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Heart rate variability – from cardiology labs into the world of recreational and professional sport

Variabilnost srčne frekvence – iz kardioloških laboratorijev v svet rekreativnega in profesionalnega športa

Juš Kšela

Department of cardiovascular surgery, Division of Surgery, University Medical Centre Ljubljana, Ljubljana, Slovenia

Correspondence/ Korespondenca: Juš Kšela, e: jus.ksela@kclj.si

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Abstract

Heart rate variability (HRV) is one of the most recognized noninvasive tools in the assessment of cardiac autonomic modulation. The development of commercially available wireless heart rate monitors, detecting R-R intervals with a high resolution and accurately calculating HRV parameters, has pushed the methodology beyond the borders of exercise physiology laboratories into the world of recreational and professional sportsmen and coaches. Therefore, a growing number of Slovenian physicians are nowadays faced with questions about the physiological mechanisms of HRV and interpretational dilemmas in individuals with changed HRV parameters. Hence, the aim of the article is to clarify the physiological background of HRV, to describe conventional linear and non-linear HRV parameters and to elucidate how HRV parameters change under various physiological and pathological conditions.

Izvleček

Variabilnost srčne ferkvence (*angl.* heart rate variability, HRV) je najpogosteje uporabljena metoda za oceno avtonomne regulacije srca. S tehnološkim razvojem komercialnih merilcev srčnega utripa in pripadajočih računalniških programskih sistemov, ki omogočajo verodostojno izračunavanje, se je uporaba metodologije preselila iz kardioloških laboratorijev in specialističnih ambulant v vsakodnevno prakso rekreativnih športnih navdušencev, profesionalnih športnikov in trenerjev. Prav zato se vse več slovenskih zdravnikov pri svojem vsakdanjem kliničnem delu srečuje z vprašanji o fizioloških osnovah HRV, o načinih njenega določanja in o pravilni interpretaciji analiz. Namen članka je zato razložiti osnovne fiziološke mehanizme variabilnosti srčne frekvence, opisati konvencionalne in novejše analize in kazalnike ter opredeliti in pojasniti spremembe srčne frekvence v različnih fizioloških in patoloških stanjih organizma.

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1 Introduction

A healthy heart has the ability to generate spontaneous heartbeats, as it contains specialized rhythm-generating cells; their main characteristic is spontaneous generation of nerve impulse (1,2). Despite this automaticity or rather, inherent rhythmicity of the heart, the heart muscle is innervated by the sympathetic and parasympathetic branches of the autonomic nervous system, which regulate its contractility and heart rate. The heart is usually under the tonic influence of both branches of the autonomic nervous system, which, due to different anatomical areas of innervation, different neurotransmitters, and different receptors, elicit an opposing physiological response. While sympathetic stimulation increases heart rate and heart muscle contractility, parasympathetic stimulation slows the heart rate and reduces contractility. Parasympathetic branch activity is dominant in an inactive healthy adult (2,3). The influence of the sympathetic-parasympathetic balance on the heart, i.e. autonomic regulation of the heart, can be described by a number of indicators, and in the vast majority of studies to date, heart rate variability has been shown to be one of the best indicators of this balance (2).

The sympathetic-vagal effect on the heart changes with age and with the appearance of various cardiovascular diseases (2,4,5). Determining that we can assess the rate of progression of cardiovascular diseases by detecting changes in the autonomic regulation, or that they can also be used as predictors of complications of these diseases and even of mortality, have popularized sympathetic-vagal balance analyses in the scientific and cardiac clinical settings (2).

Studies in recent years undoubtedly show that HRV changes not only with the aging of the organism and with disease states, but also with the level of physical fitness or the level of training of an individual (6-9). Along with the development of commercially available heart rate monitors (such as Polar, Ithlet, HRV Fit, Mega Electronics), which support the calculation of RR interval variability, these findings have in recent years popularized the use of HRV analysis also in everyday life, among individuals undertaking recreational activities, and professional athletes and coaches (10,11). With the use of HRV analysis switching from cardiology laboratories and specialist clinics to the home environment, Slovenian doctors in their daily clinical work are increasingly faced with questions about the physiological basis of HRV, how to determine it and how to correctly interpret such analyses. Therefore, the purpose of this article is to explain the basic physiological mechanisms of heart rate variability, to describe conventional and recent analyses and indicators, and to define and explain changes in heart rate in correlation to various physiological and pathological conditions of the organism.

