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Heart Rate Variability in Athletes

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Contents

Abstract

This review examines the influence on heart rate variability (HRV) indices in athletes from training status, different types of exercise training, sex and ageing, presented from both cross-sectional and longitudinal studies. The predictability of HRV in over-training, athletic condition and athletic performance is also included. Finally, some recommendations concerning the application of HRV methods in athletes are made.

The cardiovascular system is mostly controlled by autonomic regulation through the activity of sympathetic and parasympathetic pathways of the autonomic nervous system. Analysis of HRV permits insight in this control mechanism. It can easily be determined from ECG recordings, resulting in time series (RR-intervals) that are usually analysed in time and frequency domains. As a first approach, it can be assumed that power in different frequency bands corresponds to activity of sympathetic (0.04–0.15Hz) and parasympathetic (0.15–0.4Hz) nerves. However, other mechanisms (and feedback loops) are also at work, especially in the low frequency band.

During dynamic exercise, it is generally assumed that heart rate increases due to both a parasympathetic withdrawal and an augmented sympathetic activity. However, because some authors disagree with the former statement and the fact that during exercise there is also a technical problem related to the non-stationary signals, a critical look at interpretation of results is needed.

It is strongly suggested that, when presenting reports on HRV studies related to exercise physiology in general or concerned with athletes, a detailed description should be provided on analysis methods, as well as concerning population, and training schedule, intensity and duration. Most studies concern relatively small numbers of study participants, diminishing the power of statistics. Therefore, multicentre studies would be preferable.

In order to further develop this fascinating research field, we advocate prospective, randomised, controlled, long-term studies using validated measurement methods. Finally, there is a strong need for basic research on the nature of the control and regulating mechanism exerted by the autonomic nervous system on cardiovascular function in athletes, preferably with a multidisciplinary approach between cardiologists, exercise physiologists, pulmonary physiologists, coaches and biomedical engineers.

The manner in which the intact organism (in general) and the cardiovascular system (more specifically) responds to the stress of exercise has intrigued sports physiologists for the past century. The cardiovascular adjustments necessary to meet the extraordinary demands of the working musculature, which begins even before the onset of exercise, remains an area of intense investigation and speculation.[1,2] Also, anatomical geometry and cardiovascular function of the heart are altered after chronic physical activity.[3] For example, on the one hand, persistent volume load such as that elicited after endurance training (or its pathological equivalent after aortic or mitral insufficiency) leads to enlargement of the left ventricular internal diameter and a proportional increase in wall thickness.[4,5] This type of adaptation is called eccentric left ventricular hypertrophy. On the other hand, a pressure load such as elicited after power training (or its pathological equivalent of aortic stenosis or hypertension) leads to a thickening of the ventricular wall and an unchanged internal dimension. This type of adaptation is called concentric left ventricular hypertrophy. An essential difference between exercise and pathological conditions is that the load on the heart is continuous in the latter case and intermittent in the former.

Other adjustments take place in almost every organ system of the body and involve all aspects of cardiac and peripheral vascular control, including regulation by the autonomic nervous system (ANS). Neural mechanisms appear to be of great importance in mediating the initial response to exercise, which involves very rapid changes in heart rate and blood pressure. All these phenomena involving heart rate and blood pressure are described as 'cardiovascular variability'. Both phenomena covered in this review, exercise training and its relation to control and regulation of the cardiovascular function by the ANS, also have the following important clinical aspects: (i) can exercise training be used to retard the advance of coronary and other heart diseases?; and (ii) can HRV be used as a predictor or as a marker of the progression of cardiovascular disease?

Understanding interactions between cardiovascular function, activity of the ANS and exercise training, remains a difficult problem. The disciplines of medicine, exercise and environmental physiology, physical education and biomedical engineering are all closely allied to study the effects of exercise and other stresses on cardiac structure and function.

This review discusses how some of the consequences of exercise training on the cardiovascular system can be deducted from measured basic experimental data of heart rate variability (HRV), aortic blood pressure variability (BPV) and baroreflex sensitivity (BRS). More specifically, time and frequency analysis of heart rate will be described as a valuable tool to investigate the reflex mechanisms of cardiovascular regulation in active athletes in a fully non-invasive way.

The parameters of HRV, BPV and BRS can simply be obtained from the measurement of the ECG (and heart rate) and (non-invasive) blood pressure as will be shown in section 2. Indices from HRV and BPV can be studied in time (statistical studies) and frequency domain (power spectrum). These indices can be a valuable non-invasive tool to investigate the reflex mechanisms of cardiovascular regulation during and after exercising, for de-training and over-training, sex differences and the effects of ageing.

This review discusses consecutively: (i) control mechanisms of heart rate and blood pressure and the role of the ANS; (ii) how to measure experimentally and analyse HRV and BPV, starting from the ECG and (non-invasive) blood pressure signals; (iii) correlation between HRV and physical and physiological parameters; and (iv) HRV data obtained from studies on athletes and related to training, training overload, and age and sex differences.

1. Control of Heart Rate: the Autonomic Nervous System (ANS)

The cardiovascular system, the heart and circulation, are mostly controlled by higher brain centres (central command) and cardiovascular control areas in the brain stem through the activity of sympathetic and parasympathetic nerves.[6] Control is also affected by baroreceptors, chemoreceptors, muscle afferent, local tissue metabolism and circulating hormones.[7] Study of cardiovascular variability mainly allows access to the activity of the nerves and the baroreceptors.

The ANS describes those nerves that are concerned predominantly with the regulation of bodily functions. These nerves generally function without consciousness or volition. Autonomic nerves comprise sympathetic nerves and parasympathetic nerves (the latter often being used as a synonym of vagal, because the parasympathetic supply to the heart runs in the vagal nerves). Both divisions contain both afferent and efferent nerves and both myelinated and non-myelinated fibres. In general, the effects of the two divisions are complementary, with activity in sympathetic nerves exciting the heart (increasing heart rate), constricting blood vessels, decreasing gastrointestinal motility and constricting sphincters, and parasympathetic nerves inducing the opposite response. The autonomic system supplies both afferent and efferent nerves to the heart, with sympathetic nerve endings all over the myocardium and parasympathetic on the sino-atrial node, on the atrial myocardium and the atrio-ventricular node. These nerves not only control heart rate and force, but both sympathetic and parasympathetic nerves supply important reflexogenic areas in various parts of the heart, which when excited by either mechanical or chemical stimuli, give rise to reflexes that influence both the heart itself and the state of constriction of blood vessels.[8] These neural pathways are also closely linked to baroreceptor reflex activity, with changes in blood pressure playing a key role in either increasing or decreasing activity of one or the other pathway.

Analysis of cardiovascular variability permitted insight into the neural control mechanism of the heart, leading to a new discipline: 'neurocardiology'.[9-11] This area combines the disciplines of neurosciences and cardiovascular physiology on the research side, and of neurology and cardiology on the clinical side.

Normal heartbeat and blood pressure vary secondary to respiration (respiratory sinus arrhythmia), in response to physical, environmental, mental and multiple other factors and is characterised by a circadian variation. Both the basic heart rate and its modulation are primarily determined by alterations in autonomic activity. Increased parasympathetic nervous activity slows the heart rate and increased sympathetic activity increases the heart rate (figure

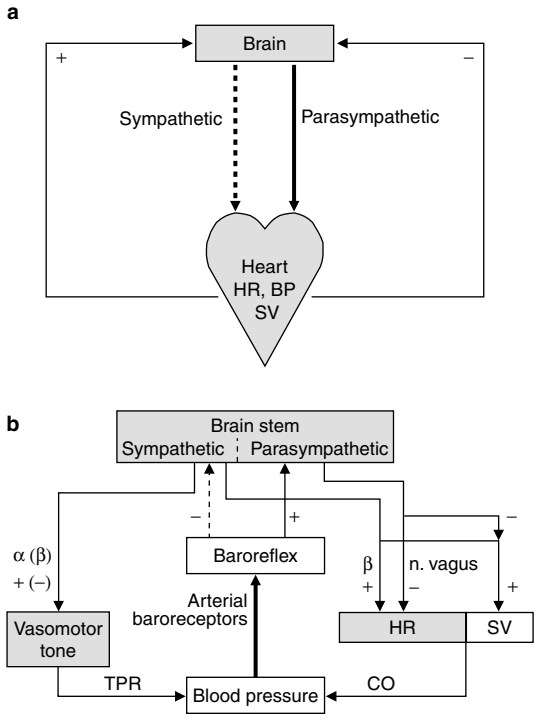

Fig. 1. (**a**) A very simple model illustrating the influence of the sympathetic (increase in heart rate) and parasympathetic (decrease in heart rate) nervous activity on heart rate, the so called 'balance model'. (**b**) A more elaborate working model of cardiovascular control mechanisms of HR, BP and the feedback mechanism from the baroreflex. This illustrates independent actions of the vagal, α- and β-sympathetic systems. Their action can be assessed by measuring heart rate variability, blood pressure variability and the baroreflex mechanism. The parasympathetic activity is responsible for the bradycardia accompanying baroreceptor stimulation and for the tachycardia accompanying baroreceptor deactivation, with the sympathetic nervous system also playing a minor role. **BP** = blood pressure; **CO** = cardiac output; **HR** = heart rate; **n. vagus** = nervus vagus; **SV** = stroke volume; **TPR** = total peripheral resistance; α = α-sympathetic system; β = β-sympathetic system.

1).[12] However, in reality the situation is much more complex and figure 1b depicts a more evolved working model that, starting from central cardiovascular control as a black box, identifies the output of the ANS to blood pressure and heart rate, and describes the feedback loop via the baroreceptors.[13] In a healthy individual, the role of the ANS in the beat-to-beat adjustment of haemodynamic parameters is essential to adequate cardiovascular functioning. Therefore, cardiovascular control, as expressed by the time-dependence of haemodynamic variables, is a direct reflection of autonomic activity. It may be a useful tool to examine autonomic fluctuations under different physiological circumstances[14] or to study external influences such as the effect of training.

Autonomic nerves, therefore, have a pivotal role in the regulation of the cardiovascular system both in ensuring optimal function during various activities in health under varying physical conditions, even during weightlessness,[15] and also in mediating several of the manifestations of cardiac diseases.

