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Literature review

Heart rate variability: from recording precautions to applicability to physical exercise

Variabilidade da frequência cardíaca: dos cuidados no registro à aplicabilidade ao exercício físico

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ABSTRACT

Aim: This narrative review sought to address heart rate variability (HRV) based on concepts and definitions, methods of recording and processing signals, signal interpretation, influencing factors, and applicability to physical exercise. **Methods:** Bibliographic survey of works published between 2000 and 2022 in PubMed, Scopus, Web of Science, Scielo databases. **Results:** HRV is a non-invasive method for measuring the action of the autonomic nervous system in the heart and has been used as a marker of physical and mental health. HRV measurements can be performed by recording an electrocardiogram or frequency meter, which allows the extraction of several HRV parameters using linear and non-linear methods. **Conclusion:** HRV is a variable that can be influenced by several factors, and its role can be explained based on four theories. Physical training can be used as a marker of exercise intensity control.

Keywords: autonomic nervous system; heart rate variability; physical training; electrocardiogram.

RESUMO

Objetivo: Esta revisão narrativa buscou abordar a variabilidade da frequência cardíaca (VFC) em relação aos conceitos e definições, formas de registro e processamento do sinal, interpretação do sinal, fatores influenciadores e aplicações ao exercício. **Métodos:** Levantamento bibliográfico de trabalhos publicados entre 2000 e 2022 nas base de dados PubMed, Scopus, Web of Science, Scielo. **Resultados e discussão:** A VFC é um método não invasivo de mensurar a atuação do sistema nervoso autônomo no coração, que vem sendo utilizada como marcador de saúde física e mental. A mensuração da VFC pode ser feita através do registro do eletrocardiograma ou frequencímetro, que permitem a extração de diversos parâmetros da VFC utilizando métodos lineares e não lineares. **Conclusão:** A VFC é uma variável que pode ser influenciada por diversos fatores, e seu papel pode ser explicado com base em quatro teorias. No que se refere ao treinamento físico, pode ser usada como um marcador de controle de intensidade de exercício.

Palavras-chave: sistema nervoso autônomo; variabilidade da frequência cardíaca; treinamento físico; eletrocardiograma.