2 Heart rate variability

Today, it is known that a healthy heart, which has a sinus rhythm, does not beat evenly, but the time lengths of heart cycles (RR intervals) differ greatly from each other at the level of milliseconds (1,2). Continuous variability in the length of RR intervals is called heart rate variability (HRV) and is a reflection of the functioning of the autonomic nervous system on the sinoatrial (SA) node. (2). The constant changes in the tonus of the sympathetic and parasympathetic branches of the autonomic nervous system, which are crucial for maintaining homeostasis in the body, cause constant fluctuations in RR intervals around the mean value. The most well-known examples of physiological periodic changes in heart cycle length are respiratory sinus arrhythmia and nocturnal sinus bradycardia (1). Over the last two decades, numerous studies have shown that changes in HRV reflect changes in physiological

and pathological processes very well, and at the same time, HRV has been proven to be one of the strongest predictors of mortality after myocardial infarction, onset of benign and malignant arrhythmias, progression of cardiac failure and sudden cardiac death (2,5,12-15).

The advantages of HRV analysis are: non-invasive data acquisition, fairly simple computer applicability of otherwise complex mathematical models for its calculation and repeatability of the method. For this reason, HRV analysis is still considered as the gold standard in the assessment of autonomic regulation of the heart in various physiological and pathological conditions (2).

The physiological understanding of HRV has in recent decades shifted from reductionist perspectives to theories of complex biological systems. Reductionist perspectives perceive HRV simply as an indicator of the relationship between sympathetic and parasympathetic activation. It is perceived only as an indicator of vegetative effects on the heart and is primarily explained as a reflection of respiratory variability - either due to specific haemodynamics (altered venous inflow associated with variability in chest pressure during respiration, to which baroreceptor reflex responds with the rest), or due to intertwining of neurophysiological efferent pathways of the autonomic nervous system and respiratory stimuli - or as a reflection of the effects of the vegetative nervous system on vascular tone or circadian rhythms. Reductionist theories are linear. As such, they are limited and fail to explain certain deviations (e.g., the paradoxical behaviour of HRV in some diseases, such as endocrinological diseases, or in overtraining). That is why modern interpretations of HRV (and models for its analysis) increasingly use theories of complex biological systems: small variability is understood as inadequate adaptability of the system to external stimuli, and HRV is interpreted as a time series in which (by monitoring time variability) repetitive patterns in a complex system are "searched for" (2,4,5,12-16).

HRV can therefore be analysed by linear and newer non-linear methods, and in both cases, we measure the variations of RR intervals in the ECG records.

2.1 Linear analyses of HRV

Linear HRV methods include time and frequency domains of the analyses, with time domain indicators reflecting the magnitude of the change in heart rate and frequency domain indicators reflecting the rate of change in heart rate (2).

HRV time domain indicators are usually determined from long, 24-hour ECG recordings, and are divided into two groups: a) indicators obtained by observing individual NN intervals, (NN intervals are RR intervals caused by sinus node depolarization); and b) indicators obtained by observing differences between NN intervals. In practice, the most commonly used are: NN intervals (mean value of NN intervals), SDNN (standard deviation of all NN intervals), SDANN (standard deviation of average NN intervals, calculated from 5-minute intervals), RMSSD (square root of the mean squared difference between adjacent NN intervals), SDNN index (mean of standard deviations of all NN intervals, obtained from 5-minute intervals), SDSD (standard deviation of the difference between two adjacent NN intervals) and pNN50 (frequency of adjacent NN intervals, differing by more than 50 ms). As part of the time domain of HRV analysis, there is also the so-called geometric method that contributes a triangular index to the HRV (total number of NN intervals divided by the number of NN intervals in the modal bin), and TINN (triangular interpolation of NN intervals on the histogram). Time domain indicators mainly reflect parasympathetic activity (2).

Frequency domain indicators are determined from short-term, usually 2- to 5-minute ECG recordings. The analysis

is based on the decomposition of the sequence of NN intervals into groups of sinusoidal curves of different amplitudes and frequencies by means of a fast Fourier transform. The result of the analysis is presented as the height of the variability of the frequency function, which is denoted as the power of the spectrum. In frequency analysis, we observe the total power of the spectrum (TP) in the frequency interval between 0.01 and 0.4 Hz and the power of individual areas: high-frequency component (HF, 0.15–0.4 Hz), which is an indicator of vagal activity, low-frequency component (LF, 0.04–0.15 Hz), which is an indicator of modulated sympathetic activity, and the very low-frequency component (VLF, 0.01–0.04 Hz), which is still poorly explained, although some authors associate it with the activation of the renin-angiotensin-aldosterone system. As such, it is supposed to reflect sympathetic activities (17). Power ratio of low-frequency and high-frequency components (LF : HF) mirrors the sympathetic-vagal balance (2).