2. Methodology and Analysis of Cardiovascular Variability: Heart Rate Variability (HRV), Blood Pressure Variability and Baroreflex Sensitivity

The first step for the analysis of HRV and BPV signals is obtaining high-quality ECG and (non-invasive) blood pressure tracings under stationary conditions (figure 2). As the analysis of the ECG and blood pressure are very similar, only the ECG will be discussed further. Duration of recordings can extend from a minimum of 10 minutes to 24 hours in Holter recordings. The duration has to be sufficiently long and stationary during that period, allowing a good frequency resolution. For frequency domain measurements, it is recommended that the duration of the recording is at least two-times the wavelength of the lowest frequency component. Accordingly, the minimum duration for the assessment of the high frequency (HF) component (0.15Hz) would be 13.3 seconds and for the low frequency (LF) component (0.04Hz) 50 seconds. However, it is generally recommended to have minimum duration recordings of 5 minutes or even better 10 minutes. For the study of circadian variations, Holter recordings (24-hour) covering a full day/night cycle are needed. Also, as will be in sections 2.1 and 2.2, many HRV indices depend upon the duration of the recording. Thus, it is inappropriate to compare HRV indices obtained from recordings of different duration with each other.

While laboratory conditions may be closely controlled, artefacts are present in almost all Holter recordings or telemetry recordings as obtained in the field. These signals are analogue/digital converted

for computer processing. In order to have a good time resolution and event definition, a sampling rate of at least 250Hz and up to 1000Hz (giving a time resolution of 1ms) is recommended.

The second step is the recognition of the QRS complex. Peak detection is often performed with commercially available software included in the Holter analysis systems. An algorithm was developed in-house for threshold detection.[16] This algorithm functions as well on the ECG as on the blood pressure recordings. The result is a discrete, unevenly spaced time event series: the tachogram, obtained from the ECG. It is crucial that before processing, these signals are corrected for ectopic and missed beats.[11-17] This is performed with filtering (elimination of spurious peaks) and interpolation algorithms (i.e. replacing beats to be corrected by the mean of a combination of preceding and following beats).[18] After this step, a normal-to-normal interval (NN) is obtained.

A final step is needed before spectral analysis can be performed. Computation of the spectral components of the tachogram requires a signal sampled at regular intervals, which is not the case for the

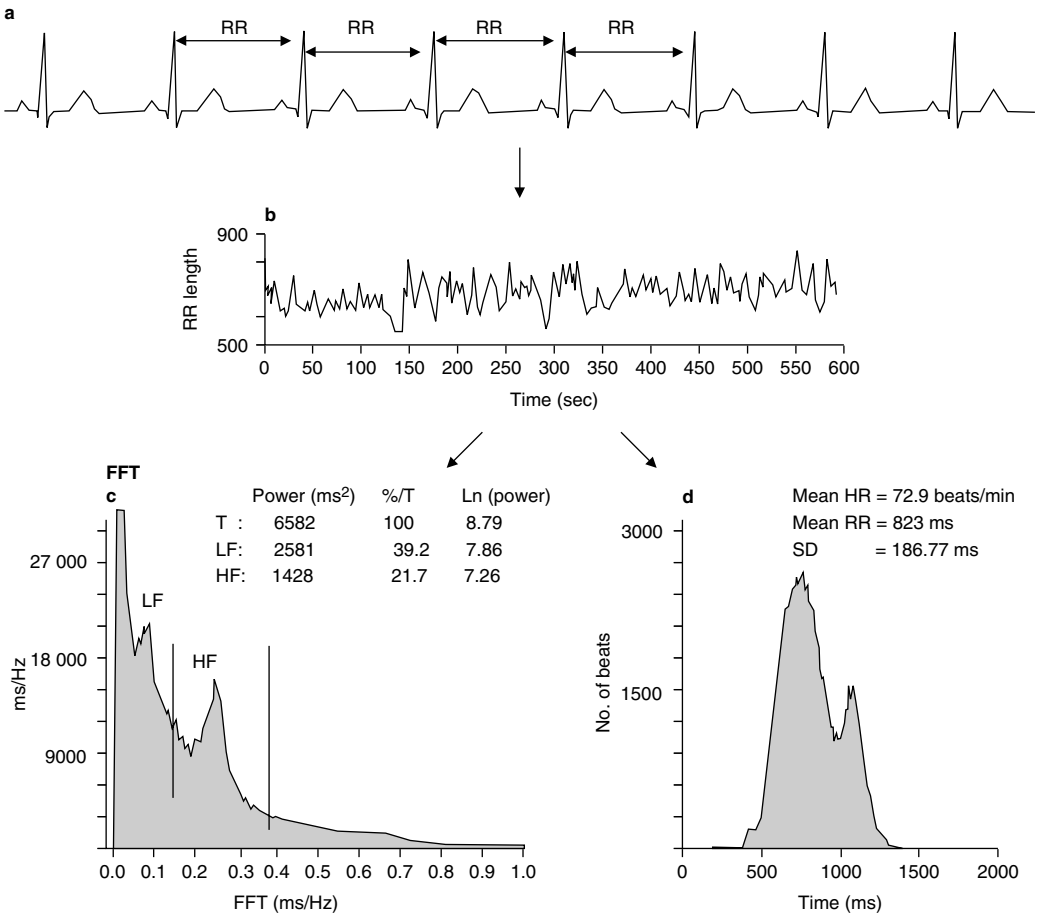

Fig. 2. Analysis of heart rate variability. Calculation of consecutive RR intervals (**a**) on the ECG, results in the tachogram (**b**) that can be analysed in the frequency domain (**c**) and the time domain (**d**). The spectral analysis (**c**) and the histogram (**d**) are results from a 24-hour Holter recording. The histogram shows two peaks: one is around 1100ms, which corresponds to mean heart rate at night, and the other is around 750ms, which corresponds to mean heart rate during the day. **FFT** = fast Fourier transform; **HF** = high frequency; **HR** = heart rate; **LF** = low frequency; **Ln** = natural logarithm; **T** = total.

Table I. Heart rate variability parameters in the time domain obtained from ten control (sedentary) individuals and ten aerobically-trained athletes. Values are mean ± standard deviation (reproduced from Aubert et al.,[27] with permission)

	Mean NN (ms)	SDNN (ms)	rMSSD (ms)	pNN50 (%)
Supine				
Control	880.7 ± 263.8	69.7 ± 37	45.5 ± 26.8	21.8 ± 19.7
Aerobic	1100.3 ± 158.5*	97.9 ± 15.7*	73.5 ± 23.7*	40.1 ± 16.6*
Standing				
Control	749.7 ± 165.6	65.4 ± 38.9	30.6 ± 16.9	10.5 ± 12.4
Aerobic	947.7 ± 108.8	92.9 ± 30.9	47.2 ± 11.1*	22.4 ± 8.9*

NN = normal-to-normal interval; **pNN50** = percentage of successive interval differences larger than 50ms; **rMSSD** = square root of the mean squared successive differences between adjacent RR intervals; **SDNN** = standard deviation of the NN interval; * $p < 0.05$.

tachogram, sampled by each (variable) heartbeat. A regular signal is obtained by modifying the tachogram. An interpolation is performed and, on this last signal, equidistant points are sampled every 0.5 seconds. Different algorithms have been proposed to achieve equidistant sampling.[18-20]

Non-invasive blood pressure can be measured using finger cuffs[21,22] or a pulse displacement device.[23] Both methods allow continuous recording of blood pressure and can be calibrated with a conventional arm cuff device. The analysis of blood pressure signals is very similar, therefore, a separate description will not be given. The only supplementary differences are: (i) maxima (systolic blood pressure values) and minima (diastolic blood pressure values) should also be detected; and (ii) on the contrary to the QRS peak where only the timing of its occurrence has to be recorded, here both coordinates (amplitude in mm Hg, and timing in seconds) have to be recorded. The variations in systolic blood pressure lead to the systogram and the variations in diastolic blood pressure to the diastogram.

Data analysis on all these graphs can be approached from different viewpoints, accentuating different underlying physiological mechanisms. Traditionally, the time and frequency domains have been considered, and recently non-linear dynamics methods have also been added.

2.1 Time Domain

Parameters in the time domain are easily computed with simple statistical methods, even from short time frames. Their main limitation is the lack of discrimination between the activity of the different autonomic branches.

Recommendations for a standardisation of valid parameters have been published.[24] These parameters are highly correlated to HF power in the frequency domain and represent markers for vagal modulation[25] (as will be explained in section 2.3). The definitions for the most frequently used time domain parameters are listed as follows:

- Standard deviation (SD) of the NN interval (SDNN) [ms] over the recorded time interval (result from corrected signals for ectopic and missed beats by filtering and interpolation algorithms). Theoretically, heart rate variance, equal to $(SDNN)^2$ and total power, are mathematically identical. In practical terms, however, correspondence between SDNN and the total spectral power depends on data processing, e.g. treatment of ectopic beats, interpolation, definition of total power.[26] SDNN depends largely on the duration of the recording; therefore, SDNN values from recordings of different duration should not be compared.
- Standard deviation of the 5-minute mean NN interval (SDANN) [ms] over the entire recording. As SDANN values are obtained from successive short 5-minute periods, it can only estimate changes in heart rate caused by cycles shorter than 5 minutes. Previous indices can be obtained from statistical methods such as shown in the histogram in figure 2d. It provides mean values, standard deviation, coefficient of variation and related parameters.

- The square root of the mean squared successive differences between adjacent RR intervals rMSSD (ms) over the entire recording.
- The percentage of successive interval differences larger than 50ms (pNN50) [%]: computed over the entire recording.

Some typical values of previously mentioned parameters are shown in table I. It gives values for mean NN, SDNN, rMSSD and pNN50, obtained from a control group of ten individuals and ten aerobically-trained athletes[27] in supine and standing position. Aerobically-trained athletes show a higher NN (lower heart rate) compared with the control group, and higher rMSSD and pNN50 in supine as well as in standing position. Also, rMSSD and pNN50 are significantly larger ($p < 0.05$) in supine compared with standing position. This corresponds to a larger HF modulation in supine position compared with standing (more vagal modulation) as will be discussed in section 2.3 (also see figure 3).

Another possibility to process RR intervals in the time domain is the use of geometrical methods.[28] The simplest one is the sample histogram (figure 2d), of which parameters related to the distribution can be calculated: mode (value that occurs most often), skewness (a measure of symmetry) and kurtosis (a measure of peakedness). Lorenz or Poincaré maps plot the duration of each RR interval against the duration of the immediately preceding RR interval. The practical use of the geometrical methods seems to be rather limited and up to now, not so often used in the literature.

2.2 Frequency Analysis

By definition, spectral analysis decomposes any steady, stationary, fluctuating time-dependent signal into its sinusoidal components. It allows plotting the power of each such component as a function of its frequency and the computation of the power in defined frequency regions. Power spectral analysis has been performed by fast Fourier transform (FFT),[29] by autoregressive (AR) modelling[30] and by wavelet decomposition.[31]

2.2.1 Fast Fourier Transform Approach

The FFT method is an objective method because no information is lost: the tachogram can be shown in the frequency domain after FFT and the latter signal can be backward transformed to retrieve the original tachogram. Units of the spectral components are: ms^2/Hz for HRV and $mm\ Hg^2/Hz$ for BPV. The advantage of the classical FFT approach consists mainly in its computational efficiency and its simple implementation (figure 2c). However, these advantages are counterbalanced by some limitations. These are mainly related to the limited frequency resolution,[18] which is directly related to the duration of the recording period (which also determines the lower limit of the spectrum, the latter equals the inverse of the recording length) which is affected by the windowing process as well. The upper frequency limit (1Hz in humans) is imposed by the Nyquist criterion: it equals half the sampling rate, which in the case of resampling the signal every 0.5 seconds corresponds to 2Hz. Therefore, the upper frequency limit is at 1Hz.

