



Review Paper

Heart Rate Variability as a method for assessing the risk of metabolic disorders

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ARTICLE INFO

Article history

Received: May 13, 2026

Accepted: September 12, 2026

Available online: October 6, 2026

Keywords

Heart Rate Variability

Autonomic nervous system

Autonomic dysfunction

Metabolic disorders

Cardiometabolic risk

Doi

<https://doi.org/10.31648/paom/236720>

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ABSTRACT

Introduction: Heart Rate Variability (HRV) is a non-invasive method for assessing autonomic nervous system function that plays an important role in physiological regulation and may provide complementary information on autonomic alterations associated with cardiometabolic disorders.

Aim: To summarize current scientific evidence on the role of HRV alterations in major metabolic disorders, and also to identify common autonomic patterns across different stages of cardiometabolic disease progression and evaluate the potential role of HRV as an early marker of cardiometabolic risk.

Material and methods: A narrative review of clinical studies on HRV in patients with cardiometabolic disorders (obesity, dyslipidemia, hypertension, type 2 diabetes mellitus (T2DM)) was performed. Literature was searched in PubMed, Scopus, Web of Science, and Google Scholar using relevant keywords, mainly covering the last five years.

Results and discussion: Metabolic disorders are characterized by autonomic imbalance with a predominance of sympathetic activity and reduced parasympathetic tone. This is reflected by decreased SDNN, RMSSD, pNN50, and HF, along with increased LF and LF/HF ratio. These changes have been reported at early subclinical stages and are associated with blood pressure, glycemia, insulin resistance, lipid profile, and anthropometric parameters. Comorbid conditions are associated with more pronounced HRV reduction and progressive autonomic dysregulation.

Conclusions: The findings of this review suggest that HRV alterations may represent a potential marker of metabolic disorders and a promising adjunctive tool for cardiometabolic risk stratification, early diagnosis, and monitoring of metabolically associated diseases.

1. INTRODUCTION

HRV is a valuable non-invasive method reflecting physiological fluctuations in the intervals between successive heartbeats and providing an indirect assessment of autonomic nervous system (ANS) function, as well as its capacity to respond to internal and external stimuli.^{1,2} HRV parameters are influenced by multiple factors, including age, sex, circadian rhythms, genetic determinants, physiological status, physical activity level, body mass, smoking, alcohol consumption, dietary habits, chronic stress, as well as environmental factors such as noise exposure, temperature variability, and hypoxia. Importantly, a wide range of pathological conditions significantly disrupt the balance between sympathetic and parasympathetic branches of the ANS. Inflammatory and infectious diseases, cardiovascular (CVD) and metabolic disorders, oncological diseases, chronic pulmonary and renal pathology, psychiatric conditions, rheumatoid arthritis, sleep disturbances, and previous stroke are all associated with alterations in HRV mediated by chronic inflammation and neurohumoral dysregulation.³

There is increasing scientific interest in HRV as a clinical and research method for monitoring health status, potentially supporting the early identification of cardiometabolic alterations, and assessment of accelerated aging. Efferent vagal signaling plays a key role in modulating the inflammatory response through inhibition of cytokine release by macrophages. During aging and age-related diseases, a maladaptive shift toward sympathetic overactivity occurs, accompanied by reduced parasympathetic (vagal) tone, promoting a pro-inflammatory state. Reduced vagal activity, which is closely linked to insulin resistance, is associated with impaired regulation of insulin secretion and tissue sensitivity. In combination with sympathetic predominance, this contributes to increased peripheral vascular resistance, arterial hypertension, and dyslipidemia. These processes result in disruption of neuro-immune-endocrine homeostasis, contributing to cardiac autonomic neuropathy, silent myocardial ischemia, myocardial dysfunction, and increased cardiovascular risk, all of which are reflected in altered HRV parameters. Therefore, autonomic imbalance characterized by reduced parasympathetic and increased sympathetic activity represents a key pathogenetic mechanism of metabolic disorders, while HRV analysis is used as a relevant marker of metabolic and cardiovascular alterations across different stages of life.^{4,5}

2. AIM

The aim of this study was to summarize and critically analyze current scientific evidence regarding alterations in HRV in major metabolic disorders. In addition, the review sought to identify common patterns of autonomic dysfunction across different stages of cardiometabolic disease progression and to evaluate the potential utility of HRV as a non-invasive biomarker and promising adjunctive tool for

the early identification, risk stratification, and monitoring of cardiometabolic disorders.