2.2 Non-linear analyses of HRV

Non-linear HRV analyses are based on mathematics of complex dynamics, chaos theory, and fractal geometry (12,16,18).

A dynamic system or process that is seemingly random, but essentially precisely regulated according to its own intrinsic rules, is said to be chaotic or in a state of chaos (19). In nature, the properties of chaotic behaviour are shown by many processes, such as electrical circuits, oscillating chemical reactions or the dynamics of various liquids (19,20). Chaotic processes form fractals. They are geometric objects that can be divided into smaller parts, all of which reflect the structure of the original whole: they are self-similar objects independent of the observation size class. In nature, clouds, snowflakes, some plants, or lightning during storms show a certain degree of self-similarity. Studies over the last two decades have unequivocally demonstrated that complex dynamic behaviour in time and space is also demonstrated by biological systems and processes. In the human body, fractal properties are shown by a number of structures, such as branching of the arterial and venous systems, bronchial branches and His-Purkinje nerve bundles, and nerve entanglements in the central nervous system. Today, chaotic behaviour is also known to be exhibited by the heartbeat. The ECG records indicate an apparent periodicity, but the resting heart rate of a healthy person is a very dynamic process. It has the properties of statistical self-similarity, which is reflected in many time size classes, from a few milliseconds to a few hours (12,16,18). While linear HRV analyses have neglected the complex dynamics of the heartbeat as if they were "useless murmurs," non-linear methods show that the aforementioned fractality contains a range of important, hitherto hidden information. Studies conducted so far have shown that the breakdown of the fractal organization of the heartbeat into excessive order or into unrelated coincidence is a sign of system's reduced ability to adapt to change. It is characteristic of aging and various disease states (4,12).

The complexity of fractals due to their irregularity cannot be described by conventional mathematical methods and Euclidean geometry, but is described by Mandelbrot's fractal geometry or mathematics of complex dynamics and chaos theory (12,16,19). By using such mathematical methods in biomedicine, new possibilities have opened up for the analysis of a series of seemingly irregular biological structures and phenomena (4,12,16,18). A number of non-linear methods are used to assess heart rate dynamics, the most commonly used being detrended fluctuation analysis (DFA), fractal dimension (FD), Approximate entropy (ApEn), Hurst exponent (H), 1/f noise, symbolic dynamics, and Lyapunov exponent (19). Some authors even consider that the time series of the ECG records is so non-stationary and

inhomogeneous that a large number of local fractal exponents, known as multifractal analysis, need to be used for a credible description of its dynamics (21).

Among the mentioned non-linear methods, the DFA analysis has proven to be the most reliable indicator of the complex dynamics of ECG recordings in previous studies (4,12,16,18). DFA is a method of quantitatively assessing the self-similarity of nonstationary time series and is based on detrending time series and determining the trend line according to the least squares principle of error. In this case, we obtain a line whose slope is represented by the self-similarity coefficient α. Values of α around 1 indicate the self-similarity of the time series. DFA in a logarithmic graph does not provide a completely linear line, but a "two-segment line" consisting of a line with a breaking point at a time series size of about 11 RR intervals. Therefore, many authors prefer to calculate the exponent of each part of a two-segment line separately, namely: the short-term exponent α 1 and the long-term exponent α 2. Values of α 1 around 1.5 and α 2 around 1 indicate self-similarity of the ECG records (20).

Normal values of the most commonly used linear indicators and the most established non-linear DFA indicator are shown in Table 1.