The main reason why FFT analysis is so popular in the scientific community is that it is relatively simple to apply, gives a nice graphical representation and is readily available for application on computers; it is even used for analysis of running velocity.[32]

2.2.2 Autoregressive Modelling

This approach considers the time series as a difference equation, such that the signal at every time step is expressed as a linear function of its values at J (the order of the parametric model) previous time steps. Therefore, the AR model requires an *a priori* choice of the value of J to provide the best fit to the data that are being processed. Visually, the AR spectrum presents smoother spectral components, which can be distinguished independently of pre-selected frequency bands.[30] The power content in these peaks can be calculated without the need for predefined spectral bands.

The limitations of this method are linked with the adequacy of the choice of the order J, which may affect the accuracy of the determination of the time series and the power spectra. The model order J,

even if selected objectively by information theory criteria, importantly determines both centre frequency and the magnitude of the spectral components.[33]

2.2.3 Wavelet Decomposition

Wavelet transform (WT),[31-34] a relatively recent development, provides a general signal processing technique that can be used in numerous biomedical applications. Its development was originally motivated by the desire to overcome the drawbacks of traditional Fourier analysis (e.g. FFT), simultaneously providing time and frequency information of the signal. The WT indicates which frequencies occur at what time, showing good time resolution at high frequencies and good frequency resolution at low frequencies. Therefore, this multi-resolution joint time-frequency analysis is suited for the examination of non-stationary signals. Real signals, like an ECG or a tachogram, are mostly non-stationary. The information obtained by the wavelet decomposition can be used to compare differences in power or standard deviations at each of the wavelet levels analysed.

WT offers superior time resolution and time localisation compared with FFT or AR models. Also, WT analysis is not restricted to stationary signals. The advantage of WT over AR modelling is that no assumptions have to be made about model parameters. It offers rapid frequency decomposition with time resolution, useful when one is interested in a particular power spectral band over time, and has a potential use to assess fractal characteristics.

A limitation of the method consists in the choice of the basic wavelet function (the mother wavelet), which has to possess some specific properties. Furthermore, the WT results in coefficients, which have to be related to power in specific frequency bands.

The previously mentioned frequency analysis methods are compared in figure 4. Both FFT and AR models provide very comparable results, with AR models providing a smoother spectral shape. It also allows decomposition of the spectrum (division of the spectrum in its root components) without the need for predefined spectral bands.

In the same figure (figure 4b), power bands obtained from FFT and from WT are compared between the same two groups (control group and aerobically-trained athletes) as described in table I. Two conclusions can be drawn from this figure: (i) FFT and WT provide very comparable results; and (ii) aerobically-trained athletes, with a low resting heart

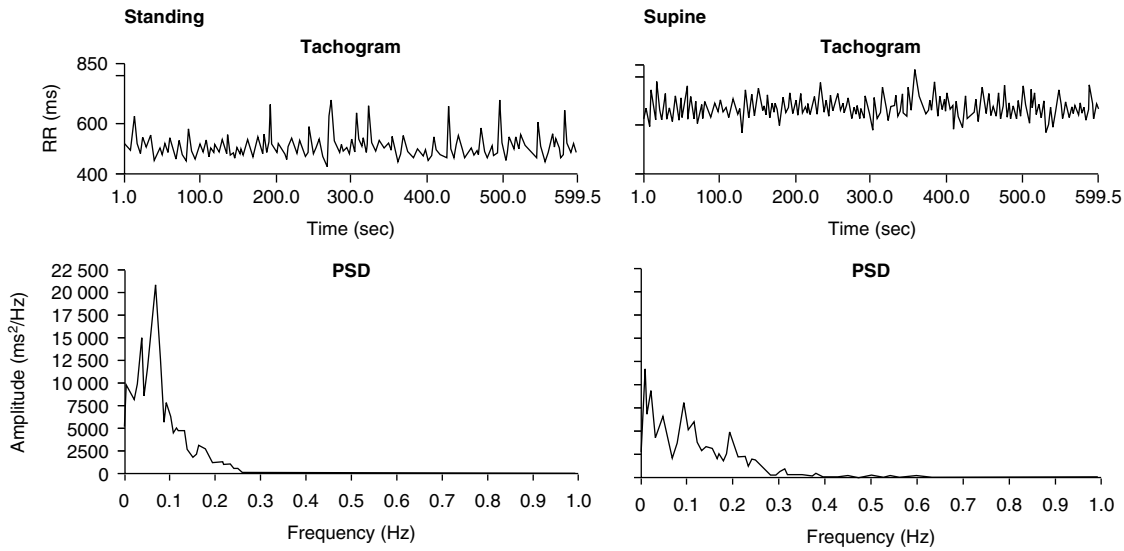

Fig. 3. Tachogram and corresponding power spectral density (PSD) of a standing individual (left) and a supine individual (right). Heart rate rises from supine to standing (RR intervals become shorter) and high frequency power (parasympathetic) is depressed compared with supine, whereas low frequency power (partially sympathetic) increases.

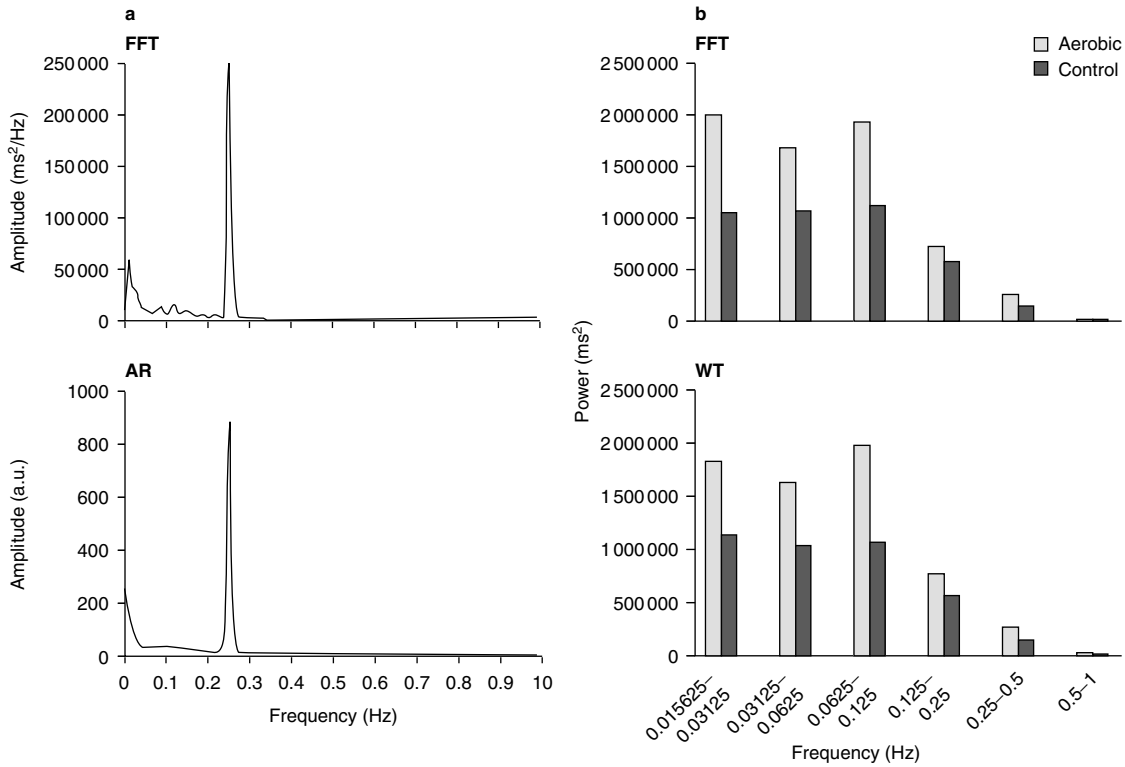

Fig. 4. (**a**) Comparison of spectral analysis methods. Upper panel shows FFT, lower panel shows AR (order is 24). Peaks (due to respiration at fixed rate) are at the same frequency, but the AR signal is smoother than the FFT signal. (**b**) Comparison of power bands as obtained from FFT (upper panel) and WT (lower panel). Control measurements were from ten (sedentary) control individuals, and aerobic measurements were from ten aerobically-trained athletes. Recordings were obtained in the supine position (reproduced from Verlinde et al.,[31] with permission). **AR** = autoregressive model; **a.u.** = arbitrary unit; **FFT** = fast Fourier transform; **WT** = wavelet transform.

rate, have indications of increased power in all frequency bands compared with the control (sedentary) group. This implies an increased modulation of heart rate by the ANS, especially of the parasympathetic component.

2.3 Selection of the Most Relevant Frequency Ranges and Physiological Significance

The power spectrum of the HRV signal, as obtained from spectral analysis (FFT, AR modelling or WT), was proposed to be used as a quantitative probe to assess cardiovascular control mechanisms.[14]

In a typical heart rate power spectral density (which is the integral of the amplitude-frequency curve and is expressed in ms^2 for HRV and in $mmHg^2$ for BPV) three main frequency bands can be observed: very low frequency (VLF), LF and HF components (figure 2c). Power in the LF and HF bands can also be expressed in normalised units: LFnu and Hfnu. These are the values of LF and HF divided by the total power minus VLF and multiplied by 100 (expressed as a percentage). The distribution of the power and the central frequency of these components are not fixed but may vary in relation to changes in autonomic modulation of heart rate and blood pressure.[17] In humans, the spectral components are usually integrated over two frequency regions defined as LF (0.04–0.15Hz, with a central frequency around 0.1Hz) and HF (0.15–0.4Hz, with a central frequency at the respiratory rate around 0.25Hz). The LF and HF bands are

indicated in figure 2c. In other mammals, these regions are differently chosen according to the heart rate of the specific species.[18]

Which neural mechanisms are underlying these spectral bands fluctuations? Parasympathetic efferent activity was considered responsible for HF, i.e. respiration-linked oscillation of HRV. This statement was made in conclusion after experiments with vagotomy performed in experiments on decerebrate cats,[35] or after muscarinic receptor blockade in conscious dogs[36] and in humans.[37] Both parasympathetic and sympathetic outflows were considered to determine LF, together with other regulatory mechanisms such as the renin-angiotensin system and baroreflex.[37,38] The LF/HF ratio can assess the fractional distribution of power,[39] although like any ratio, it can emphasise the opposite changes.