3. MATERIAL AND METHODS

A narrative review of publications focusing on HRV parameters and their alterations in patients with obesity, dyslipidemia, arterial hypertension, and T2DM, as well as their combined forms, was conducted. The literature search was performed using the keywords: “heart rate variability”, “HRV”, “metabolic disorders”, “cardiometabolic risk”, “hypertension”, “type 2 diabetes mellitus”, “obesity”, “overweight”, “dyslipidemia”, and “autonomic dysfunction” in electronic databases, including PubMed, Scopus, Web of Science, and Google Scholar, with a primary focus on publications from the last five years. In addition, the American Heart Association (AHA) Scientific Statement on cardiovascular-kidney-metabolic syndrome and the European Atherosclerosis Society (EAS) consensus statement on the clinical staging of metabolic disorders were analyzed.

The search initially identified 488 records. After removal of duplicates, 406 publications remained for title and abstract

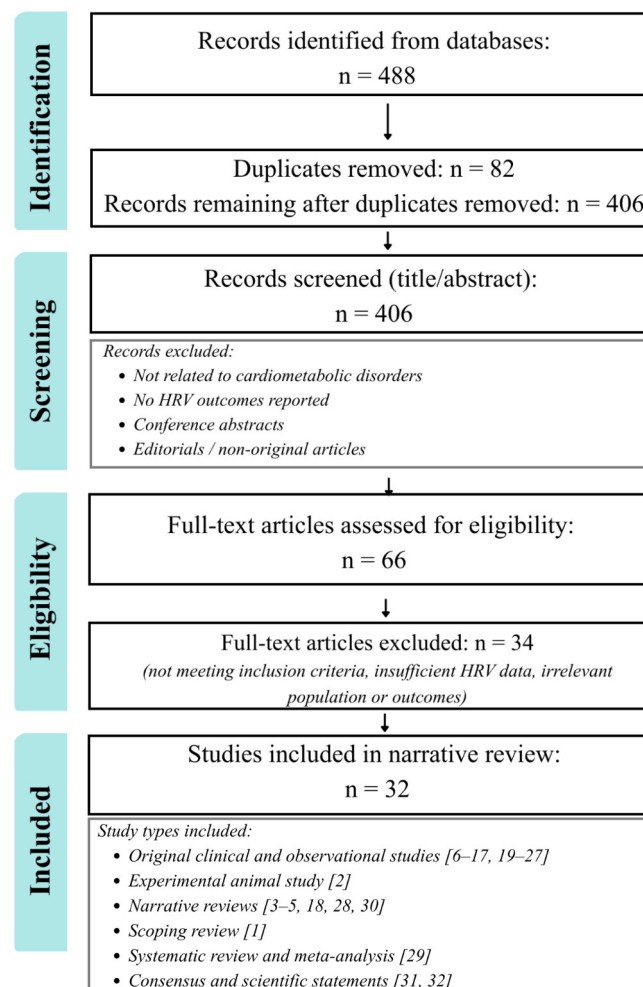


Figure 1. A flowchart of the study selection process.

screening. Studies unrelated to cardiometabolic disorders, articles not reporting HRV outcomes, conference abstracts, and editorials were excluded. Following title and abstract screening, 66 potentially relevant articles were selected for full-text assessment. Of these, 32 publications met the pre-defined eligibility criteria and were included in the final narrative review. These studies comprised original clinical and observational studies, an experimental animal study, narrative reviews, a scoping review, a systematic review and meta-analysis, an EAS consensus statement, and an AHA scientific statement addressing autonomic dysfunction and HRV alterations in cardiometabolic diseases.