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Introduction

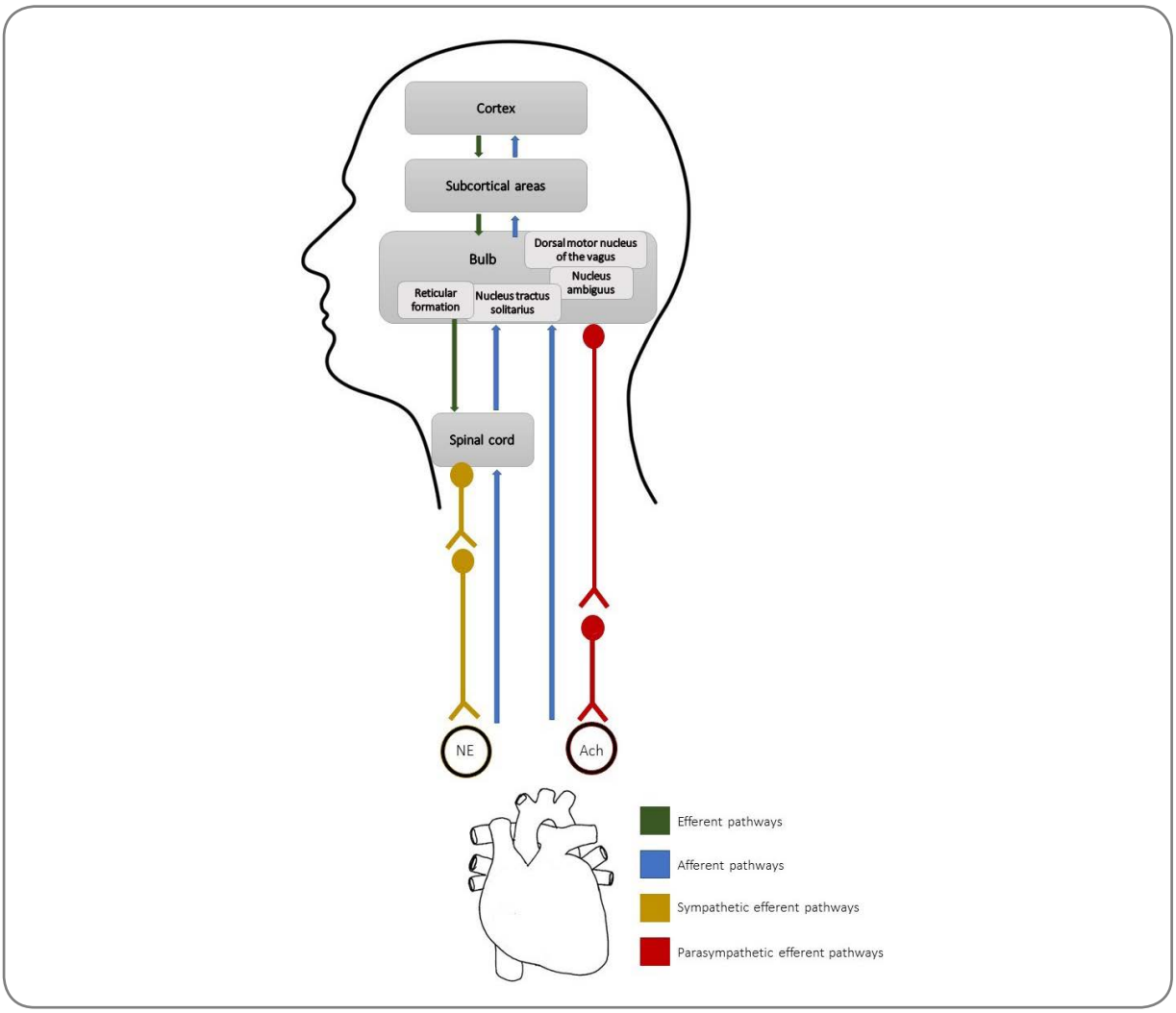
The autonomic nervous system (ANS) modulates the heart by regulating its extrinsic rhythm, thereby influencing the frequency of contractions [1]. Communication between the ANS and heart is dependent on information from mechanoreceptors (cardiopulmonary and baroreceptors) and chemoreceptors (carotid, aortic, and ventricular). Influences derived from the respiratory, vasomotor, renin–angiotensin–aldosterone, and thermoregulatory systems and from higher centers, such as the amygdala and hypothalamus, reach the medullary cardiovascular centers, which modulate sympathetic and parasympathetic (vagal) autonomic activity in the heart [2].

Autonomic pathways are formed by preganglionic and postganglionic fibers. The parasympathetic system is composed of neurons that originate in the medullary nuclei (more specifically, in the dorsal motor nucleus of the vagus and nucleus ambiguus), with preganglionic fibers, which have elongated axons and establish synapses with postganglionic neurons, which have short axons located in the cardiac plexus and reach the cardiac muscle through the sinus and atrioventricular nodes [3]. With the stimulation of the parasympathetic ANS, acetylcholine is released by the preganglionic and postganglionic fibers, reducing the sinus node firing rate, cardiac output, and blood pressure. Generally, this type of stimulation is predominant in situations of rest, safety, and digestion [4].

Sympathetic pathways are formed by fibers originating in the medulla (more specifically, in the reticular formation) with projections to preganglionic fibers located in the thoracic spinal cord that synapse with postganglionic fibers located in the paravertebral ganglia, finally reaching the sinus node and distributing to the atrioventricular node and most of the myocardium. Upon stimulation of the sympathetic ANS, norepinephrine is released, which increases the sinus node firing rate, electrical conduction velocity, excitability, and contraction force in all portions of the heart. Therefore, cardiac output and ejection volume increase, thus increasing the blood pressure [3]. An overview of the anatomy of the sympathetic and parasympathetic pathways is shown in Figure 1.

The signaling mechanisms of the sympathetic and parasympathetic branches, as well as their temporal courses, are distinct. Sympathetic influence on the heart has a slower course of action than parasympathetic influence. Changes in heart rate (HR) resulting from sympathetic activation occur more slowly, with a peak effect observed approximately 4 s after the start of stimulation and a return to baseline occurring approximately 20 s after the cessation of stimulation. In contrast, parasympathetic modulation on the heart has low response latency, with a peak effect within 0.5 s after the start of stimulation and a return to baseline within 1 s after cessation. Thus, the parasympathetic modulation of the heart occurs faster than sympathetic modulation. These differences in the time of action occur because of the different types of intracellular signaling mechanisms underlying different receptors.