Below the LF frequency range (referred to as VLF), there is often a continuous increase in power. In part, this is the expression of very slow frequency oscillations, probably related to thermoregulation, but also non-harmonic direct current noise and the windowing process. These rhythms cannot be satisfactorily resolved and quantified by the traditional spectral analysis methods that are performed on short recordings (of the order of minutes). Different techniques and specific methodologies have to be applied for a correct understanding and quantification of these complex and not yet fully clarified mechanisms. Spectral analysis of 24-hour traces provides information down to 10^{-5}Hz and shows a circadian pattern. The long-term power spectrum of heart rate[34,40-42] seems to display a 1/f shaped frequency dependence (with a slope around –1 in humans), raising the question whether the cardiovascular control mechanism is of fractal nature.

A simple autonomic provocation consists in an active change of posture from supine (figure 3 right) to standing (figure 3 left) [see also table I]. This results in a shift of blood away from the chest to the venous system below the diaphragm, usually referred to as venous pooling. Almost invariably in all healthy volunteers, an increase in heart rate is the result (from mean value of 85 beats/min supine to 120 beats/min standing in figure 3). While standing, the regulatory system increases heart rate, cardiac contractility and vascular tone by a decrease in parasympathetic outflow and an increase in sympathetic outflow. The latter increase is reflected in the LF content of the power spectral density (figure 3 left) and the former decrease in the HF content. While being supine, there is a parasympathetic predominance, which switches to sympathetic predominance on standing.

2.4 Non-Linear Methods

Chaotic behaviour exhibits a number of characteristics that distinguish it from periodic and random behaviour,[43,44] i.e. HRV spectra show a broad band noise-like variability over a large frequency span.[45,46] This seems to be due to non-linearity in the cardiovascular control network. The long-term regulation of heart rate contains both short-time periodic (e.g. respiratory) modulations and entirely non-periodic fluctuations. There are indications that a reduction in complexity comes along with a decrease in parasympathetic activity, suggesting that a considerable amount of non-linear behaviour be provided by this branch of the ANS. Methods of non-linear dynamics define parameters that quantify complicated interactions of independent and interrelated components, which can be described as 'complexity measures'.[47-49]

Non-linear dynamical methods have made their appearance in the analysis of HRV only recently and methods have still to be established. Methods related to the chaos theory are used to describe the non-linear properties of heart rate fluctuations (attractors, 1/f behaviour of the power spectrum, fractal dimension[50,51] and correlation dimension,[52] Poincaré- and higher order moment plots, approximate entropy,[53] pointwise correlation dimension, detrended fluctuation analysis,[48] and Lyapunov exponents[54]).

The use of the new methods from non-linear dynamics for HRV analysis may provide a more sensitive way to characterise function or dysfunction of the control mechanism of the cardiovascular system. These tools are promising with regard to the understanding of the latter mechanism, but are still

under development and evaluation. Moreover, these methods require more powerful computing and are less visually attractive compared with frequency analysis.

2.5 Baroreflex Sensitivity

Evaluation of RR interval changes corresponding to aorta blood pressure variations, allow assessment of the activity of the baroreceptive mechanism.[55] Results from combined HRV and BPV signal analysis lead to different methods that relate to the baroreflex mechanism. The enormous complexity of baroreflex interactions has been extensively reviewed recently.[56]

Several methods have been described to study arterial baroreflex activity. The majority of the methods depend on pharmacological or physiological manoeuvres that produce an abrupt increase or decrease in blood pressure.[57] Subsequently, quantification of the (linear) relation between blood pressure and corresponding heart rate changes is performed by calculation of the slope of the fitted linear curve.[58] With standing or passive tilt, transient hypotension occurs that results in a reflex increase in heart rate, whereas the post-Valsalva increase in blood pressure causes reflex slowing.[59] Manoeuvres like neck suction or neck pressure that alter the transmural pressure or stretch in the carotid sinus also can be used in humans to activate (load) or deactivate (unload) arterial baroreceptor reflexes.[60] Drugs such as α-adrenergic agents (phenylephrine) or angiotensin II that increase blood pressure produce reflex slowing of the heart rate, whereas drugs such as nitrates or sodium nitroprusside that lower blood pressure directly by relaxing vascular smooth muscle, augment sympathetic efferent nerve activity and cause tachycardia and an increase in cardiac contractility. A high slope of the regression line is interpreted as indicating the presence of strong vagal reflexes, while a relatively flat slope indicates the presence of weak vagal reflexes, possibly associated with high reflex sympathetic activity.[61]

The usefulness and constraints of traditionally used methods have been reviewed elsewhere.[62] Some investigators have even viewed the traditional drug-induced baroreflex as misleading.[63] Recently, several methods have been developed to quantify spontaneous BRS. Some are based on the use of the spectral analysis of both RR and BPV variabilities (α-index),[64] on the analysis of sequences of concurrent alterations in BP and HR (sequence method),[65] or on the method of statistical dependence.[66-68] The spontaneous BRS has a number of important advantages: it does not require the use of intravenous drugs or a neck chamber apparatus, and it measures BRS in the normal physiological range over a period of time rather than brief and extreme perturbations as induced by other methods. In this respect, it represents a true steady-state assessment of the cardiac baroreflex under stationary conditions.

It is out of the scope of this review, but suffice it to mention that HRV methods have many physiological and clinical applications studying the influence of: ageing and sex studies;[69,70] anxiety, stress[71] and depression;[72,73] smoking;[74,75] caffeine[76,77] and alcohol consumption;[78,79] risk assessment after myocardial infarction[80] or predictor of mortality;[81,82] haemodialysis;[83] congestive heart failure[84]and heart transplant patients;[85] diabetes;[86] hypertension;[87] drug testing;[88,89] sudden infant death syndrome;[90] influence of gravity;[91,92] exercise training in patients after coronary artery disease[93] or heart transplantation.[94,95]

All the HRV and BPV analysis methods described in section 2 have been implemented in appropriate algorithms in our laboratory and software was accordingly developed in-house. All programs were implemented in LabVIEW, (which is a graphical language) and variability parameters determined according to the standards provided in the Task Force on HRV[24] and extensively tested and validated.[16,18,26,48,53,68]

3. Exercise Physiology Aspects as Related to HRV

The cardiovascular adjustments in exercise represent a combination and integration of neural and local chemical factors. The neural factors consist of: (i) central command; (ii) reflexes originating in the

contracting muscle; and (iii) the baroreflex. Central command is the cerebrocortical activation of the sympathetic nervous system that produces cardiac acceleration, increased myocardial contractile force and peripheral vasoconstriction. When exercise stops, an abrupt decrease in heart rate and cardiac output occurs and the sympathetic drive to the heart is essentially removed. Blood pressure will be stabilised by the baroreflex and parasympathetic activity will be enhanced.

3.1 General Cardiovascular Changes Due to Exercise

Physical activity is associated with haemodynamic changes and alters the loading conditions of the heart.[96] Cardiovascular responses to physical activity depend on the type and intensity of exercise. The main difference, at the heart level, is the increased volume load during endurance exercise in contrast to pressure load during strength exercises.[97] These differences in loading will cause various cardiovascular responses to physical activity. After long-term athletic training, left ventricular diastolic cavity dimensions, wall thickness and mass will increase.[4,5] These changes are described as the 'athlete's heart'. However, compared with males, female athletes show smaller left ventricular mass.[98] This sex difference has been associated with a lower systolic blood pressure during 24-hour Holter recordings and during exercise in female athletes.[99]

The volume load during endurance training results in adaptive changes in many aspects of cardiovascular function.[2] The heart improves its ability to pump blood, mainly by increasing its stroke volume, which occurs because of an increase in end-diastolic volume and a small increase in left ventricular mass. In contrast, strength training results in larger increases in left ventricular mass. There is little or no change in ventricular volume. Endurance exercise also decreases the metabolic load on the heart at rest and at any submaximal exercise intensity. It does so by increasing stroke volume and decreasing heart rate. The result is a more efficient pressure-time relationship.

Heart rate is the predominant mechanism by which cardiac output rises during exercise under physiological circumstances.[100] Tachycardia can occur either by neural stimulation or by an elevation in circulating catecholamines.[101] Increased heart volume and contractility will lead to higher values of stroke volume during rest as well as during submaximal and maximal exercise. Also, the lower heart rate will increase stroke volume because of longer periods of diastole. The heart ejects the extra blood due to the Frank-Starling mechanism.[2] Another factor inducing higher stroke volume is the larger blood volume in athletes.[102]

Endurance training reduces resting and submaximal exercise systolic, diastolic and mean arterial blood pressures.[103] The mechanism of reduced blood pressure at rest is not known. Endurance training will also influence the release of catecholamines. Norepinephrine is released by the sympathetic nerve processes. An endurance training programme will result in less catecholamine response to submaximal exercise but not to maximal exercise.[104]

3.2 Exercise and the ANS

Heart rate is generally regulated predominantly by the ANS.[7] The two major efferent mechanisms by which tachycardia occurs are either through a decrease in parasympathetic or through an increase in sympathetic stimulation.[6] The latter can occur either by neural stimulation or by an elevation in circulating catecholamines. The mechanism of the (exercise-induced) tachycardia appears to involve parasympathetic and spinal sympathetic reflex circuits (Brainbridge reflex). The latter mechanism is important to mention, since stimulation of cardiovascular sympathetic afferent fibres produce cardiovascular reflexes that operate through a positive feedback mechanism and thus may be particularly responsible for the increased sympatho-adrenal activity of exercise.[2] This is opposed to reflex responses initiated by baroreceptor or parasympathetic innervated cardiopulmonary receptors that operate through the negative feedback mechanisms.[7] Thus, both the sympathetic and parasympa-

thetic arms of the ANS play a pivotal role during exercise. It can therefore be expected to find changes in HRV indices according to the degree and duration of training and/or the kind of training.[105]

Long-term physical training influences cardiac rhythm. It induces sinus bradycardia in resting conditions, and a slower increase in heart rate at any degree of submaximal oxygen uptake due to a shift of the sympathovagal balance[106] towards parasympathetic dominance.[107] However, the latter point has been questioned recently[108,109] and a direct involvement of the sinus node was suggested. This point will be discussed in section 4.

Heart rate during exercise is regulated by increased sympathetic modulation and withdrawal of parasympathetic activity.[8] It varies within an individual according to heredity (size of the left ventricle; predisposition for certain sport activities), fitness level, exercise mode (endurance or static training) and skill (economy of exercise). Body posture (supine, sitting, standing[110]), environmental variables (temperature,[111] humidity, altitude[112]), state of mood[113] and hormonal status[114] also alter heart rate response. Heart rate and HRV are also affected by drugs, stimulants[77] and eating habits.