4. RESULTS AND DISCUSSION

Autonomic dysfunction is a broad concept encompassing impaired ANS function and may occur at different stages of life. In early life, it represents genetically determined forms, whereas in adulthood it reflects acquired manifestations associated with neurodegenerative diseases, metabolic disorders, and CVD. In the context of aging, dysautonomia is increasingly viewed as a major contributor to age-related disease development, driven by persistent autonomic imbalance.⁴

HRV in overweight and obesity

Emerging evidence suggests that adiposity-related metabolic alterations are closely associated with autonomic nervous system imbalance, as reflected by changes in HRV parameters.^{5–10} According to Strüven A. et al., individuals who are overweight demonstrated increased sympathetic activity and reduced RMSSD and LF, which correlated not only with visceral fat percentage but also with the waist-to-hip ratio (WHR).⁵ Autonomic imbalance characterized by sympathetic predominance in pre-obese and obese individuals was further confirmed by Surana Gandhi N. et al. The authors reported that men with BMI ≥ 25 kg/m², waist circumference ≥ 90 cm, and WHR ≥ 0.9 exhibited significantly higher LF and LF/HF values ($p < 0.05$). These alterations were attributed to chronic low-grade inflammation in obesity, the influence of adipocyte-derived vasoactive hormones, impaired baroreflex function, and dysregulation of adipokine secretion.⁶ Studies conducted in women who were overweight or obese also showed frequency-domain alterations, including a significant increase in LF and a decrease in HF ($p < 0.05$), as well as correlations between lipid accumulation index, WHR, and LF/HF ratio, reflecting increased sympathetic and reduced parasympathetic activity.^{7,8} Muthukrishnan S. et al. further confirmed that young individuals with overweight and obesity also exhibited time-domain HRV alterations, including reduced SDNN, RMSSD, and pNN50% ($p < 0.05$).⁹ Moreover, reductions in HF, SDNN, RMSSD, and pNN50% were more pronounced in individuals with obesity and concomitant metabolic disturbances aged 40–49 years.¹⁰

An increased proportion of adipose tissue, particularly visceral and central obesity, is associated with autonomic

imbalance characterized by sympathetic predominance and reduced parasympathetic activity. This is reflected by decreased time-domain (SDNN, RMSSD, pNN50) and frequency-domain (HF) parameters, along with increased LF and LF/HF ratio. These changes are observed in both sexes and appear to become more pronounced with age, highlighting the role of anthropometric parameters in the development of autonomic dysfunction.

HRV in dyslipidemia

Alterations in autonomic regulation associated with lipid metabolism disorders have been increasingly documented in recent studies. Nussinovitch U. reported that the presence of dyslipidemia was associated with reduced RMSSD, SDNN, and pNN50 ($p < 0.05$).¹¹ Wiley C.R. et al. identified the lipid accumulation index, derived from waist circumference and triglyceride levels, as a strong predictor of cardiometabolic disorders, demonstrating significant correlations with HRV parameters.¹² Findings by Zeid S. et al. indicate that lipid profile abnormalities are associated with increased sympathetic nervous system activity and are reflected in HRV alterations. In particular, patients with dyslipidemia exhibited reduced time-domain HRV parameters (SDNN, SDANN, RMSSD, pNN50) as well as decreased frequency-domain components (LF, HF), accompanied by an increased LF/HF ratio ($p < 0.05$).¹³ Similar observations were reported by Plaza-Florido A. et al., who demonstrated that in young women, LF/HF alterations were associated with high-density lipoprotein cholesterol levels.¹⁴ In a subsequent study, de Almeida L.V. et al. showed that these associations between HRV parameters and lipid profile components were independent of sex. The authors reported a negative correlation between total cholesterol and triglyceride levels and time-domain HRV indices (SDNN, RMSSD), along with a positive correlation with the LF component, reflecting sympathetic predominance and autonomic imbalance.¹⁵ Kumar R. et al. demonstrated that these HRV alterations were associated not only with dyslipidemia but also with the progression of atherosclerotic changes, suggesting the potential role of HRV as an adjunctive measure for cardiometabolic and cardiovascular risk assessment.¹⁶

Dyslipidemia is characterized by a shift in autonomic balance toward sympathetic predominance. These changes correlate with triglyceride and cholesterol levels as well as the lipid accumulation index and are observed across both sexes. Overall, HRV alterations in patients with dyslipidemia and atherosclerosis suggest that HRV may be a promising adjunctive tool for cardiometabolic risk assessment.