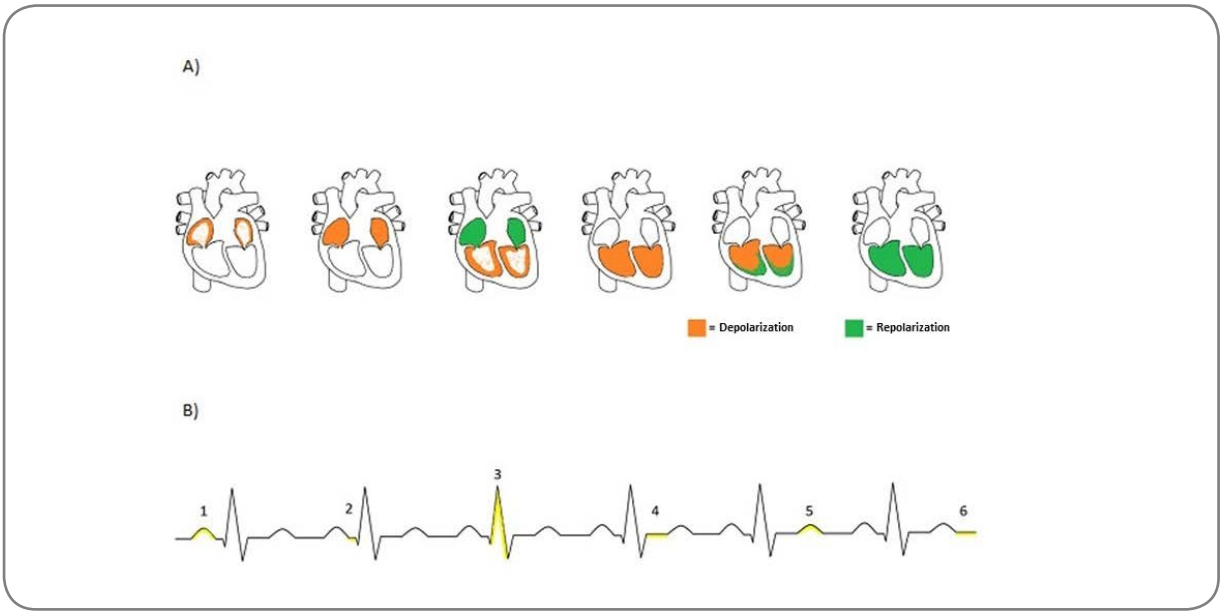
Sympathetic activity occurs through the action of noradrenaline on β1-adrenergic receptors, which generates a cascade of intracellular reactions in multiple stages, culminating in the opening of Na+ and Ca2+ channels and closing of K+ channels. Owing to these stages, sympathetic activity generates slow fluctuations in the heart (not greater than 0.15 Hz in the interval between heartbeats). However, parasympathetic activity occurs through the action of acetylcholine on metabotropic muscarinic receptors (M2), which opens the K+ and Ca2+ channels. Changes are initiated 30–100 ms after binding of acetylcholine to its receptor. Thus, vagal activity can generate high-frequency fluctuations in the interval between heartbeats between 0.15 and 0.4 Hz [5]. Therefore, it is understood that the HR variations are determined by the integration between the slow and fast modulation generated by the sympathetic and parasympathetic branches, respectively [6-8].



NE = norepinephrine; Ach = acetylcholine. Source: author himself
Figure 1 - Communication pathways between the brain and the heart, which are responsible for generating heart rate variability

Stimulation arising from sympathetic and parasympathetic modulation reaches the heart and can be studied by recording the electrocardiograms, represented by waves P, Q, R, S, T, and U (Figure 2A). Cardiac excitation begins with an impulse

generated by autorhythmic cells in the sinus node, which is distributed by the atrial syncytium, resulting in the onset of depolarization of the atria (P wave), followed by the complete depolarization of the atria (Q wave). This impulse quickly reaches the atrioventricular node and is conducted from the atrioventricular node to the ventricles by the atrioventricular bundle and Purkinje fibers, resulting in atrial repolarization and the onset of ventricular depolarization (R wave), followed by complete depolarization of the ventricle (S wave), by the onset by ventricular repolarization (T wave), and finally by ventricular repolarization (U wave) [9]. The demarcation of each of these moments is shown in the electrocardiogram graph (Figure 2B).