Reflex adjustments initiated by the stimulation of afferent nerve fibres from the exercising muscles are also likely to play a role in the cardiovascular response to exercise.[115] There is evidence that reflex cardiovascular adjustments originating in the contracting muscles are not mediated by muscle spindle afferents but rather by small myelinated and unmyelinated afferent fibres.[116]

Since exercise is accompanied by major cardiovascular alterations, including marked tachycardia, increases in cardiac output and in arterial and atrial pressures, and a reduction in total peripheral resistance, it could be expected that a cardiovascular regulating mechanism as important as the arterial baroreceptor reflex would play a significant role in mediating and modifying the exercise response.[117] Investigations into the role of the arterial baroreflex in the control of the cardiovascular system during exercise have yielded conflicting conclusions as to their importance.[118] At first it was suggested that the baroreflex is just as active during exercise as at rest. On the other hand, if the baroreflex was also important during exercise, then the occurrence of tachycardia associated with an elevated pressure is opposite to the predicted response, since the baroreceptor should act to restrain heart rate in the face of an elevated pressure.[119]

There is now a large body of evidence suggesting the lack of importance of the baroreflex during exercise[120,121] (a similar response to moderate exercise in intact dogs and arterial baroreceptor denervation[122]).

In reality, cardiovascular control mechanisms are much more complex (see figure 1) as was recently shown in a review by Malpas.[123] Stroke volume and end-diastolic volume also contribute[13] in an intricate feedback system.

Taking all these considerations together concerning HRV and its relationship to training, some questions still remain unanswered: (i) are differences in ANS control of the cardiovascular system between trained athletes and a sedentary population due to a training effect or are other factors involved?; and (ii) can cardiovascular variability (HRV and BPV) parameters be used as a predictive factor for athletic achievements, or in other words, can HRV and BPV be used to predict optimal training and athletic performance?

4. Changes in HRV Related to Exercise Training

Highly-trained athletes have a lower resting heart rate than sedentary controls.[3] Anticipation of physical activity inhibits the vagal nerve impulses to the heart and increases sympathetic discharge.[124,125] The concerted inhibition of parasympathetic control areas and activation of sympathetic control areas in the medulla oblongata elicit an increase in heart rate and myocardial contractility.

Technically, a problem arises for heart rate measurements during exercise: as it is increasing according to the intensity of exercising, no steady state is obtained, which is necessary for spectral analysis. Two approaches are usually proposed in the literature to solve this problem: (i) perform measurements

Table II. Heart rate variability during exercise

Study	n	Age (y)	Sympathetic	Parasympathetic	Comments
Arai et al.[127]	43	25–69	No change	Withdrawal	FFT
Brenner et al.[130]			Increase at onset	Withdrawal	FFT
			Later on attenuated increase due to higher temperature		Review
Kamath et al.[131]	19	20–32	Decrease		AR
Maciel et al.[132]	23	25–35	No change	Withdrawal	Pharmacological
			Increase at higher activity level		Blockade
Perini et al.[126]	7	23.7 ± 0.5[a]	No change at low intensity; decrease at higher	No change at low intensity	AR
Shin et al.[133,134]	5	17–21	Decrease	Decrease	AR
	8	21–40	Decrease	Decrease	Non-athletes

a Mean ± SD.

AR = autoregressive model; **FFT** = fast Fourier transform.

at a fixed intensity level;[126] and (ii) subtract a background trend to decrease the contribution of the continuous increase in heart rate with increasing exercise intensity.[127] The latter method is based on the fact that the linear trend (first order) represents the largest non-stationarity of heart rate during and after exercise. Normally one is also only interested in resolving spectral components in the range where baroreflex and respiratory inputs are the dominant effectors of heart rate fluctuations (higher then 0.03Hz). During exercise, sometimes an exponential trend is subtracted.

ECG and/or blood pressure recordings before or after exercise cause no particular problems. Best practice is to perform these measurements in a quiet surrounding, at comparable timings for all individuals in order to avoid circadian variations among individuals. Usually, ECG is recorded as in clinical practice, sometimes only RR intervals are stored with a wrist watch. For particular studies, requiring day/night resolution or circadian variations, 2-hour Holter recordings are used.

4.1 HRV During Exercise

It has long been shown that during dynamic exercise, heart rate increases due to both a parasympathetic withdrawal and an augmented sympathetic activity.[128,129] The relative role of the two drives depends on the exercise intensity.[121-126]

Arai et al.[127] were the first to test this hypothesis with the aid of Fourier spectrum analysis of heart rate time series in 43 healthy volunteers (aged from 25–69 years) who exercised until peak level. Their data (table II) support a progressive withdrawal of parasympathetic activity during exercise but no changes in normalised values of LF and HF with respect to rest and no correlation between LF power and sympathetic activity have been observed during muscular exercise.

Maciel et al.[132] came to similar conclusions. They performed a bicycle ergometer test in a group of 23 untrained individuals at three levels (25W, 50W and 100W), before and after blockade with atropine or propranolol. Their results showed that tachycardia induced by dynamic exercise is mediated by a biphasic mechanism initially depending on rapid vagal release and an increased sympathetic activity, especially at higher levels of exercising. In a recent review article, Brenner et al.[130] also supports this hypothesis: at the onset of exercise, heart rate is increased by a reduction in parasympathetic activity and a temporary increase in sympathetic tone. A continuation of physical activity is associated with a continued withdrawal of vagal activity and an attenuation of sympathetic nervous system tone.

In contrast to Arai et al.[127] and other previously mentioned authors, Perini et al.[126] performed power spectral analysis (with AR modelling) during steady-state exercise at different intensities (three

levels: low at 50W, medium at 100W and high at 150W) and during the corresponding recovery periods in seven sedentary young males (aged 23.7 ± 0.9 years). They found only at low exercise intensities no changes in the relative power of the three components with respect to rest. Above 30% maximal oxygen uptake ($\dot{V}O_{2max}$), a marked decrease in LF normalised power coupled to an increase in VLF% was found. Their hypothesis was that above this threshold, additional mechanisms were involved in cardiovascular adjustment and that a not negligible portion of the power of HRV was in the VLF band. This component might reflect, at least in part, the sympathetic activity. However, they also mentioned a technical problem with the VLF detection after trend removal. Therefore, conclusions about this component are maybe not entirely justified.

Warren et al.[135] and Cottin et al.[136] also concluded that HRV is a valid technique for non-invasive measurement of parasympathetic activity during exercise, but its validity as a measure of sympathetic activity during exercise is equivocal. The former concluded this from measurements during exercise (progressive cycling tests at 40%, 60% and 80% of each individual's heart rate reserve) with infusion of saline, esmolol (β_1-blocker), glycopyrrolate (muscarinic blocker) or both drugs. HF power decreased exponentially with workload and was attenuated by glycopyrrolate and combined treatments. The latter group showed spectral analysis to confirm withdrawal of parasympathetic control during graded exercise load (25%, 50% and 75% of $\dot{V}O_{2max}$), as the power spectral density of the HF band significantly decreased with exercise loads. However, the LF power also decreased with exercise load, suggesting that LF and LF/HF is not a good indicator of cardiovascular modulation during exercise.

Kamath et al.[131] compared orthostatic stress (10 minutes supine followed by 10 minutes standing) and exercising on a cycle ergometer (at 50% of their maximum predicted power output) in a group of 19 healthy untrained individuals (16 males and three females aged 20–32 years). They found the same significant decrease in the LF component due to exercise, but an enhanced LF during orthostatic stress. Therefore, they concluded that humoral factors, such as circulating catecholamines, probably play a more dominant role in maintaining the tachycardia during exercise instead of neurogenic control, which takes place during orthostatic stress. The existence of a non-neural mechanism in the reduction of the HF component was also supported by a study from Casadei et al.[137]

Shin et al.[133,134] submitted five runners (18 ± 2 years) and eight sedentary individuals (27 ± 7 years) to a bicycle ergometer exercise to the point of exhaustion. They found that LF and HF gradually decreased with exercise intensity in both athletes and non-athletes. They suggested two possible reasons: a marked absence of vagal modulation may have led to reductions in LF accompanied by an influence on the baroreflex (restored at higher operating point or turned off), or hormonal factors. Possible limitations of this study are: (i) the choice of order for the AR modelling, which influences power distribution over different bands; (ii) the small number of athletes (n = 4), rather young compared with (iii) an older non-athletes[138] population.

Yamamoto et al.[139] found an increase of LF component with increasing exercise intensity. In their study, six healthy male volunteers performed an incremental exercise test on an electrically braked cycle ergometer, consisting of a 5-minute warm-up period at 50W, followed by work rate increment in a ramp fashion until exhaustion. These authors used 0.0–0.15Hz as limits for the low frequency bands, therefore, we cannot interpret these data compared with previous data, because their LF component also involves the VLF component as proposed by the Task Force.[24]

Parasympathetic activity of heart rate during exercise was investigated with a time-series analysis by way of geometrical methods (Poincaré plot) in a study in 31 individuals by Tulppo et al.[140] They showed that during recovery, parasympathetic activity decreased progressively until the ventilatory threshold level was reached, when sympathetic activation was reflected from changes in the Poincaré plot. They concluded that poor physical fitness is

associated with an impairment of cardiac parasympathetic function during exercise and that their data support the concept that good aerobic fitness may exert cardioprotective effects by enhancing the cardiac parasympathetic function during exercise.

A totally different technique to analyse heart rate variability during exercise is proposed by Anosov et al.[141] They examined a group of 22 untrained individuals (13 females, nine males; 20–40 years) on a cycle ergometer with a ramp load until exhaustion. The authors were only interested in the HF component. Therefore, the tachogram was high-pass filtered with a LF cut-off at 0.15Hz. As ramp loading leads to non-stationary time series, Fourier analysis was not applicable. To obtain the instantaneous frequency of the HF component of the HRV, the analytic signal approach was used. This method consists in constructing a complex function (the analytic function) where the real part is the time series and the complex part is the Hilbert transform of previous time series. From this complex function, the amplitude and phase of the time series can be obtained, and finally the instantaneous frequency is the derivative of the instantaneous phase. They concluded that the instantaneous frequency component of the HF power of HRV and of the respiratory signal developed in parallel during a ramp load test. Both signals were closely linked, showing a strong correlation between respiration and heart rate. Due to this correlation, the HF component of HRV was modified during ramp load and in most cases can be used for the detection of the ventilatory anaerobic threshold, because the shift in instantaneous frequency of the HF component occurred during the transition from aerobic to anaerobic work. The modulation of HRV in terms of its frequency is strong, even at high physical activity levels, whereas the absolute power of HRV is clearly reduced at high work loads.