3 Changes in HRV in various physiological and pathophysiological conditions

HRV is high in young healthy people and indicates a healthy response of sympathetic-vagal balance to minimal changes in homeostasis. When partially decreased, it is a sign of normal aging of the organism and indicates a gradual loss of vagal dominance. Severely decreased or even absent HRV results from a completely abnormal response of the autonomic nervous system to disturbances in homeostasis and is a sign of a number of cardiovascular diseases, such as coronary heart disease, acute coronary syndrome, heart failure, supraventricular and ventricular arrhythmias, diabetic neuropathy and various conditions that follow myocardial infarction, heart surgery and heart transplant (2,4,5,12-18). Although the exact pathophysiological mechanisms that lead to a decrease in HRV with these pathological conditions have not been fully explained, there are quite a few theories that identify the most likely causes of sympathetic-vagal imbalance with individual diseases (2). According to one of the theories, the drop in HRV in patients with myocardial infarction is due to the activation of cardiac sympathetic-sympathetic and sympathetic-vagal reflexes, with changes in the geometry of ventricular contraction (as a result of a necrotic and/or hibernating myocardium), causing mechanical disturbance of sensory endings, which resulted in increased triggering of sympathetic afferent fibres. This ultimately leads to the predominance of sympathetic influence over vagal influence on the SA node (2). In patients who experience a marked decrease in HRV after myocardial infarction, a reduced SA node response to neuromodulation is the more likely cause (2). In diabetes, the fall in HRV may happen due to cardiac autonomic neuropathy, associated with impaired glucose metabolism and inflammation; or it results from a reduced hypothalamic efferent stimulus for secretion of insulin and concurrent activation of the vagal nerve (2). In heart failure, reduced HRV can be explained by disrupted cardiac haemodynamics, along with the predominance of the sympathetic nervous system due to the activation of complex compensatory mechanisms that occur as part of heart failure syndrome. Under such conditions, the SA node becomes completely unresponsive to neural stimuli (2). In patients following heart transplantation, the disturbed sympathetic-vagal balance results from complete denervation of the donor heart and an isolated response of the heart muscle to the

circulating catecholamines (2). HRV is greatly altered also after any type of heart surgery. Our research team has found in previous studies that HRV is greatly reduced after both dormant and beating heart surgery and that it remains affected for at least 4 weeks after surgery, meaning that stress during surgery and damage to cardiac nerves due to manipulation of the heart and large blood vessels during surgery lead to a sympathetic predominance that lasts for at least a few weeks after surgery (22,23).

Linear indicators of the TP, HF, LF, and VLF frequency domains are statistically significantly lower in patients with coronary heart disease, heart failure, and following a myocardial infarction, indicating decreased vagal and/or increased sympathetic rhythm regulation in these individuals (2). In cases of an increased sympathetic tone, there is also a decrease in all parameters of the time domain of linear HRV analysis (2).

Linear frequency domain indicators have been shown to be good predictors of sudden cardiac death, myocardial infarction (MI) mortality, and onset of post-infarction arrhythmic events (2,24-26). In studies, SDNN and SDANN indicators proved to be among the best predictors of mortality following MI (2). The ATRAMI study has shown that the patients following an MI, with whom SDNN (determined from 24-hour ECG recordings) is < 70 ms, have a 3.2 times faster mortality rate in the first 21 months following MI than patients with SDNN > 100 ms (24). Similarly, the MPIP study has shown that patients, following AMI with SDNN < 50 ms (when compared to patients with SDNN > 100 ms), are 5.3 times more likely to die in the first 31 months after a heart attack (24,25). Patients with postinfarction VT have significantly lower LF values compared to patients with post-infarction sinus rhythm. Before the onset of sympathetically or vagally modulated AF, the values of RMSSD, HF and LF ratios are significantly reduced: HF (26). Patients with higher values of linear HRV indicators have fewer ventricular arrhythmias, suggesting a protective role of vagal tone in the occurrence of ventricular arrhythmias. In patients with coronary heart disease, the probability of hemodynamic significance of stenosis is 0.77 times, 0.75 times, 0.72 times or 0.76 times lower, respectively, for each increase in HF, SDNN, RMSSD and pNN20 levels for level 1 SD (24). Decreased HRV is an independent predictor of mortality even in patients with heart failure. The UK-Heart study showed that the annual mortality of patients in the classes NYHA I-III is 5.5% for SDNN > 100 ms, 12.7% for SDNN between 50 and 100 ms and 51.4% for SDNN < 50 ms (24). In addition, in patients with heart failure, the value of SDANN ≤ 65 ms before starting resynchronization treatment (CRT) or SDANN ≤ 76 ms 4 weeks after implantation of a CRT pacemaker, an independent predictor of the progression of heart failure to the extent that a heart transplant is required (24,25).

In cases of various cardiovascular diseases, non-linear HRV indicators are also significantly changed; they are better indicators of the progression of these diseases and better predictors for the occurrence of adverse events than linear indicators (12-18,24-26). Non-linear indicators are mainly influenced by the parasympathetic branch of the autonomic nervous system and less influenced by the sympathetic nervous system, so most parameters such as α 1, average FD and ApEn clearly reflect vagal activity, and only a few, such as α 2 and 1/f slope, reflect a sympathetic activity. Patients with significantly higher, post-infarction values of non-linear and equal values of linear indicators live longer following a myocardial infarction (25-27). The α 1 indicator has proven to be an independent predictor of the occurrence of benign and malignant cardiac arrhythmias, sudden cardiac death, and mortality in patients with reduced left ventricular ejection fraction. A statistically significant drop in the indicator is also observed after interventions on a beating or dormant

heart. In addition, changes in the parameter α 1 reflect the normal aging process of the organism very well (12-18,24-27).