Source: author himself

Figure 2 - (A) Cardiac depolarization and repolarization; (B) Electrocardiogram. Where 1 is the beginning of atrial depolarization, 2 is complete atrial depolarization, 3 is the beginning of ventricular depolarization and atrial repolarization, 4 is complete ventricular depolarization, 5 is the beginning of repolarization, and 6 is complete repolarization

The interval between two ventricular depolarizations that generate actual heartbeats is known as the RR interval [10]. From the time intervals between two R waves, in milliseconds, a graph of the intervals between heartbeats over time can be constructed [1], known as a tachogram.

The intervals between R waves on the electrocardiogram are not regular. This variation in the duration of RR intervals is healthy and expected, demonstrating that the heart does not function like a metronome [9]. According to Shaffer et al. [9], the greater the differences between these intervals, the greater the indications of homeostasis, health, and physiological regulation of the individual in the face of environmental demands. Physiological variation measured from the temporal distance between RR intervals is known as heart rate variability (HRV). HRV is a non-invasive and efficient method for assessing the activity of the ANS, that is, the sympathetic and parasympathetic branches, in the heart [11].

After this initial contextualization, the objective of the present study was to provide detailed and updated information about HRV in relation to concepts and definitions; forms of recording, processing, and interpretation of the signal; influencing factors; and applications to reach people who are starting to study the subject.

Methods

As this is a narrative review, the methodology used was based on a bibliographical survey of the subject in question conducted on different research platforms (PubMed, Scopus, Web of Science, and Scielo). The bibliographic survey covered, in almost its entirety, works published between 2000 and 2022. Notably, narrative reviews are broad research methods suitable for describing and discussing the development of a given subject from a theoretical or contextual point of view.

Registration and signal processing

According to the European Society of Cardiology and North American Society of Electrophysiology [11], HRV analysis can be performed using linear and non-linear methods. Linear methods span both time and frequency domains. Frequency-domain analysis, also known as spectral analysis, is capable of decomposing the tachogram power spectrum into various frequency components or modulation rates of the RR intervals (Table I).

The quoted frequency bands can be calculated in absolute units (ms^2) or normalized units (n.u.). In normalized units, the low- (LF) and high-frequency (HF) bands are calculated as a percentage of the total power, disregarding the very-low--frequency (VLF) power:

$$\textbf{HF (u.n.)=HF/(Total power-HRV)} \times 100$$
$$\textbf{LF (u.n.)=LF/(Total power-HRV)} \times 100$$

This normalization minimizes the effects of changes in the VLF range on the two fast frequency ranges (LF and HF), leaving only the purest effects of LF and HF [11].

In the analysis of HRV through the time domain, each normal RR interval is measured, that is, the sinus beats during a certain time interval; then, based on statistical or geometric methods (mean, standard deviation, and derived indices from the histogram or the map of Cartesian coordinates of the RR intervals), the indices that translate fluctuations in the duration of the cardiac cycles are calculated, thus providing several parameters (Chart 1).

The non-linear method has gained visibility in recent years, and perhaps for this reason, the physiological interpretation of its indices is still not well defined. Several studies have indicated possible physiological interpretations: i) Poincaré plot:

it has been proposed that the component standard deviation 1 (SD1) represents the parasympathetic activity, the SD2 represents the sympathetic activity and parasympathetic, while the SD1/SD2 ratio represents the sympathetic and parasympathetic ratio [17]; ii) Detrended Fluctuation Analysis (DFA): the proposal is that the short--term correlations extracted with DFA (α1) reflect the baroreceptor reflex, while the long-term correlations (α2) reflect the regulatory mechanisms that limit the fluctuation of the beating cycle [18]. In addition to these two parameters, the non-linear method comprises different methods for processing HRV. Some of these forms are listed in Table II and are adapted from Ferreira et al. [7].