Gonzalez-Camarena et al.[142] compared heart rate and blood pressure variabilities during static and dynamic (cycling at 30% and 60% of $\dot{V}O_{2max}$) exercise in ten individuals. They found a parasympathetic withdrawal and sympathetic augmentation during dynamic exercise and an overall increase in HRV indices during static exercise, suggesting an increased activity of both autonomic branches.

HRV analysis during exercise remains a problem. There are not so many studies and almost all of them mention the technical problem of not dealing with stationary time series. There is also a problem related to the interpretation due to the methodology. There are nearly as many protocols proposed as there are papers written on this topic. Methodology differs widely especially concerning training intensity and/or exercise intensity, even in some papers they are only vaguely mentioned. Therefore, it is strongly recommended to establish a protocol, one for performing studies in sedentary populations and another for athletes, while using appropriate blockade mechanisms and different analysis methods: time domain (and geometric methods), spectral analysis and its variations (Hilbert transform), and nonlinear methods.

4.2 Cross-Sectional Studies: Comparison of Athletic and Sedentary Groups

In this section, the differences between a sedentary group and one or more groups of athletes (table III) will be discussed as described in the literature.

Tonkins[148] reports a positive effect of time domain parameters, as obtained from Holter recordings in 39 trained athletes but did not find a difference between aerobically- and anaerobically-trained athletes. This is in contrast with results of Aubert et al.[27] They found significantly higher values of rMMSD and pNN50 between aerobically- and anaerobically-trained athletes or rugby players, the latter are involved in combined aerobic and anaerobic training. These differences were also found in the frequency spectrum: larger HF component in aerobically-trained athletes demonstrated with FFT and wavelet analysis.[31] In an earlier study[149] they came to the same conclusion: significantly higher rMSSD in 14 middle-aged athletes, compared with a sedentary age-matched population (n = 14; 35–55 years). Many other studies confirm these findings for young endurance-trained athletes (disciplines: cycling, canoeing, athletics, roller-skating, volleyball; mean age less than 30 years).[144,146,150-153]

Table III. Cross-sectional studies: athletes versus sedentary population

Study	n	Age (y)	Spectral analysis	Comments
Aubert et al.[143]	10	18–34	↑HF	FFT
	10[a]	19–31		
Dixon et al.[144]	10	22–33	↑HF	AR
	14[a]	23–33		
Furlan et al.[145]	21	16 ± 0.6[b]	↑LF	Trained
	15	16 ± 0.5[b]	↑HF	Detrained
	29[a]	16 ± 0.4[b]		
Goldsmith et al.[146]	8	24–38	↑HF	24h Holter, FFT
	8[a]	24–38		Asleep and awake
Janssen et al.[147]	18	19–32	↓LF	Supine
	11[a]	23–33		
Tonkins[148]	39	21.2 ± 3[b]	↔	24h Holter
	39[a]			Time domain
Verlinde et al.[31]	10	18–34	↑HF	Wavelet
	10[a]	19–31		

a Sedentary comparison group.

b Mean ± SD.

AR = autoregressive model; **FFT** = fast Fourier transform; **HF** = high frequency power; **LF** = low frequency power; ↔ indicates no change; ↑ indicates increase; ↓ indicates decrease.

These studies concluded that endurance training results in the enhanced vagal tone in athletes, which may contribute in part to the lower resting heart rate. Goldsmith et al.,[146] who performed a Holter study in eight endurance-trained athletes and compared them with eight age-matched untrained men, suggests that aerobic exercise training may be a useful adjunct or alternative to drug therapy in lessening the derangements of autonomic balance in many cardiovascular diseases.

In a combined RR-interval blood pressure study, Macor et al.[152] concluded that competitive cycling caused an enhanced parasympathetic drive to the sinus node, whereas the neural control of blood pressure was not affected. Furlan et al.[145] examined two groups of endurance athletes: one group in a rest period (15 detrained athletes; six males, nine females) and one group during peak season (21 swimmers; 14 male, seven female). The latter had, in contrast with the former group, elevated sympathetic activity and higher parasympathetic activity compared with a control group. They concluded that the enhanced athletic performance resulting from long-term training might depend on an increase of both parasympathetic and sympathetic modulation. Janssen et al.[147] compared athletes (18 cyclists; 19–32 years) with 11 sedentary individuals (23–33 years) in both supine and standing position. Spectral analysis was performed with AR models. Their measurements would suggest that in the supine position, the sympathovagal balance of the athletes differed from the control values, caused by lowered sympathetic and/or increased parasympathetic tone. This is mostly due to a persistent sympathetic activation after exercise, lasting up to 24 hours, which is also studied in the same work. They concluded that the differences in autonomic control between the athletes and the control group, were reflected in the quality (balance between slow and fast heart rate fluctuations) rather than in the quantity of heart rate variability.

De Meersman[154] performed a cross-sectional study for all age groups in 72 runners (15–83 years) and in 72 sedentary controls matched for age, bodyweight, blood pressure and social status. However, HRV was not determined from spectral analysis but defined as the percentage change of heart rate with breathing (imposed breathing at 6 breaths/min). Although no correlations with spectral component were made, it can be assumed that this para-

meter is related to the HF component of HRV. The physically active group had significantly higher levels of percentage change of heart rate, when compared with their sedentary counterparts. These authors concluded that habitual aerobic exercise augments some parameters of HRV and could be a beneficial modulator of heart rate variability in an ageing population. They also suggested that this augmented HRV in physically active individuals provided further support for life-long aerobic exercise as a possible non-pharmacological cardio-protective therapy. However, this statement remains highly speculative, as it is not entirely supported by their data.

All previous studies showed an increment in parasympathetic activity due to an aerobic exercise programme. Some other studies[70,106,109,155-159] did not find this positive effect on the ANS. Migliaro et al.[70] found no differences in HRV (as determined from spectral analysis: LF and HF) parameters between sedentary (n = 29; 15–24 years) and non-sedentary (n = 29, 15–24 years) young people. They also did not observe training bradycardia, which can probably explain their observation.

A recent pharmacological blockade study by Stein et al.,[109] with atropine and propranolol, caused parallel shifts in the sinus automaticity of athletes (six runners aged 29 ± 4 years, and six non-athletes aged 28 ± 5 years). Increased parasympathetic activity would cause greater heart rate response post-atropine and a reduction in sympathetic activity would cause lesser heart rate response post-propranolol in athletes compared with non-athletes. These conclusions were obtained after electrophysiological studies of the conduction system. The authors concluded that sinus automaticity and AV node conduction changes of endurance athletes were related to intrinsic electrophysiology and not to autonomic influences. The same group suggested earlier[108] that in addition to its parasympathetic effects, athletic training might induce intrinsic adaptations in the conduction system (mostly by influencing conduction velocity), which could contribute to the higher prevalence of atrioventricular abnormalities observed in athletes.

The latter study was in agreement with the results of a blockade study of Smith et al.[160] who found greater parasympathetic influence in endurance-trained individuals as well as lower intrinsic heart rate, but in disagreement with all the studies as mentioned in the first paragraph of section 4.2 and Goldsmith et al.[161] who indicated from their results that physical fitness is strongly associated with vagal modulation. Most studies[144,151,153] mention that the higher parasympathetic activity is not the only factor that contributes to the bradycardia in athletes but that it is only a part of the lower heart rate. All of these studies point to endurance training as an effector of enhanced parasympathetic activity in athletes, which may contribute, in part, to the resting bradycardia. Katona et al.[156] already found in 1982 that lower resting heart rate in endurance trained athletes (eight world class oarsmen) is solely due to a reduction in intrinsic cardiac rate, and not to an increase in parasympathetic tone. They showed it by using pharmacological blockade (propranolol and atropine) to suppress either sympathetic or parasympathetic activity of the ANS. Also, Bonaduce et al.[162] came to the conclusion that other mechanisms than changes in cardiac autonomic control could be involved in determining the profound bradycardia of athletes.

Another possible reason for the controversial results concerning ANS activity in athletes is due to a disturbance on the LF power caused by respiration. This was shown in a study from Strano et al.[163] comparing controlled versus paced breathing. A slow breathing rate, which is a common feature in athletes, caused the HF and LF components to overlap, leading to a misinterpretation of the LF power. An ECG was recorded in the supine position in athletes, while they were breathing at their spontaneous frequency and at rates of 15, 12 and 10 to 14 (in random order) breaths/min (corresponding to 0.25, 0.2, 0.16 and 0.23Hz). Uncontrolled and random breathing rates significantly altered spectral sympathetic indices. On the other hand, 15 and 12 breaths/min redistributed respiratory related power through the HF, thus yielding correct LF power estimation. The authors conclude and recommend to standardise

Table IV. The effect of training on a sedentary population

Study	n	Age (y)	Duration (wks)	Repetitions (no./week)	TP (ms^2)	LF (ms^2)	HF (ms^2)	Comments
Catai et al.[155]	10	19–21	12	3	1821	818	277	Before/awake
					2870	1048	429	Jogging/walking 70–85% peak HR
	7	50–59	12	3	2601	687	265	Before
					2942	513	253	After
	10	19–21	12	3	4862	1030	2589	Before/asleep
					3152	930	1374	After
	7	50–59	12	3	1225	357	342	Before
					1584	502	488	After
Loimaala et al.[168]	26	35–55	20			863[a]	321[a]	Control/before
						829[a]	391[a]	After training
	26			4–6		1212[a]	572[a]	Before
						1300[a]	659[a]	Jogging/walking 55%
	28			4–6		846[a]	317[a]	Before
						1054[a]	478[a]	Jogging 75%
Melanson[158]	11	25–45	16	3		234[a]	398[a]	Before
						416[a]	798[a*]	After training
	5	25–45		No training		173[a]	331[a]	Before
						169[a]	446[a]	After 16 wks

a Values transformed from log.

HF = high frequency power; **HR** = heart rate; **LF** = low frequency power; **TP** = total power; * $p < 0.05$.

respiration at 0.25Hz (15 breaths/min) in athletes for assessing ANS activity.

A possible hypothesis as to the controversy about autonomic versus non-autonomic determinants of electrophysiological adaptations in athletes could be a fundamental difference between short- and long-term physical training programmes.[109] Short-term training, as in prospective studies, could induce autonomic adaptations, with a reduction in sympathetic activity and an increase in parasympathetic activity (leading to bradycardia). On the other hand, long-term aerobic training, eliciting atrial and ventricular dilation, would induce intrinsic electrophysiological adaptations and enhance parasympathetic activity.

4.3 Longitudinal: Effect on HRV of Exercise Training of Non-Athletes

Beneficial effects of physical training have been reported in post-myocardial patients[164,165] and in heart transplant recipients.[166] Therefore, it can hypothesised that exercise training would be effective in improving the autonomic balance in the general public while also developing physical fitness.