The connections between HRV and inflammation, stress and hormonal disorders are also interesting and therefore worth mentioning. Inflammation, which is the body's basic protective response to a microbial infection or injury, is a carefully controlled process that is significantly guided and regulated by the autonomic nervous system (28), primarily via the vagally modulated cholinergic anti-inflammatory pathway (29). Meta-analyses of studies from the last two decades show that there is an inverse relationship between vagally modulated HRV indicators and laboratory indicators of inflammation, proving that the parasympathetic branch of the autonomic nervous system plays a dominant role in the inflammatory reflex and acts anti-inflammatory, while the (in this case) less important sympathetic branch of the autonomic nervous system acts either pro- or anti-inflammatory. The degree of inflammation can be reliably determined with vagally modulated HRV indicators (29). According to Selye's theory, stress is a state of endangered homeostasis, caused by internal or external stressors, to which a healthy organism responds with the so-called stress response (30). Autonomic nervous system activity is the foundation of the stress response and includes activity of both sympathetic and parasympathetic branch. In a phase of acute stress, the organism achieves homeostasis by fine regulation of the sympathetic-vagal balance, while in phases of chronic stress, with a constant increase in circulating catecholamines and cortisol, it leads to a violation of the sympathetic-vagal balance. When this happens, the parasympathetic branch is not able to respond to stressors and the simultaneous sympathetic dominance, which is the reason for a decreased HRV in the stages of chronic stress (30). Like other stressors, hormonal disorders cause a long-term and chronic disruption of homeostasis in the body and disrupt the sympathetic-vagal balance, altering HRV (2,30).

The data on the effect of different drugs on HRV indicators is also important in clinical practice. Treatment with beta-blockers increases the variability of RR intervals and with it, HRV. Some antiarrhythmics, such as flecainide and propafenone, reduce HRV, while amiodarone treatment does not affect HRV indicators. Muscarinic receptor antagonists such as atropine and scopolamine increase vagal activity and with it, HRV (2).

When we talk about the clinical significance and prognostic values of HRV in various physiological or disease states, it is of course necessary to be aware of some limitations that the analysis of variability of RR intervals has: a) HRV has usually been studied as an observed event (health indicator) in the previous studies, but not as a central subject of research (e.g. why it exists at all, what are the cellular mechanisms of its formation and maintenance, etc.), therefore many questions remain unclear about its significance; b) while the linear HRV parameters are already well known and established, the non-linear HRV indicators are currently relatively poorly defined, rarely validated (each study offers its own parameter) and verified in a limited fashion in terms of both normal values and their predictive role in various pathological conditions; c) due to the pronounced individual variability of HRV, it is still studied at the level of the population or extremely large groups of subjects (2,18).

4 Changes in HRV during physical activity

Regular intensive aerobic exercise leads to a number of adaptation mechanisms of the organism, which enable increased delivery of oxygen to the active muscle tissue and a sufficient perfusion of target organs in the phase of effort or physical exercise, and are also visible at rest (1,6,7-9). These adaptations are due to complex chang-

es at the molecular cellular level, ranging from altered gene expression and enzyme function to changes in hormonal states, receptor responses, and target organ function. With sufficient training, these mechanisms are triggered regardless of the race, gender, and age of the individual in the first 2 to 3 months of regular exercise. They improve baseline resting values by approximately 25% in well-trained individuals. Adjustments such as a decrease in resting heart rate and submaximal exertion (while the maximum heart rate usually does not change or only a slight decrease occurs), increased heart rate and cardiac minute volume, and decreased respiration at submaximal exertion are largely due to changes in the functioning of the autonomic regulation of the heart as a result of physical activity (6-9).

Individuals exposed to regular aerobic exercise have a significantly higher resting vagal tone than individuals who are not exposed to such exercise. Higher vagal tone protects physically active individuals from sudden death, from the development of cardiovascular disease, and from benign and malignant arrhythmias. The exact mechanisms that lead to increased vagal modulation in these humans are not fully known. At least part of the change in the autonomic nervous system, however, occurs due to altered levels of angiotensin II and nitric oxide (7). Comparisons of well-trained athletes with their non-physically active peers have shown that athletes have lower plasma renin levels and therefore lower levels of angiotensin II, which reduces vagal activity. This means that vagal modulation increases at lower angiotensin II levels. In addition, regular aerobic exercise increases endothelial function, thereby raising the level of available nitric oxide, which directly increases vagal tone and decreases sympathetic tone (7).