Changes in HRV parameters in patients with arterial hypertension

There is a complex relationship between the development of hypertension and autonomic dysfunction, which is characterized by increased sympathetic activity leading to elevated heart rate (HR), enhanced myocardial contractility, and peripheral vasoconstriction, as well as reduced parasympathetic tone, as evidenced by decreased HRV parameters. Recent

studies suggest that patients with hypertension demonstrate a statistically significant reduction in SDNN and RMSSD, as well as a negative correlation between these parameters and systolic (SBP) and diastolic blood pressure (DBP). Notably, alterations in time-domain HRV parameters are already detectable at the subclinical stage of hypertension.¹⁷

Changes in HRV parameters at early stages of hypertension are also confirmed by other studies.^{7,8} Sri-Amad et al. observed that a shift toward sympathetic predominance is present in young individuals with a tendency toward elevated blood pressure. Patients with lower RMSSD values had a 1.36-fold higher risk of developing hypertension within the next 9 years.¹⁸ In another study evaluating HRV parameters in an older population, individuals with high-normal blood pressure demonstrated reduced SDNN and pNN50 ($p < 0.05$). In patients with hypertension, a significant reduction in SDNN and lower LF and HF power was observed ($p < 0.01$), while no changes in the LF/HF ratio were detected.¹⁹

The study by Julario et al. demonstrated that, in patients with elevated blood pressure, reductions in SDNN and pNN50 were accompanied by alterations in additional indices of vagal function, including SDANN and NN50, as well as changes in the sympatho-vagal balance (LF/HF), particularly in individuals with uncontrolled hypertension ($p < 0.05$).²⁰ However, data regarding frequency-domain parameters remain inconsistent. According to Maciorowska et al., in young and middle-aged patients with grade 1 hypertension, no association was found between HF, LF, or LF/HF and blood pressure, whereas correlations were observed between SBP/DBP and indices of vagal control (SDNN, RMSSD), DBP, and pNN50. These parameters positively correlated with echocardiographic measures of left ventricular diastolic function (E/A ratio and e'), while total power (TP) was correlated with systolic function (left ventricular ejection fraction). Despite conflicting findings, the significant role of autonomic dysfunction in both the development of hypertension and subclinical myocardial dysfunction should be emphasized.²¹

It should be noted that the relationship between hypertension and ANS dysfunction is bidirectional, as autonomic imbalance contributes to the development of hypertension, while sustained elevation of blood pressure further exacerbates autonomic dysregulation. Reduced parasympathetic tone with increased sympathetic activity in patients with hypertension was confirmed in the study by He B. et al. The authors demonstrated that decreased SDNN and RMSSD correlated not only with blood pressure levels but also with pro-inflammatory biomarkers, which are factors of CVD progression.¹⁷

Thus, current evidence indicates that autonomic dysfunction is one of the key mechanisms underlying the development and progression of hypertension. The imbalance characterized by sympathetic predominance and reduced parasympathetic tone is detectable even at preclinical stages and is associated with decreased SDNN, RMSSD, pNN50, and SDANN. The bidirectional relationship between hypertension and ANS dysfunction results in persistent sympathetic overactivity, promoting cardiovascular damage and suggesting that HRV assessment may have potential as an adjunctive

approach for identifying early autonomic alterations associated with cardiovascular disease development.

