. [6]

Differences in the Safety Profile of Psychostymulants

Amphetamine derivatives (dextroamphetamine and lisdexamfetamine) carry a distinct proarrhythmic profile. Because they simultaneously inhibit reuptake and stimulate catecholamine release, they provoke a more robust adrenergic response than methylphenidate. While methylphenidate acts primarily as a competitive blocker of DAT and NET, amphetamines serve as substrates for these transporters. [7]

Once inside the presynaptic neuron, amphetamines disrupt the vesicular monoamine transporter 2 (VMAT2), forcing massive quantities of dopamine and norepinephrine out of synaptic vesicles and into the

cytoplasm. This process ultimately reverses the direction of DAT and NET, pumping a massive surge of catecholamines directly into the synaptic cleft.

A recent meta-analysis of 14 studies (N > 3.6 million participants) tied general psychostymulant use to an 83% increase in arrhythmia risk (HR: 1.83; 95% CI: 1.34–2.50). Stratified data showed the highest risk with amphetamines (HR: 2.67) compared to methylphenidate (HR: 1.65). This difference highlights the clinical impact of their varying mechanisms of action.

The sustained, massive surge in synaptic norepinephrine triggered by amphetamines leads to a much more pronounced increase in both systolic and diastolic blood pressure, adding significant mechanical strain to the electrical instability of the heart. Even so, none of these large population studies demonstrated a statistically significant rise in sudden cardiac death (SCD) or ventricular fibrillation (VF), confirming that life-threatening complications remain rare. [10]

Controversies and Limitations of Population Data

Some reports voice caution against broad generalizations despite this proven cardiotoxic potential. A meta-analysis by Zhang et al. (2022) involving nearly 3.9 million patients showed no global increase in cardiovascular risk. However, high heterogeneity and a low proportion of patients with baseline cardiac conditions limit these findings.

Many large-scale cohorts suffer from a "healthy user bias," where clinicians proactively avoid prescribing stimulants to patients with known or suspected cardiac issues. This selective prescribing artificially inflates the apparent safety profile of these medications in retrospective data.

Furthermore, younger populations dominate these studies, potentially masking the true cardiotoxic potential of these drugs. In younger individuals, robust physiological reserves and healthy coronary arteries can easily mask underlying electrical instability.

The accompanying editorial commentary stressed the need for strict clinical vigilance in elderly patients and individuals with established cardiovascular disease, whose coronary and electrophysiological reserves are already compromised. In these vulnerable populations, even a modest increase in heart rate or blood pressure can disrupt the delicate balance between myocardial oxygen supply and demand, potentially triggering silent ischemic events or severe tachyarrhythmias. [9]

Clinical Conclusions and Screening Recommendations

Although ADHD medications elevate relative proarrhythmic risk, the absolute incidence in the general population remains low, meaning neuropsychiatric benefits typically outweigh potential harms. Maximizing safety depends entirely on rigorous pre-treatment screening. Current guidelines mandate a comprehensive cardiovascular history—focusing on syncope, palpitations, chest pain, and a family history of SCD—before writing a prescription (Brown et al., 2018). Clinicians should also screen for history of unexplained exercise intolerance or sudden, premature deaths in first-degree relatives, which might point to undiagnosed channelopathies like Long QT Syndrome or Brugada Syndrome.

Doctors should routinely check heart rate and blood pressure following treatment initiation and any subsequent dose adjustments. For patients in high-risk structural or genetic groups, securing a prior specialist consultation and a baseline 12-lead ECG remains mandatory.

When evaluating the pre-treatment ECG, clinicians must meticulously measure the corrected QT interval (QTc) using the Bazett or Fridericia formula, and watch for signs of left ventricular hypertrophy, pre-excitation syndromes (such as a short PR interval and delta wave), or pathological T-wave inversions. If a patient develops persistent tachycardia (resting heart rate greater than 100 beats per minute) or new-onset palpitations during treatment, clinicians should immediately hold the medication and order a 24-hour Holter monitor to rule out paroxysmal arrhythmias before safely restarting therapy. [7]

Effects of ADHD Medications on Arterial Blood Pressure

Among the cardiovascular parameters affected by ADHD pharmacotherapy, changes in blood pressure have received particular attention due to the potential implications of sustained hypertension for long-term cardiovascular health. Methylphenidate and amphetamine derivatives exert their therapeutic effects primarily by increasing synaptic concentrations of dopamine and norepinephrine. Enhanced noradrenergic neurotransmission increases sympathetic nervous system activity, stimulating adrenergic receptors in the heart and blood vessels, which may increase vascular tone, cardiac output, and peripheral vascular resistance, ultimately resulting in modest elevations in blood pressure and heart rate. These haemodynamic changes are generally considered predictable pharmacodynamic effects of sympathomimetic therapy rather than evidence of cardiovascular toxicity. [11] Nevertheless, the magnitude of these effects varies substantially between

individuals and may be influenced by factors such as baseline cardiovascular status, medication dose, treatment duration, and individual pharmacokinetic characteristics. [12]