Directly during aerobic exercise, the heart rate and cardiac output increase in order for the body to provide a sufficient amount of oxygen in the active muscle and to provide a sufficient perfusion of vital organs (1). In the initial phase of exercise or at lower loads, the increase in heart rate and minute volume is due to reduced vagal tone, when an individual reaches about 40% of their maximum aerobic capacity (then the heart rate is usually around 100 beats per minute). At the same time, the sympathetic activity also begins to increase (7). At maximum effort, when an individual reaches (sub)maximum aerobic capacity and (sub)maximum heart rate, there is a highly increased sympathetic activity with simultaneous, practically eliminated vagal modulation. Maximum aerobic performance can be greatly increased in well-trained individuals and is considered to be the best indicator of an athlete's readiness or training. After a short (10- to 20-minute) (sub)maximum exercise, the vagus activity rises to baseline levels within a few minutes, and after a long workout, only after a few hours or days. In better-trained athletes, autonomic heart regulation improves faster than in less-trained athletes, and the duration of HRV correction can also serve as an indicator of an athlete's training or physical fitness.

In accordance with changes in the sympathetic-vagus balance, regular and intense aerobic exercise also increases heart rate variability, which is a reflection of a healthy heart response to changes in homeostasis (1,7). Individuals exposed to regular aerobic exercise have elevated all linear indicators that reflect vagal activity, and the α 1 indicator in these individuals is about 1.5, indicating a higher degree of self-similarity of the ECG records. Directly during aerobic exercise, all linear indicators that reflect vagal activity fall, and linear indicators that reflect sympathetic activity increase. Interestingly, after the initial drop, the linear indicators no longer decrease despite the increase in aerobic load. The opposite is true for non-linear indicators. At a lower load level, the α 1 indicator shows a loss of self-similarity of the ECG record with a change towards Brownian motion or the so-called random walk when an individual exceeds a load of

approximately 40% of maximum aerobic capacity (is, therefore, at the limit). However, when the fall in vagal modulation is accompanied by a gradual increase in sympathetic activity, α 1 begins to fall linearly toward white noise or complete non- or anticorrelation of the ECG recordings (6-9).

The endurance of a recreational or professional athlete and their training can be objectified today with a number of "endurance indicators", such as the ventilation threshold and the point of respiratory compensation, which are considered to be the most frequently used indicators of an individual's physical fitness. Endurance indicators are assessed in specially adapted sports and cardiology laboratories with gas analysis of inhaled and exhaled air and blood tests to determine lactate levels be-

Table 1: Reference values of the most established linear and non-linear indicators of heart rate variability (summarized after Report) (2).

Indicator	Units	Description	Activity indicators	Reference values (mean value ± SD)
Linear time domain indicators (24-hour recordings)				
SDNN	ms	standard deviation of all NN intervals	psy	141 ± 39
SDANN	ms	standard deviation of average NN intervals, calculated from 5-minute intervals	psy	127 ± 35
RMSSD	ms	the square root of the mean squared difference between adjacent NN intervals	psy	27 ± 12
HRV triangular index	N/A	the total number of NN intervals divided by the number of NN intervals in the modal bin	psy	37 ± 15
Linear frequency domain indicators (5–15 minute recordings)				
TP	ms^2	the total power of the spectrum in the frequency range between 0.01 and 0.4 Hz		3466 ± 1018
LF	ms^2	low-frequency component of the spectrum in the frequency interval between 0.04 and 0.15 Hz	sy	1170 ± 416
HF	ms^2	high-frequency component of the spectrum in the frequency interval between 0.15 and 0.4 Hz	psy	975 ± 203
nLF	nu	low-frequency component of the spectrum, expressed in normalized units: LF/(TP–VLF)x100	sy	54 ± 4
nHF	nu	high-frequency component of the spectrum, expressed in normalized units: HF/(TP–VLF)x100	psy	29 ± 3
LF/HF ratio	N/A	the ratio between the low- and high-frequency component of the spectrum		1,5-2,0
Non-linear indicators (5–15 minute recordings)				
DFA α 1	N/A	short-term exponent of quantitative assessment of self-similarity, of non-stationary time series	psy	1,5
DFA α 2	N/A	long-term exponent of quantitative assessment of self-similarity, of non-stationary time series	sy	1