Among all the non-linear methods mentioned, the Poincaré plot method or the Poincaré plot is highly detailed, as this is the most studied method in the literature. This is the analysis of a scatter plot, where each point is represented on the x-axis as RRn and the y-axis as RRn+1, where RRn is the time between two successive R waves, and RRn+1 is the time between two successive R peaks. When the plot is fitted using the ellipse-fitting method, the analysis provided three indices: the SD of instantaneous beat-to-beat interval variability (SD1), long-term variability of continuous RR intervals (SD2), and SD1/SD2. In the Poincaré plot, SD1 is the width, and SD2 is the length of the ellipse. It is also possible to extract the area of an imaginary ellipse (S) using the length axes (S = SD1 × SD2) [19-21].

A Poincaré plot can be analyzed qualitatively or quantitatively. Qualitatively, the analysis is performed by observing the shape of the cloud of points and, consequently, the ellipse. A graph with a very concentrated cloud of points may be indicative of some pathology; however, high dispersions in the time series may indicate good health [22-24].

For the quantitative analysis of the Poincaré plot, SD1 reflects the instantaneous beat-to-beat variability of HR and represents parasympathetic activity, and SD2 reflects the continuous variability from beat-to-beat and represents the global HRV and sympathetic and parasympathetic activity [22,25].

Interpretation of the HRV signal

There are currently some theories that attempt to explain the evolutionary role of HRV, among which four stand out: i) polyvagal theory [26,27], ii) behavioral biological model [28], iii) neurovisceral integration model [29], and iv) respiratory rate resonance model [30,31].

According to the polyvagal theory developed by Porges in 1995, during the evolution of mammals, anatomical and physiological changes occur in the ANS, which are responsible for influencing HRV in these animals. One of these changes was the myelination of the motor fibers of the vagus nerve originating in the nucleus ambiguus, which provided rapid modulation of the HRV that occurs in contexts where the animal is safe, facilitating affiliative behaviors, care for the offspring, and social approach [26,27].

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Chart 1 - Linear Methods of Heart Rate Variability Analysis

Domain	Parameter	Unit	Definition in english	Definition in portuguese	Physiological interpretation
TIME	Statistical methods				
	SDT RR (SDNN)	ms	Standard deviation of NN intervals	Desvio padrão dos intervalos NN	Sympathetic and parasympathetic activities [11] In short-term recordings, it predominantly reflects parasympathetic activity [9]
	SDNNi	ms	Mean of the standard deviations of all the NN intervals for each 5 min segment of a 24 h HRV recording	Média dos desvios padrão de todos os intervalos NN para cada segmento de 5 min de um registro de 24 h VFC	Sympathetic and parasympathetic activities [11]
	SDANN	ms	Standard deviation of the average NN intervals for each 5 min segment of a 24 h HRV recording	Desvio padrão das médias dos intervalos NN para cada segmento de 5 min de um registro de 24 h VFC	Sympathetic and parasympathetic activities [11]
	RMSSD	ms	Root mean square of successive RR interval differences	Raiz quadrada da média dos quadrados das diferenças entre os intervalos RR	Parasympathetic Activity [11]
	NN50	Uni.	Number of successive RR intervals that differ by more than 50 ms	Número de diferenças sucessivas entre os intervalos RR que são > 50 ms	Parasympathetic Activity [11]
	pNN50	%	Percentage of successive RR intervals that differ by more than 50 ms	Percentagem das diferenças sucessivas entre os intervalos RR que são > 50 ms.	Parasympathetic Activity [11]
	Geometric methods				
	HRV triangular index	-	Integral of the density of the RR interval histogram divided by its height	Integral do histograma do intervalo RR dividido pela altura do histograma.	Sympathetic and parasympathetic activities [11]
	TINN	ms	Baseline width of the RR interval histogram	Largura da base do histograma dos intervalos RR.	Global activity of the autonomic nervous system [12]
FREQUENCY	**ULF**	ms²	Ultra-low-frequency band	Ultra baixa frequência	Humoral, vasomotor and temperature regulation, and activity of the renin–angiotensin–aldosterone system [13]
	VLF	ms²	Very-low-frequency band	Muito baixa frequência (< 0.04 Hz)	Humoral, vasomotor and temperature regulation, and activity of the renin–angiotensin–aldosterone system [13]
	LF	ms²	Low-frequency band	Baixa frequência (0.04–0.15 Hz)	Sympathetic Activity (mainly in normalized units - n.u.) [11] Baroreflex sensitivity [14] Primarily parasympathetic activity (LF = (0.50 x ANSp) + (0.25 x ANSs) + (0.25 x other effects) [15]
	HF	ms²	High-frequency band	Alta frequência (0.15–0.4 Hz)	Parasympathetic Activity [11] Mainly Parasympathetic activity HF = (0.9 x ANSp) + (0.1 x ANSs) [15]
	LF/HF	ms²	Ratio of LF-to-HF power	Razão entre o LF e o HF	Sympathetic/Vagal Balance or Sympathetic Modulation [11] LF/HF = (0.50 x ANSp) + (0.25 x ANSs) + (0.25 x others) / (0.9 x ANSp) + (0.1 x ANSs)[15]
	Total power	ms²	Total spectral potential	Potencial total espectral	Sympathetic and parasympathetic activities [16]