Hennissen et al. conducted a systematic review and meta-analysis evaluating the cardiovascular effects of stimulant and non-stimulant ADHD medications in children and adolescents. Eighteen clinical trials including 5837 participants were analyzed, with a mean treatment duration of 28.7 weeks. The study demonstrated that methylphenidate, amphetamine derivatives, and atomoxetine were all associated with small but statistically significant increases in systolic blood pressure (SBP). The magnitude of SBP elevation was significant for methylphenidate (SMD 0.25), amphetamines (SMD 0.09), and atomoxetine (SMD 0.16), with no significant differences observed between medications in direct comparisons. Regarding diastolic blood pressure (DBP), significant increases were observed with amphetamine and atomoxetine treatment, whereas methylphenidate did not demonstrate a statistically significant effect. The authors concluded that cardiovascular effects are an inherent characteristic of these medications due to their sympathomimetic properties and increased noradrenergic and dopaminergic neurotransmission. However, the observed changes were generally modest, with average increases of blood pressure not exceeding approximately 5 mmHg at the population level. [13]

The findings reported by Hennissen et al. were subsequently supported by the systematic review and meta-analysis conducted by Liang et al. [12], which included both paediatric and adult populations. In contrast to the previous review, Liang et al. compared methylphenidate and atomoxetine with placebo and directly evaluated differences between the two medications. The authors confirmed that treatment with methylphenidate was associated with significant increases in systolic blood pressure and heart rate compared with placebo across both children and adults. Furthermore, children and adolescents receiving atomoxetine experienced significantly greater increases in systolic blood pressure and heart rate than those treated with methylphenidate, suggesting that atomoxetine may exert a more pronounced haemodynamic effect despite being classified as a non-stimulant medication. Importantly, despite these statistically significant changes in cardiovascular parameters, the analysis found no increase in the incidence of serious adverse cardiac events in patients treated with either methylphenidate or atomoxetine. The authors therefore concluded that although both medications produce measurable increases in blood pressure and heart rate, these changes are generally modest and should be interpreted in the context of individual cardiovascular risk rather than regarded as evidence of clinically significant cardiotoxicity. [12]

Taken together, the available meta-analytic evidence consistently demonstrates that ADHD medications are associated with statistically detectable but clinically minor changes in systolic blood pressure. While atomoxetine appears to produce slightly greater haemodynamic changes than methylphenidate in paediatric populations, current evidence does not indicate an increased risk of serious adverse cardiovascular events in otherwise healthy individuals.

Although the meta-analysis demonstrated statistically significant changes in cardiovascular parameters, the clinical interpretation of these findings requires caution. [12; 13]

Small average increases in blood pressure at the population level do not necessarily translate into clinically meaningful hypertension in individual patients. [12] Nevertheless, ADHD pharmacotherapy is frequently initiated during childhood and may continue for many years, raising questions regarding the potential impact of chronic exposure to even modest increases in blood pressure. [14; 11] Furthermore, mean values may underestimate individual variability, as a subgroup of patients may experience more pronounced haemodynamic responses. [13]

Methylphenidate remains the most extensively studied stimulant medication for the treatment of ADHD. [14] Across clinical trials, its effect on blood pressure appears to be relatively consistent, with most studies reporting modest increases in systolic blood pressure and smaller or non-significant changes in diastolic blood pressure. [13; 12] The magnitude of these cardiovascular effects may be influenced by factors such as medication dose and treatment duration, both of which have been identified as significant moderators of blood pressure changes in meta-regression analyses. [12] Nevertheless, in otherwise healthy individuals, these haemodynamic changes are generally mild and rarely necessitate discontinuation of therapy, although regular cardiovascular monitoring remains recommended throughout treatment. [14; 12]

Amphetamine-based medications, including dexamphetamine and lisdexamfetamine, increase synaptic catecholamine concentrations by promoting the release of dopamine and norepinephrine from presynaptic neurons, thereby enhancing sympathetic nervous system activity. [11] Compared with methylphenidate, amphetamine derivatives possess a greater pharmacological capacity to increase catecholaminergic neurotransmission; however, comparative clinical evidence has not consistently demonstrated clinically

meaningful differences in cardiovascular safety between stimulant classes. [15] Clinical studies indicate that amphetamine treatment is associated with modest increases in blood pressure, supporting current guideline recommendations for routine cardiovascular monitoring, particularly in individuals with pre-existing cardiovascular risk factors. [13; 14]

Although atomoxetine is classified as a non-stimulant medication, its mechanism of action involves selective inhibition of the norepinephrine transporter, resulting in increased noradrenergic signalling. [11] Therefore, cardiovascular effects similar to those observed with stimulant medications may occur, including modest increases in blood pressure and heart rate [13; 12] Liang et al. (2018) reported that atomoxetine was associated with significant increases in systolic blood pressure and heart rate, with greater changes compared with methylphenidate in paediatric populations. Interindividual variability in cardiovascular response may be partly explained by differences in metabolism, particularly CYP2D6 activity, as poor metabolizers may achieve higher plasma concentrations of atomoxetine and potentially experience stronger pharmacological effects. [16; 11]