NOTES: psy - parasympathetic activity, sy - sympathetic activity, VLF - very low-frequency component of the spectrum in the frequency interval between 0.01 and 0.04 Hz, DFA - detrended fluctuation analysis, nu - normalized units).

tween different levels of (sub)maximum aerobic and anaerobic load, which means that these are technically more demanding, more expensive and time-consuming test methods (7-9). In recent years, a number of studies has shown that during exercise, changes in HRV indicators coincide well with changes in ventilation curves obtained during classical gas analysis of inhaled and exhaled air during exercise testing (31,32). The exact mechanisms of the connection between ventilation and HRV have yet to be fully explained. Most likely, a healthy organism achieves a new homeostasis through changes in the sympathetic-vagal balance through changes in blood pressure, tissue metabolism and circulating hormones. Findings that changes in HRV indicators could also be used to determine endurance indicators such as aerobic and anaerobic ventilation threshold, maximum oxygen consumption and respiratory compensation point in a non-invasive and fairly simple manner, have further popularized the method of HRV analysis among professional athletes and coaches. Studies show that the aerobic ventilation threshold is most easily determined by observing changes in non-linear HRV indicators (e.g., in Poincaré groups). The anaerobic threshold is determined by observing changes in linear indicators (e.g. HF vagal modulation indicator) (31-33). Clinically, the most interesting evidence is that significant changes in linear HRV indicators (significant decrease in vagal indicators and/or significant increase in synaptic indicators) – measured during exercise with an increasing intensity – coincide extremely well with the onset of anaerobic threshold at which exponential blood lactate accumulation begins. These statistically significant changes in HRV indicators indicate a significant drop in vagal activity at the time of anaerobic threshold and a complete sympathetic dominance (31-33). Also, research in recent years has been clinically extremely interesting, proving that it is possible to use balanced models – with balanced consideration of anthropometric data (age, sex, height, body fat and muscle mass), resting heart rate and linear and non-linear HRV indicators – in a non-invasive way to determine the maximum oxygen consumption, which is considered one of the best indicators of the functional ability of athletes (34,35). However, as there is little data so far, we cannot unfortunately yet say with certainty what are the specificity and sensitivity of these non-invasive and, for the athletes, extremely welcome new non-invasive methods (31-35).

Intense aerobic exercise in principle similarly changes the sympathetic-vagal balance and HRV indicators in all individuals. Nevertheless, the actual state of autonomic balance (and therefore the values of HRV indicators) for each individual athlete depends on a number of factors, such as the type of sport (it does not matter whether they are runners, cyclists, skiers or biathletes), manner of training (predominant aerobic or anaerobic exercise), exercise intensity, duration of exercise and rest intervals and, last but not least, the initial aerobic fitness level of the individual (6-9). Given such a pronounced individual difference in the status of the autonomous system in well-trained athletes, it is currently impossible to determine exactly which values of HRV indicators are considered "normal" in an individual and at which values they become clinically significantly changed. That is why today, most athletes use HRV for self-monitoring; values in the individual are therefore self-control at different stages of training and at different stages of training. For proper self-monitoring, it is most often advised that individuals measure their so-called basal HRV after 7 to 10 days of rest (non-training interval), preferably early in the morning, after about five minutes of standing, after urination, and before food intake. This provides the most realistic assessment of basal vagal modulation, not disturbed by daily physical activity, postprandial metabolism, and stress. Then, in the phase of repeated aerobic exercise, they

should regularly monitor HRV indicators and train in accordance with the obtained results: if vagal activity increases compared to the basic level, they can increase the intensity of training, and if it decreases, they should reduce the intensity of aerobic exercise (31,32)When HRV is reduced over a long period of time and does not improve despite a decrease in physical activity, this is an indicator of overtraining syndrome (36). Overtraining occurs when the intensity and frequency of exercise are such that the body is not able to regenerate normally during the phases of rest during exercise. Overtraining syndrome is characterized by a constant predominance of the sympathetic nervous system both in the resting and in the exercise phase. (36).