Chart 2 - Non-linear **Heart Rate Variability** analysis methods

Analyze	Acronym	Unity	Definition in english	Definition in portuguese
POINCARÉ	SD1	ms	Standard deviation of the instantaneous RR intervals.	Desvio padrão dos intervalos RR instantâneos
	SD2	ms	Standard deviation of long-term RR intervals	Desvio padrão dos intervalos RR à longo prazo
	SD1/SD2		Ratio between short and long variations in RR intervals.	Razão entre as variações curtas e longas dos intervalos RR.
ApEn		-	Approximate Entropy	Entropia Aproximada
SampEn		-	Sample Entropy	Entropia da Amostra
Correlation dimension D_2		-	Correlation dimension	Dimensão de correlação
DFA	α_1	-	Detrended fluctuation analysis	Análise de flutuação tendência
	α_2	-		
RECURRENCE	Lmean	Beats	Average diagonal line length	Comprimento médio da linha diagonal
	Lmax	Beats	Maximum diagonal line length	Comprimento máximo da linha diagonal
	REC	%	Recurrence rate	Taxa de recorrência
	DET	%	Determinism	Determinismo
	ShanEn	-	Shannon's entropy	Entropia de Shannon

Grossman and Taylor [28] developed a behavioral biological model, a highly comprehensive alternative that emphasizes the primary function of HRV as synchronization between the respiratory and cardiovascular systems, enabling energy conservation by optimizing the efficiency of gas exchange. This model emphasizes that HRV is not necessarily a reliable measure of vagal activity generated by the nucleus ambiguus in the SA node of the heart but may reflect a wide variety of physiological and behavioral changes in the body with an energy-saving function.

The neurovisceral integration model developed by Thayer and Lane [29] uses HRV as an index of integration between the central nervous system and ANS. This model associates individual differences in cognitive performance, especially executive function, with HRV, particularly in aspects mediated by the vagus nerve. This suggests that HRV may serve as a peripheral index of central nervous system integrity, which aids goal-directed behavior. In the security context, subcortical circuits are inhibited via prefrontal cortical circuits, allowing for an increase in HRV and an improvement in executive function. In contrast, in contexts of threat, uncertainty or novelty, subcortical circuits are activated due to prefrontal hypoactivation; thus, sympathoexcitatory preparation for action occurs, resulting in a decrease in HRV.

Lehrer and Gevirtz [31] proposed a respiratory rate resonance model that emphasized the influence of breathing on HRV by modulating vagal and baroreflex activities. An important practical implication derived from this model is that the HRV of individuals can be maximized at individualized respiratory rates, which can be stimulated through cardiorespiratory biofeedback training [30,31].

These theories encompass different aspects of HRV, which provide considerable enrichment for the interpretation of changes in different HRV parameters, especially parasympathetic parameters, in experimental contexts and the expansion of perspectives in several research fields that use this research tool.

Factors that can influence HRV

HRV is influenced by several factors. Owing to the diversity of factors, this review presents the most studied factors in the literature: types and intensity of physical exercises practiced regularly and/or during electrocardiogram collection, cognitive tasks, anthropometric variables, sex, age, circadian rhythm, breathing (frequency and amplitude), food, hydration, nicotine, drugs, and physical conditioning. As such factors affect HRV and can weaken the results of the studies, it is recommended to eliminate them or to control them statistically, regardless of the chosen method of signal processing (linear or non-linear).