A major clinical question is whether medication-related increases in blood pressure translate into increased cardiovascular morbidity. Current evidence does not demonstrate a substantial increase in serious cardiovascular events among children, adolescents, or adults receiving ADHD medication. [17; 18; 12] Large observational studies and meta-analyses suggest that although measurable haemodynamic changes occur, clinically significant cardiovascular complications remain uncommon. [13; 12] Therefore, treatment decisions should not be based solely on the possibility of mild blood pressure elevation but should incorporate individual risk factors, including personal cardiovascular history, family history of sudden cardiac death, and the presence of baseline hypertension. [14]

Current NICE guidelines recommend measuring baseline blood pressure and pulse, together with a cardiovascular assessment, before initiating stimulant or non-stimulant ADHD medication to identify patients at increased cardiovascular risk. Because ADHD medications may affect cardiovascular function, NICE recommends monitoring blood pressure and heart rate before and after each dose adjustment and at least every six months during maintenance treatment. NICE further recommends dose reduction and referral to a specialist if sustained systolic blood pressure above the 95th percentile, or a clinically significant increase in blood pressure, is documented on two separate occasions during treatment. These recommendations reflect the balance between maintaining the substantial clinical benefits of ADHD pharmacotherapy and minimizing potential cardiovascular risks. Importantly, routine monitoring does not imply that ADHD medications are unsafe but rather acknowledges predictable pharmacological effects that can be identified and managed through appropriate follow-up. [14]

In contrast to stimulant medications and atomoxetine, α_2-adrenergic receptor agonists, particularly guanfacine, demonstrate an opposite haemodynamic profile. By reducing central sympathetic outflow, guanfacine is associated with modest reductions in systolic and diastolic blood pressure as well as heart rate. These effects are generally mild but may increase the risk of hypotension or bradycardia in susceptible individuals. Consequently, blood pressure monitoring remains important not only for detecting hypertension during stimulant therapy but also for identifying excessive hypotension in patients receiving α_2-adrenergic agonists. [15; 14]

IV. Discussion

The cardiovascular effects of medications used in the treatment of attention-deficit/hyperactivity disorder (ADHD) remain an important subject of clinical discussion, particularly in relation to changes in arterial blood pressure and the potential risk of cardiac arrhythmias. The available evidence suggests that stimulant medications, including methylphenidate and amphetamine derivatives, may produce modest increases in heart rate and systolic and diastolic blood pressure, most likely as a consequence of increased central and peripheral noradrenergic activity. Although these changes are generally small and clinically well tolerated in otherwise healthy individuals, they may be of greater significance in patients with pre-existing hypertension, cardiovascular disease, structural heart abnormalities, or an increased susceptibility to cardiac rhythm disturbances. Non-stimulant medications may demonstrate different cardiovascular profiles. Atomoxetine may also increase heart rate and blood pressure due to inhibition of norepinephrine reuptake, whereas α2-adrenergic agonists, such as clonidine and guanfacine, may reduce sympathetic activity and consequently lower blood pressure and heart rate. The potential occurrence of arrhythmias remains a clinically relevant concern, although current evidence does not consistently demonstrate a substantial increase in serious cardiovascular events among appropriately selected patients receiving ADHD pharmacotherapy. Nevertheless,

individual variability in treatment response, the presence of cardiovascular comorbidities, drug interactions, and differences in dose and duration of therapy should be taken into consideration. Therefore, treatment decisions should be individualized, with appropriate assessment of cardiovascular history and risk factors before initiating therapy and regular monitoring of blood pressure and heart rate during treatment. Further long-term studies are needed to better define the cardiovascular safety profiles of individual ADHD medications and to identify patient subgroups who may be particularly vulnerable to clinically significant hypertension, hypotension, or cardiac arrhythmias.

V. Conclusions

Medications used in the treatment of attention-deficit/hyperactivity disorder (ADHD) may influence cardiovascular function, particularly arterial blood pressure, heart rate, and cardiac rhythm. Stimulant medications, including methylphenidate and amphetamine derivatives, are generally associated with modest increases in blood pressure and heart rate, while non-stimulant medications may have variable cardiovascular effects depending on their pharmacological mechanism. Atomoxetine may similarly increase blood pressure and heart rate, whereas α2-adrenergic agonists, such as clonidine and guanfacine, may reduce sympathetic activity and contribute to lower blood pressure. Although serious cardiovascular complications and clinically significant arrhythmias appear to be uncommon in appropriately selected patients, individual cardiovascular risk factors should be carefully considered before and during treatment. Baseline assessment and regular monitoring of blood pressure, heart rate, and relevant cardiovascular symptoms are important components of safe ADHD pharmacotherapy. Further long-term research is needed to clarify the cardiovascular consequences of prolonged treatment and to identify patients who may be particularly susceptible to hypertension, hypotension, or cardiac rhythm disturbances.

Conflicts of interest: No conflicts of interest to declare.

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