5 HRV estimates with commercial heart rate monitors

Until recently, we exclusively used traditional ECG meters to determine HRV indicators, which, due to their technological dispositions, enable the recording of short-term or Holter ECG recordings in controlled laboratory conditions or in everyday life in a home environment with only moderate physical activity. With the technological development of commercial heart rate monitors and computer-aided systems for calculating variability, the possibilities of observing HRV in the daily, real life of an individual have opened up: in phases of moderate or (sub)maximum loads, in phases of regular and interval loads, in phases of rest and regeneration, at day-to-day intervals or while engaging in various sports (10,11,37). This, of course, raises two questions: (1) Whether commercially available meters capture information about actual RR intervals well enough (and therefore actually detect heart rate or perhaps just noise due to vibrations in sports activity); (2) whether computer systems that calculate HRV indicators can reliably calculate variability from the data obtained in this way. Studies in recent years suggest that the answer to both is yes (10,11,37,38). A comparison of hospital Holter monitors and commercially available monitors showed that commercially available heart rate monitors are trustworthy and reliably capture true RR intervals, and the associated computer systems dependably and reliably filter the recordings and provide comparable heart rate variability calculations. Most commercial heart rate monitors available today calculate linear HRV indicators.

Finding that commercially available monitors provide credible HRV values comparable to those obtained in cardiology laboratories opened up new possibilities for conducting investigations in regular clinical work (37,38). While the use of hospital systems is often expensive, patient-unfriendly, less functional and more difficult to access, the use of commercially available monitors is - on the contrary - easier to access, significantly cheaper, more patient-friendly and allows for normal daily activities as well as greater physical activity loads in a wide variety of environments. Of course, the fact remains that most commercial heart rate monitors provide only RR intervals, while hospital systems also contain multi-channel ECG recordings, which are often essential for clinical and therapeutic decisions.

For a clinician who encounters individuals who use heart rate monitors in their daily routine, and gets asked questions about the interpretation of the results and is asked to provide advice on exercise, it is crucial to know which information said clinician can expect from the patient/athlete and what they can advise such an individual on the basis of this information. The vast majority of widely available and used heart rate monitors today calculate a strong vagal linear indicator, such as RMSSD, SDNN, pNN50 or HF, from the measured RR intervals; before interpreting the results, it is necessary to know which indicator is calculated, since different indicators have different target values.

It is important to know that the levels of HRV indicators are individually different and that the target values obtained from different studies have a large standard deviation, which means that it is difficult and pointless to compare individuals among each other. It is much more reliable to compare the patient/athlete to themselves in different periods or in periods of different phases of training or different physical loads. In addition, it is important to be aware that a single measurement of HRV indicators does not have a large predictive value, but it is always necessary to compare an individual measurement with the trend of HRV indicators over a longer period for each user. Therefore, users should be reminded that the first meaningful assessments and interpretations of the results will be possible only after a few weeks of regular use of the monitoring device, when the first trend curves of an individual's HRV will have been produced. As the number of measurements performed increases, the data will become more and more reliable, as trend curves will increasingly reflect the variability of heart rate in each individual. Users need to be told that smaller declines in HRV values from their own trend curves during the training phases are normal. In such cases, it is necessary to reduce the level of activity and allow the body to regenerate normally. This is shown by the re-increase of HRV. However, significant declines in the value of HRV, which do not improve despite the reduction in physical activity, indicate an overtraining syndrome. In this case, a longer regeneration period with the cessation of extreme physical exertion is necessary (36).

6 Conclusion

HRV, which has been considered the gold standard among cardiologists and electrophysiologists for decades in assessing autonomic heart regulation, has left cardiology laboratories in recent years and is paving the way for the world of sports and developing new possibilities for controlled training in everyday life. At the same time, care must be taken when interpreting HRV results, as incorrect understanding of the physiological basis and misinterpretation of changes in HRV indicators can force an individual into an unhealthy and harmful way of exercise. Healthy, young people have a high heart rate variability, which declines with age and cardiovascular disease. Well-trained endurance athletes have a high HRV, which means they have a greatly increased vagal tone. During physical activity, HRV begins to decrease, and is completely lost in the range of maximum load, when the predominance of the sympathetic nervous system completely predominates, with virtually nullified vagal activity. The greater the endurance of an individual or their physical fitness, the longer such an athlete will have a higher vagal tone and with it, a higher maintained HRV. Smaller declines in the value of HRV during training phases are normal. In these cases, it is necessary to reduce the level of activity and allow the body to regenerate normally, which is manifested by a re-increase in vagal activity or an increase in HRV. Large declines in HRV, which do not improve despite a decrease in physical activity, indicate an overtraining syndrome. It is a level of fitness that does not allow the body to regenerate normally in the resting phases in between training and is manifested by the constant predominance of the sympathetic nervous system both in the resting and in the exercise phase.

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