Melanson and Freedson showed influence of exercise training on HRV parameters on a young male population (n = 11; 25–40 years).[167] The study participants performed moderate- to vigorous-intensity stationary cycling for 3 days each week for 30 minutes per session. In their study, they showed that a moderate- to vigorous-intensity endurance training programme in adult, previously sedentary men increased markers of cardiac parasympathetic activity after 12 weeks. This was proven by a significant increase in HF power after training (table IV) and a significant increase in time domain parameters related to parasympathetic activity (pNN50 and rMSSD).

Boutcher and Stein[169] found no change in HRV in a group of 19 middle-aged men (46.2 ± 1.6 years) compared with an age-matched control group (n = 15). HRV was assessed after 24 exercise sessions of moderate intensity exercise training (during 8 weeks). The study participants exercised three times each week at an intensity of 60% of heart rate,

determined through baseline at maximal exercise heart rate. The exercise session consisted of a 400m warm-up walk, a series of stretches, an aerobic exercise period (20 minutes for the first three sessions, 15 minutes for the next three, and 30 minutes for 7–24 sessions), a 400m cool-down walk and a repeat of the stretching. LF and HF components were obtained after band pass filtering of the tachogram and variance was determined in these bands. In the exercise group, $\dot{V}O_{2max}$ increased (12% absolute value) after the training period, but without altering HRV. These results show that short duration and moderate intensity aerobic training in a middle-aged population, is insufficient to alter HRV parameters in that age group.

The same conclusion was reached by Perini et al.[170] in a training programme in an elderly population of seven men and eight women (73.9 ± 3.5 years). They reported no changes in HRV parameters after an intense 8-week aerobic training programme. However, Schuit et al.[171] found a general increase in HRV after a training programme of 6 months (three aerobic supervised training sessions per week lasting 45 minutes) in an elderly population (n = 51; 67 ± 5.1 years). They specifically showed that the VLF and LF power bands, were significantly increased compared with a control group. Their conclusion was: "In older subjects physical training may be an effective means to modify positively a factor that is associated with increased incidence of cardiac events"; however, this is questionable as LF power is associated with arrhythmogenic activity and low LF in pre-menopausal women is cardioprotective.[69]

Again, in a 5-month duration aerobic training programme in 83 middle-aged men (35–55 years), Loimaala et al.[168] found no changes in HRV parameters in both time and frequency domains (table IV). Individuals were trained four to six times a week during 30 minutes in two different groups: (i) jogging at a heart rate level corresponding to 55% of the $\dot{V}O_{2max}$ measured at baseline; and (ii) jogging at a heart rate level corresponding to 75% of the $\dot{V}O_{2max}$ measured at baseline. Indices reflecting tonic parasympathetic outflow (SDNN, pNN50 and HF power) did not change significantly during the intervention. They concluded: "exercise training was not able to modify the cardiac parasympathetic activity in sedentary, middle-aged persons".

No consistent changes were observed in BRS, although a significant reduction in heart rate was found. The authors blame the short duration of the training programme and suggest that in order to obtain any effect on HRV the training programme should last for a period of at least a year.[168]

Many factors affect the physiological significance of these studies. One of the most important is the age factor, which contributes to the discrepant findings in the literature. It is well known that HRV parameters are decreasing with age[69] (and also a function of sex). Exercise training studies in young adults[172] generally report increases in measures of HRV, whereas studies in middle-aged[169] and older adults[173] show no changes in cardiac autonomic function, as determined from HRV.

Duration and intensity of training, the accent of the programme, even sex distribution, also vary widely among different studies. In one study it was even suggested that endurance should be practiced for a prolonged period, even extending over many years[168] in a middle-aged population. On the other hand, in a young population (20–22 years) we have seen (figure 5) some influence on HF of HRV after only 6 weeks of training (unpublished data). Dynamic exercise is performed in most of the studies; however, in some studies static training is also used.[142] This again, if not taken into account, can lead to differing conclusions. A last factor is usually the small number of study participants in the training programme. Working with small numbers reduces the statistical power, making it more difficult to detect differences due to the training. Therefore, whether age or other factors would modulate the effects of training on HRV parameters is still unclear and is an area for further investigation.

Levy et al.[174] submitted an elderly (n = 13; 60–82 years) and a younger group of men (n = 11; 24–32 years) to a 6-month aerobic training programme (walking, jogging and cycling). The men trained as follows: 10 minutes warm up, 45 minutes

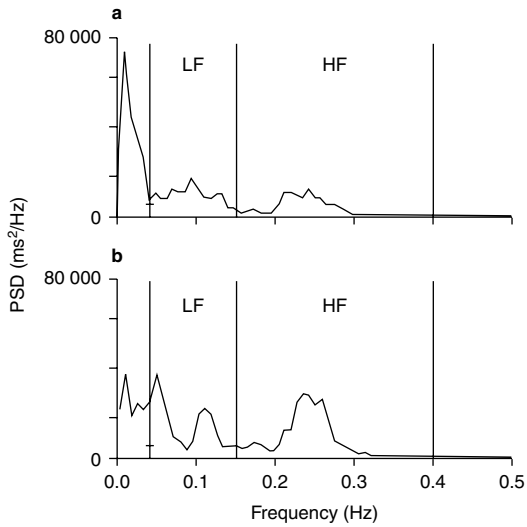

Fig. 5. (**a**) Tachogram and power spectral density of a recording in a young sedentary individual before training (HF = 812.3 ms²); and (**b**) the same individual after a 6-month aerobic training programme (HF = 1878.4 ms²). **HF** = high frequency; **LF** = low frequency; **PSD** = power spectral density.

exercise and 10 minutes cool down. Training began at 50–60% of heart rate reserve and increased to 80–85% by the tenth month. However, HRV was only measured as SD (ms) of all normal RR intervals during a 2-minute acquisition. They found an increase in this parameter of 68% in the elderly and of 17% in the younger men. Their conclusion was that exercise training increases parasympathetic tone in both healthy elderly and young men. However, it has been proven,[18] and it is a mathematical law (Parseval's theorem), that SD corresponds to total power and as such is a combination of sympathetic and of parasympathetic activity; therefore, their conclusion is incorrect, or at least an overstatement. A contribution by each division of autonomic modulation to HRV is only possible when this variable is represented in the frequency domain as a power spectral density graph.

Catai et al.[155] also trained an elderly (50–59 years) and a younger (19–24 years) population and reported HRV values obtained in the frequency domain, awake and during sleep (table IV). The training programmes were conducted for 3 months on a field track and included stretching for 10 minutes followed by walking and/or jogging for 40 minutes; three times a week at a prescribed heart rate corresponding to 70–80% of peak heart rate. The authors found no significant changes in HRV associated with an increase in aerobic capacity induced by aerobic training. They concluded that resting bradycardia induced by short-term aerobic training in both young and middle-aged men is more related to intrinsic alterations in the sinus node than to changes in efferent parasympathetic-sympathetic modulation. As they mentioned, however, the primary goal of the experimental design was directed to evaluate the cardiorespiratory adaptation in short-term training; they only used two 1000s epochs out of a 24-hour Holter recording (awake and asleep). The training period was very short (12 weeks) with a small number of study participants.

In summary, it can be stated that there are conflicting reports in the literature concerning the effects of aerobic training in a general population on HRV parameters under resting conditions. While some studies have reported an increase in the magnitude of HRV in the time domain,[175] in the frequency domain others have reported absence of modifications,[169] and an increase[145] or decrease[153] of sympathovagal balance in the sinus node. Therefore, studies of aerobic training effects on HRV parameters on a previously not-trained (young and/or elderly) population still remain necessary, preferably under well-controlled conditions.

4.4 Differences Due to Age and Sex

Many studies with a large number of study participants have focused on the influence of age and sex on cardiac autonomic tone (HRV parameters).[69,176-180] The general conclusions of these studies was that: (i) ageing reduced the global measure of HRV, at rest, in general and of both its spectral components (LF and HF). Therefore, this decline might reflect reduced responsiveness of autonomic activity with age; and (ii) all HRV parameters, except for HF power were higher in men and this sex difference was confined to the age categories less than 40–50 years. The lower sympathetic tone (LF) in women might provide protection

against arrhythmias and the development of coronary heart disease.

A potential confounding effect of the menstrual cycle can arise in studies that address sex differences in HRV parameters. Effects of the menstrual cycle have been shown on cardiac autonomic function as assessed by HRV methods,[181-183] and even of hormonal replacement therapy in post-menopausal women.[183] All studies agreed that regulation of the autonomic tone is modified during the menstrual cycle. The alteration in the balance of ovarian hormones might be responsible for these changes in the cardiac autonomic activity. Results suggest that parasympathetic nerve activity is predominant in the follicular phase. Unfortunately, in the few sex studies concerning young female athletes, no mention is made of timing within the menstrual cycle.

This view is also supported in a study from Boutcher et al.[184] and confirmed by Davy et al.[185] and McCole et al.[114] who found that older women athletes (post-menopausal women), who had habitually performed vigorous endurance training, had higher stroke volume and cardiac outputs during maximal exercise, than their sedentary post-menopausal peers. On the other hand, in young female athletes, similar results are found when compared with their male counterparts. Pigozzi et al.[186] performed a 24-hour Holter study (spectral analysis with AR) in 26 highly trained female athletes (24.5 ± 1.9 years). They were assigned to a 5-week aerobic training programme during a yearly rest period. They concluded that from the relative night-time increase in LF and the decrease in the day-night difference in time domain indexes, exercise training is able to induce an increase in the sympathetic modulation of the sinus node, coexisting with signs of reduced or unaffected vagal modulation in this group of young female athletes.

A sex difference was obtained by Hedelin et al.[187] in junior athletes. They compared short-term HRV recordings (AR power spectrum) in 17 cross-country skiers (nine females, eight males; 16–19 years) before and after the competitive season. After the intensive training/competition season there was a general increase in HRV. No difference in resting heart rate was found, pre- and post-season; however, they found a higher level of parasympathetic activity in females than in males, reflected by a consistently higher HF and total variability.

A difficulty comparing previous data is that: (i) training level is different; (ii) training duration is different (short-term or long-term effects); and (iii) duration of ECG recording is different (24-hour Holter recordings versus short duration ECG recordings).