HRV in patients with type 2 diabetes mellitus

Within the Tromsø Study, changes in SDNN were investigated using plethysmography in patients with metabolic disorders, T2DM, cardiovascular diseases, and albuminuria. The authors found a correlation between HRV parameters (SDNN, RMSSD) and components of metabolic disorders, including the presence of hypertension, obesity, and dyslipidemia. HRV indices progressively decreased with increasing severity of metabolic disturbances, which was most pronounced in the group with T2DM, where a significant reduction in SDNN was observed in association with increasing glycated hemoglobin (HbA1c) levels ($p < 0.05$).²² The impact of T2DM on HRV parameters is also supported by other studies. Wang K et al. investigated the effect of hyperglycemia on HRV changes. Changes in mean heart rate were associated with fasting plasma glucose, insulin levels, and HOMA-IR, with these findings being more pronounced in young individuals with newly diagnosed T2DM. However, reductions in SDNN and RMSSD, when adjusted for heart rate, were associated only with fasting glucose levels.²³ Current research is focused on the influence of not only time-domain but also frequency-domain HRV parameters (HF, LF, LF/HF) in patients with T2DM, particularly under conditions of chronic stress.^{24–26} Short-term HRV analysis in patients with T2DM demonstrated a statistically significant increase in heart rate, a global reduction in HRV (manifested by decreased RMSSD and pNN50), and suppression of parasympathetic activity (reduced HF, increased LF, and elevated LF/HF ratio) ($p < 0.05$).²⁴ The adverse impact of chronic stress on glucose metabolism and HRV parameters was also confirmed in the study by Pisaruk A.V. et al. The authors demonstrated a significant reduction in total power (TP) and markers of parasympathetic activity (RMSSD, pNN50, HF), which was associated with higher fasting glucose levels, oral glucose tolerance test results, elevated insulin levels, and increased HOMA-IR. At the same time, an increase in biological age compared to the pre-war period by 10.6 years was observed ($p < 0.05$).²⁵

The analyzed data indicate a relationship between type 2 diabetes mellitus and alterations in HRV parameters. In particular, impaired glycemic control is associated with reductions in SDNN and RMSSD, disturbances in frequency-domain parameters (HF, LF, LF/HF), and enhanced sympathetic activity accompanied by reduced parasympathetic tone. These changes are associated with poor glycemic control. In addition, chronic stress potentiates these alterations, contributing to further HRV reduction and being associated with worsening metabolic control, increased insulin resistance, and accelerated biological aging.

HRV in patients with combined arterial hypertension and type 2 diabetes mellitus

In a study examining HRV alterations in patients with arterial hypertension and T2DM, Solanki et al. demonstrated

that these changes were more pronounced in individuals with suboptimal glycemic and blood pressure control. Regardless of the presence of concomitant hypertension, patients with T2DM demonstrated abnormally elevated LF/HF values compared with the hypertension-only group ($p < 0.05$), indicating a more detrimental effect of hyperglycemia on HRV parameters. In addition, alterations in autonomic regulation, particularly a shift toward sympathetic predominance, were already evident at the prediabetic stage.²⁶ Previous findings by Bassi D. et al. also confirmed HRV alterations in the comorbid course of arterial hypertension and T2DM, demonstrating more pronounced changes in RMSSD, LF, HF, and total power (TP) ($p < 0.05$).²⁷

Patients with combined arterial hypertension and T2DM demonstrate characteristic changes in HRV, including an increased LF/HF ratio and reduced RMSSD, HF, and TP. These abnormalities are further exacerbated by poor glycemic and blood pressure control and may already be evident at the prediabetic stage. The combined course of hypertension and T2DM is characterized by progressive autonomic dysregulation and an increased cardiometabolic risk.

The main findings of the reviewed studies are summarized in Table 1, which provides an overview of HRV alterations in cardiometabolic disorders, including obesity, dyslipidemia, arterial hypertension, T2DM, and their comorbid conditions. The table outlines study populations and the most relevant changes in time- and frequency-domain HRV parameters, indicating an overall pattern of autonomic imbalance characterized by reduced parasympathetic activity and sympathetic predominance across the conditions examined.