Anthropometric variables (body mass, body mass index [BMI], waist and hip circumference, and fat percentage) can influence HRV. Epidemiological studies have shown that poor anthropometric indicators, such as high waist circumference, visceral fat area, and BMI, can be negatively associated with HRV components, especially those related to parasympathetic activity [32,33].

Sex and age can also influence HRV. Xhyheri et al. [34] showed that healthy women have lower HRV values than healthy men with increasing age, owing to decreased estrogen release in women. There is a tendency for HRV to decrease in both sexes, with differences between sexes, and disappear with age. This has been expounded in several other studies [35-38].

Regarding the circadian rhythm, it is possible to observe that the HRV increases during the night and decreases during the day [39]. After evaluating 26 original studies, Sammito et al. [40] pointed out that almost all studies detected that the circadian rhythm influenced the HRV parameters analyzed and that the HRV increased during the night, with a peak identified during the second half of the night. More specifically, Li et al. [41] proposed that HRV is higher at night and decreases at approximately 10 am, remaining low until 7 pm, when it rises again.

Another very important variable to mention is breathing. HR increases during the inspiration phase and decreases during the expiration phase [42]. This effect occurs because of the inhibition of parasympathetic discharge in the cardiovascular center during inspiration, causing the HR to increase. During inspiration, there is a decrease in vagal activity, which causes tachycardia; during expiration, there is an increase in activity, which causes bradycardia. This physiological alteration is called Respiratory Sinus Arrhythmia and leads to variations in HR due to breathing. Thus, there is a predominance of activation of the parasympathetic ANS (during expiration), mainly responsible for the ASR. As the respiratory rhythm during rest is approximately 0.25 Hz, approximately 15 inspirations and expirations per minute, and the frequency of the HRV HF band is between 0.15 and 0.4, it is inferred that there is

a correspondence between the ASR and the HF component of HRV. Most power in the HF band is generated by respiration [43]. Therefore, HF represents ASR, and as ASR occurs due to the activity of the parasympathetic ANS, HF represents cardiac parasympathetic modulation [42]. Thus, given that breathing influences HRV, there have been several proposals for its measurement or control during HRV recording [17,44].

Food and liquid intake are rarely considered in research using HRV; however, both deserve attention. For example, meals of 500 kcal can reduce the vagal parameters of HRV even after an hour of ingestion [45]. In contrast, fasting for long periods may increase HRV vagal activity [46,47]. Therefore, it is recommended that research volunteers consume a light meal approximately 2 h before HRV recording [48]. Water consumption can also reduce HRV, particularly in the HF component [49], owing to a vagal tamponade response to the pressor effect caused by hypoosmotic fluids [50]. Other authors also support the idea that bladder and stomach distention can reduce HRV [51,52]. Therefore, it is recommended that volunteers empty their bladders immediately before the experiment [48,53].

Another important point to be controlled and studied in the literature is nicotine consumption. Nicotine consumption is associated with a reduction in the activity of the parasympathetic nervous system [54], and in some studies, this goes beyond this statement. Arastoo et al. [54], for example, evaluated 100 smokers, including 58 chronic e-cigarette users and 42 chronic tobacco cigarette smokers, and observed that both types of chronic smokers exhibited a similar baseline HRV pattern, which consisted of a reduction in the components of parasympathetic compared with non-smokers.

There is also a relationship between medication use and changes in HRV. Several drugs, especially antidepressants, antipsychotics, and antihypertensives, cause a reduction in HRV [55-57]. Among psychotropic medications, a systematic review revealed that only tricyclic antidepressants and clozapine statistically influenced HRV [58]. However, it is recommended to document and, when possible, exclude volunteers who use any medication that acts on the nervous, cardiovascular, and respiratory systems.

Finally, during the practice of physical exercise, it is expected that the HRV parameters associated with vagal activity decrease and, after its end, return to resting levels, which is considered an expected, flexible, and adaptive autonomic response [59,60]. Notably, during exercise, the intensity can produce different responses that vary according to the level of effort. Maximum or submaximal physical exertion, for example, may influence vagal withdrawal differently [61]. Vagal activity decreases progressively until complete withdrawal at approximately 50–60% of peak oxygen consumption for maximal exercise protocols, and this intensity-differentiated response extends for more than 10 min after the end of exercise [62]. Another factor is the level of fitness and endurance [62]. Individuals with low levels of physical conditioning have high sympathetic activity and low parasympathetic activity at rest [63].