Factors limiting HRV interpretation

Although HRV is an important non-invasive marker for assessing autonomic regulatory mechanisms in the human body, this method has several important limitations that reduce its specificity as an indicator of autonomic function. HRV parameters are significantly influenced by both external and internal factors, including age, sex, level of physical activity, sleep quality, psycho-emotional status, and environmental conditions. In particular, older age, low physical activity, poor sleep quality, and chronic psychological stress are associated with a reduction in overall HRV, which is reflected by decreased SDNN and RMSSD, reduced indices of parasympathetic activity (HF), and a relative increase in sympathetic influence (LF and LF/HF ratio).²⁸

Secondly, pharmacological interventions, particularly β -blockers, generally enhance vagal tone and lead to increases in RMSSD, SDNN, and HF, along with a reduction in heart rate, whereas antidepressants (especially tricyclic antidepressants) and antipsychotics are more often associated with decreased HRV, reduced parasympathetic activity, and a relative shift toward sympathetic predominance.²⁹

Thirdly, methodological limitations exist due to the lack of full standardization of ECG recording protocols, recording duration, body position, and analytical approaches, resulting in considerable heterogeneity across studies. Moreover, current evidence challenges the simplified interpretation of

spectral HRV components (LF and HF) as direct markers of sympathetic and parasympathetic activity.³⁰

Thus, HRV should be regarded as a sensitive but non-specific integrative marker of autonomic regulation that requires careful consideration of clinical, behavioral, and methodological factors during interpretation.

Study limitations and future perspectives

Further work is required to clarify and validate the potential clinical utility of HRV across different stages of cardiometabolic disease progression. This remains an important area for future research, as several questions are still unresolved regarding the role of autonomic dysfunction in early metabolic remodeling.

According to the most recent consensus of the EAS, a step-wise model of cardiometabolic disease development has been introduced, replacing the traditional concept of metabolic syndrome. This framework distinguishes three stages: early subclinical metabolic disturbances (including insulin resistance/prediabetes, overweight/obesity, isolated hepatic steatosis, arterial hypertension, and/or atherogenic dyslipidemia); early target organ damage with overt clinical conditions (T2DM, asymptomatic diastolic dysfunction, metabolic dysfunction-associated steatohepatitis or hepatic fibrosis, albuminuria/chronic kidney disease stages 1–2, and subclinical atherosclerosis); and cardiometabolic complications.^{31,32}

While existing studies have separately evaluated HRV alterations across individual components of metabolic disorders, there is currently a lack of evidence integrating HRV assessment within this stage-based cardiometabolic framework. More broadly, further research is needed to determine whether HRV parameters may serve as sensitive markers of changes associated with transitions between different stages of cardiometabolic disease progression. This line of research extends current knowledge of autonomic regulation in metabolic disorders by highlighting the potential role of HRV as an early indicator of systemic metabolic dysregulation. The findings of this study may inform future research, particularly on cardiometabolic risk stratification and the development of early identification strategies based on autonomic biomarkers. Future studies should focus on determining whether HRV parameters can be validated as stage-specific biomarkers of cardiometabolic disease and on evaluating their potential prognostic value in longitudinal clinical settings. In this context, it would be of interest to evaluate the impact of incorporating HRV monitoring into risk prediction models for early metabolic dysfunction.

Overall, the analyzed literature suggests an association between HRV alterations and cardiometabolic risk across different disease stages. However, despite this evidence, several key questions remain unresolved. In particular, it is still unclear at what time point and within which age ranges HRV impairment first emerges across different stages of cardiometabolic disease progression. Further research is needed to elucidate age-dependent patterns of autonomic dysfunction and to determine whether early HRV changes precede or parallel structural and functional cardiovascular remodeling.

Table 1. Summary of studies on HRV alterations in cardiometabolic disorders.