Applicability to physical exercise

Therefore, the cardioprotective effects of physical exercise are undeniable. However, acutely, especially at high intensities, it is considered a stressful stimulus that increases the risk of lethal arrhythmias [64,65]. Thus, cardiac autonomic changes, such as parasympathetic (vagal) reduction and sympathetic increase, which occur during physical exercise, create a situation conducive to the development of ventricular ectopic activities, which can culminate in cardiac arrest or sudden death of an individual [66]. Based on this, Albert et al. [65] proposed the schematic model of the "window of exposure" to cardiovascular risks. According to them, owing to the unfavorable autonomic state during and a few minutes immediately after physical exercise, the practitioner is exposed to very high cardiovascular risks, which only cease when the autonomic parameters are reestablished at resting values. Nevertheless, regular physical exercise is one way to improve vagal flexibility, which is defined as the ability of the ANS to respond to a given stimulus [67].

Although HRV at rest has been well studied, its evaluation during exercise is still not completely understood, mainly because of the inconsistent results of HRV during exercise, such as the lack of vagal withdrawal during a physical test. Such conditions may be due to the variety of methods and protocols (maximum and submaximal efforts) used [68-71], physical fitness levels, resistance, body composition, and sex differences [62,63]. In general, it is thought that during physical exercise, HRV decreases relative to rest and returns to baseline values during recovery. In addition, during exercise, there is a gradual decrease in parasympathetic activity and an increase, also progressive, in sympathetic stimulation, which may be related to the increase in exercise intensity [72].

Among the possible HRV influencers listed above, we reinforce the idea that individuals with low levels of physical fitness have high sympathetic activity and low parasympathetic activity at rest, which is a negative effect, especially when evaluated under the influence of physical exercise [63]. As a result, the resting HRV of these individuals is lower, and the "window of exposure" to cardiovascular risks is greater, that is, they take longer to recover from exercise.

During exercise, intensity can produce different responses, varying according to effort level and sex. Vagal activity decreases progressively until its complete withdrawal at approximately 50-60% of peak maximal oxygen consumption for maximum protocols, and this response, differentiated by intensity, extends for more than 10 minutes after the end of the exercise [62,63]. Finally, we also point out that HRV components can be related (inversely proportional relationship) to the lactate threshold or ventilatory thresholds 1 and 2, which are important parameters for measuring or controlling the intensity of physical exercise [73-75].

Conclusion

Considering that heart rate variability is a low-cost, easy-to-acquire, and noninvasive parameter for assessing the ANS, this review aimed to provide researchers with an overview of the different issues that permeate the use of HRV parameters. It is worth mentioning that these recommendations seek to standardize HRV recording and processing and identify the influencing factors for researchers to plan their experiments in the most adequate way possible so that the results faithfully represent the changes in HRV components and are not affected by confounding variables. Finally, the interpretation of HRV is important for providing meaning and relevance to studies in this area. Given that HRV has been consistently identified as a marker of physical and mental health, public health policies are expected to be implemented in the future to allow routine use of this index by multidisciplinary teams, especially during physical exercise. Finally, we emphasize that the HRV is an important means for controlling the intensity of the exercise, including with regards to the cost-benefit, such as the devices necessary for assessing the ventilatory threshold and lactate.

Academic attachment
This article represents part of the literature review used in the thesis by Perciliany Martins de Souza, supervised by Professor Ph. D. Gabriela Guerra Leal Souza, and co-supervised by Dr. Eduardo Bearzoti at the Federal University of Ouro Preto, Ouro Preto.

Conflict of interests
The authors declare that there is no conflict of interest.

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Authors' contributions
Research conception: Souza PM and Souza GGL; **Funding:** Souza PM and Souza GGL; **Writing of the manuscript:** Souza PM, Araújo CRV, Mocaiber I, North CE, Becker LK, and Souza GGL; **Critical review of the manuscript for important intellectual content:** Becker LK and Souza GGL.

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