Authors	Year	Study design	Population	HRV findings
Strüven et al. ⁵	2021	Narrative review	Overweight/obesity (review of clinical and interventional studies)	↓SDNN, RMSSD, LF
Surana Gandhi et al. ⁶	2024	Cross-sectional observational study	132 overweight adults	↑LF, LF/HF; sympathetic predominance
Al-Khassaki & Al-Galani ⁷	2024	Cross-sectional observational study	53 healthy females with varying body fat	↑LF, HF in higher adiposity groups
Chang et al. ⁸	2021	Observational study	92 females with obesity	↓HF, SDNN, RMSSD, and pNN50%, obesity-related autonomic imbalance
Muthukrishnan et al. ⁹	2025	Observational study	157 adults with overweight/obesity	↓SDNN, RMSSD, pNN50
Rastović et al. ¹⁰	2019	Observational study	99 adults with obesity	↓SDNN, RMSSD, pNN50, HF, TP
Nussinovitch ¹¹	2020	Observational study	59 adults with dyslipidemia	↓RMSSD, SDNN, pNN50
Wiley et al. ¹²	2025	Observational study	227 adults with overweight/obesity	Lipid accumulation strongly correlated with HRV and inflammation
Zeid et al. ¹³	2024	Observational study	3289 high-risk CVD participants	↓SDNN, RMSSD, LF & HF; LF/HF ratio
Plaza-Florido et al. ¹⁴	2022	Cross-sectional observational study	132 young adults with cardiometabolic risk factors	↓SDNN, RMSSD
de Almeida et al. ¹⁵	2025	Observational study	73 individuals with metabolic risk	↓SDNN, RMSSD, HF, TP, LF, LF/HF
Kumar et al. ¹⁶	2024	Observational study	47 progressive atherosclerosis patients	↓SDNN, RMSSD, HF, LF
He et al. ¹⁷	2024	Clinical observational study	50 patients with hypertension	↓SDNN, RMSSD
Yugar et al. ¹⁸	2023	Narrative review	Hypertension patients (narrative review)	↓SDNN, RMSSD, HF, LF/HF
Sri-Amad et al. ¹⁹	2024	Observational study	Older adults with hypertension (HT, n = 21), prehypertension (n = 33), and normotension (n = 10); total n = 64	↓SDNN, pNN50, LF power, and HF power
Julario et al. ²⁰	2021	Observational study	52 patients with hypertension	↓SDNN, RMSSD, pNN50, LF, HF
Maciorowska et al. ²¹	2022	Observational study	144 patients with primary hypertension	↓SDNN, RMSSD, pNN50, TP, LF/HF
Azulay et al. ²²	2022	Prospective cohort study	Tromsø Study (n = 7,880 adults with metabolic syndrome)	↓SDNN, RMSSD
Wang et al. ²³	2023	Prospective cohort study	Rotterdam Study (n = 7,630; 871 patients developed T2DM in 8.6 years)	↓SDNN, RMSSD
Serhiyenko et al. ²⁴	2025	Clinical observational study	32 T2DM patients	↓RMSSD, pNN50, HF, TP, HR, LF
Pisaruk et al. ²⁵	2024	Comparative observational study	Women exposed to chronic war-related stress (n = 91) vs pre-war controls (n = 79)	↓SDNN, RMSSD
Solanki et al. ²⁶	2023	Comparative observational study	203 participants (diabetics, hypertensives, diabetic-hypertensives and normotensive-nondiabetics)	↓SDNN, RMSSD, HF, LF/HF
Bassi et al. ²⁷	2018	Comparative observational study	60 patients (32 T2DM vs 28 T2DM + arterial hypertension patients)	↓RMSSD, HF, TP, LF

5. CONCLUSIONS

Based on current evidence, it can be concluded that HRV represents a sensitive, non-invasive measure of autonomic nervous system function and may serve as a potential marker of cardiometabolic disturbances. Metabolic disorders are characterized by autonomic imbalance with a shift toward sympathetic predominance, which is reflected by reduced HRV parameters (SDNN, RMSSD, pNN50, HF) and increased LF and LF/HF ratio. These alterations are detectable even at early subclinical stages, correlate with established cardiovascular risk factors, and are further accentuated by aging, chronic stress, and concomitant metabolic disturbances. However, the interpretation of HRV is limited by physiological, pharmacological, and methodological confounders, which should be considered when applying HRV in clinical and research settings. Despite these limitations, HRV may represent a promising adjunctive tool for cardiometabolic risk assessment and the early identification of metabolic alterations, rather than an independent diagnostic or prognostic measure.

Ethics approval

None declared.

Conflict of interest

None declared.

Funding

None declared.

Author contributions

Study design: AT, OK

Data collection: AT

Data interpretation: AT

Manuscript preparation: AT, OK

Literature search: AT

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