In general, the literature proposes three conclusions concerning ageing:

- cardiovascular and cardiorespiratory function are higher in elderly athletes than in age-comparable sedentary groups;[188]
- the capacity for significant function in endurance and power persists throughout life in trained individuals;
- strength decreases more rapidly than endurance.[189]

How do these findings in a general population relate to the ageing athlete? There are many physiological, structural and psychological differences that distinguish elderly athletes from younger ones and from a similar aged sedentary group, especially if still active. Regular exercise may be able to retard the physiological decline.[190] This is supported by the very few HRV studies performed in senior athletes so far (table V). Yataco et al.[191] determined the age-associated decline in HRV by comparing HRV parameters in older athletes (n = 15; 69 ± 7 years) with age-matched sedentary persons (n = 14; 69 ± 4 years). They showed positive correlations between HRV parameters and aerobic fitness (as determined from maximal treadmill exercise). Frequency analysis was performed after Holter monitoring. Senior competitive athletes had increased HRV and parasympathetic heart rate activity (table V) when compared with their sedentary counterparts. This work supports the hypothesis that the age-associated decline in HRV parameters is due, in part, to lifestyle and not solely to ageing. Similar results were shown in the study by Banach et al.,[192] i.e. higher HRV parameters in middle-aged athletes compared with a sedentary population (table V),

Table V. Heart rate variability parameters in elderly athletes and an age-matched sedentary population

Study	n	Age (y) [mean ± SD]	LF (ms^2) [mean ± SD]	HF (ms^2) [mean ± SD]	Comments
Banach et al.[192]	9	52.9 ± 7.2	1088*[a]	920[a]*	Athletes
	9	52.9 ± 7.2	220[a]	294[a]	Sedentary
Jensen-Urstad et al.[151]	11	73.2 ± 2.8	673 ± 244	353 ± 349	Athletes/24h Holter
	12	74.5 ± 2.7	492 ± 290	209 ± 172	Sedentary
			764 ± 327	475 ± 654	Athletes/night
			728 ± 485	328 ± 48	Sedentary
			587 ± 250*	267 ± 163*	Athletes/day
			346 ± 177	127 ± 41	Sedentary
Yataco et al.[191]	15	69 ± 7	891[b]*	575[b]*	Athletes/24h Holter
	14		537[b]	102[b]	Sedentary

a Mean only. No SD available.

b Values transformed from log.

HF = high frequency power; **LF** = low frequency power; * $p < 0.05$.

indicating that the autonomic activity in sportsmen is not affected by ageing until the sixth decade of life.

Jensen-Urstad et al.[193] showed that elderly athletes (n = 11; 73.2 ± 2.8 years) with a lifelong training history seem to have more complex arrhythmias and profound brady-arrhythmias than do healthy elderly controls, which may increase the risk of sudden cardiac death. In contrast, however, the age-related decrease in HRV also seems retarded as in previous studies (table V). The latter has a positive prognostic value and may decrease the risk of life-threatening ventricular arrhythmias.

Results from the few HRV studies in elderly athletes all point in the same direction, i.e. the decline in HRV parameters associated with age is overcome to some extent by sustained endurance training into old age. However, more studies are needed, especially to show any beneficial effect of lifelong regular training on quality of life and on life expectancy.

4.5 Over-Training and the ANS

In athletic training, workloads are gradually increased, thereby exceeding the previously employed workload. This 'overload' principle is an important component of modern training,[194] and is a positive stressor that can be quantified according to load, repetition, rest and frequency.[2] Application of too much training stress and too many training sessions can result in exhaustion of a physiological system. 'Over-training syndrome' or 'staleness' in athletes results from long-term stress or exhaustion due to prolonged imbalance between training and other external and internal stressors and recovery.[195-197]

It is well known that over-training causes hormonal imbalance in athletes.[198,199] Due to these hormonal changes, over-training will lead to an autonomic imbalance.[200-202] In which way the ANS changes (parasympathetic versus sympathetic) is still controversial. From a clinical standpoint, Israel[201] distinguished between a parasympathetic type or vagal type (Addison type) over-training syndrome and a sympathetic type (Basedow type). The two types of over-training were the consequence of an imbalance between training and rest periods, but it was expected that a sympathetic type over-training syndrome might rather be the consequence of too much accompanying psycho-emotional stress, such as too many competitions and too many non-training stress factors (e.g. social, educational, nutritional).[200] Kuipers[198] hypothesised that during the early stage of the over-training syndrome, the sympathetic system was continuously altered, whereas during advanced over-training the activity of the sympathetic system was inhibited, resulting in a marked dominance of the parasympathetic system.

The cardiac autonomic imbalance observed in over-trained athletes implies changes in HRV and therefore would suggest that heart rate variability

Table VI. The effect of over-training on heart rate variability parameters

Study	n	Age (y)	TP (ms^2)	LF (ms^2)	HF (ms^2)	Comments
Hedelin et al.[203]a	9	18–23	3.71 ± 0.29	2.9 ± 0.57	3.4 ± 0.27	Control
			3.66 ± 0.26	2.77 ± 0.29	3.36 ± 0.43	Over-trained
Uusitalo et al.[204,205]b	6	19–27	5100 ± 900	800 ± 200	2800 ± 700	Control
			8600 ± 3700	700 ± 200	5600 ± 3200	Light training
	9	20–27	5500 ± 100	600 ± 100	2700 ± 600	Control
			5500 ± 1200	900 ± 200*	2900 ± 700	Over-training

a Values from Hedelin et al. have been log transformed (mean ± SD).

b Values from Uusitalo et al. are absolute values (mean ± standard error of the mean).

HF = high frequency power; **LF** = low frequency power; **TP** = total power; * $p < 0.05$.

could provide useful parameters to detect over-training in athletes. Despite these expectations, little is known about changes in heart rate variability due to over-training and only a few studies are available (table VI).

Hedelin et al.[206] investigated nine canoeists (six men and three women; 18–23 years) before and after a training regimen corresponding to a 50% increase in normal training load applied during 6 days. Heart rates reduced (−5 to −8 beats/min) both at submaximal and maximal levels, which could be due to hypervolaemia leading to increased stroke volume and maintenance of cardiac output with lower heart rates. Unlike these changes in heart rate, no significant differences were found in HRV parameters, neither when stressing the parasympathetic system (controlled breathing) nor when stressing the sympathetic system (tilt test, i.e. the study participant starts in a supine position on a special bed that is raised passively to an angle of 60º). They then concluded that these HRV data did not support an altered autonomic balance in these athletes. A possible explanation could be that a 6-day training period has only a small effect on individual HRV parameters and also that group differences would be difficult to determine in small groups. A case study of the same authors[203] in a cross-country skier, showed a relative parasympathetic dominance when the athlete was over-trained.

We suggest that it is impossible to find group changes in HRV because of the two types of over-training; however, individual HRV can change due to over-training. These hypotheses were confirmed by Uusitalo et al.[204] who investigated HRV and BPV of young female athletes during a 6- to 9-week training period (table VI). They compared a high-intensity training group (four long distance runners, one cross country skier, two triathletes, one orienteer), with a low intensity training group (one long distance runner, three cross country skiers, one triathlete, one orienteer). The purpose of the experimental training period was to over-train this group after a period of 6–9 weeks. Heavy endurance training seemed to induce a significant increase in LF of HRV during supine position, but not in the low intensity training group. In many individuals, the changes in supine and standing heart rate variability seemed to be rather contrary. Since there were no large uniform findings in the over-trained athletes, the authors also looked at individual results during a tilt-test. Increased as well as decreased changes due to upright tilt were found in the over-trained athletes compared with their values in the normal training state. This is a sign of either increased or decreased ability to increase sympathetic discharge during standing, and corresponds to the two over-training types. However, the changes were not specific to over-training because there also were similar changes in the not over-trained athletes.

Portier et al.[207] tested eight runners twice (after a relative rest period of 3 weeks and after a 12-week intense training period for endurance) and each time determined HRV parameters. Although the athletes were not trained until over-training, they concluded that spectral analysis could be a means of demonstrating impairment of autonomic balance for the purpose of detecting a state of fatigue that could result in over-training. Pichot et al.[208] came to

similar conclusions. They assessed ANS activity in seven middle distance runners (24.6 ± 4.8 years) during their training cycle (3 weeks heavy training, followed by a relative resting week). HRV was analysed using FFT and WT. Their results confirmed that heavy training shifted the cardiac autonomic balance of the sympathetic over the parasympathetic drive. Night-time results of HRV parameters proved a good tool to estimate cumulated physical fatigue. Therefore, they concluded that HRV could be valuable for optimising individual training profiles.

No definite conclusions can be reached concerning the use of HRV methods during over-training in athletes as only very few studies are available, even so with conflicting results. It remains to be proven that the autonomic imbalance observed in over-trained athletes, manifests itself from HRV indices.

5. Conclusions

Innumerable studies have been published concerning training in general (an Internet search on the keyword 'training' results in 409 395 hits) and concerning the physical and physiological condition of athletes. However, only very few papers discuss studies of HRV and its application to athletics (117 hits). Therefore, cardiovascular variability studies in athletes are still an almost unexplored domain. Much work still needs to be done to advance an understanding of the action of the ANS in athletes as a function of athletic discipline, age, sex, intensity and duration of training, detraining and over-training effects, comparison with sedentary population, and so on.

Another key issue is that almost no studies are available as a longitudinal section for the follow-up of athletes during ageing, as well as very few studies on active elderly athletes.

For further studies, the following standardised conditions are recommended: (i) selection of study participants, i.e. age, sex, training or physical fitness level, athletic discipline and focus on aerobic or anaerobic training; (ii) measurements, with the minimal number of parameters proposed being ECG, (non-invasive) blood pressure, and eventually respiration; (iii) measurements at rest with a minimum of 10 minutes supine and 10 minutes standing to activate the sympatho-vagal balance, eventually breathing at different fixed frequencies, to activate primarily the parasympathetic system, 24-hour Holter monitoring when day to night separation is needed for circadian pattern detection; and (iv) measurements during exercise, either with adapted trend removal or else at constant work levels in order to have stationary signals.

For interpretation of the data in the time and frequency domain, the use of guidelines[24] are recommended in order to be able to compare different studies.

It is strongly suggested that, when presenting reports on HRV studies related to exercise physiology in general or concerned with athletes, a detailed description should be provided on analysis methods, as well as concerning population, training schedule, intensity and duration. Only with such information will it be possible to understand and evaluate conclusions drawn and compare results with other studies. Until now, this has not been the case in most studies on HRV in athletes as found in the literature, therefore, it is only possible to make general comparisons.

Most studies concern relatively small numbers of study participants, diminishing the power of statistics. It is of course not easy to find and motivate large numbers of athletes to participate in scientific studies; the usual answer (especially from coaches) is that the athletes should train and refrain from losing time on other topics such as specific physiological measurements. Therefore, multicentre studies would be preferable to: (i) enhance the value of the study and motivate the study participants; and (ii) increase significantly the number of participants. This would also facilitate a multidisciplinary approach between cardiologists, exercise physiologists, pulmonary physiologists, coaches and biomedical engineers needed to evaluate the many different and interrelated aspects of cardiovascular variability in the athlete.

In order to further develop this fascinating research field, we advocate prospective, randomised,

controlled, long-term studies using validated measurement methods. However, there is a strong need for basic research on the nature of the control and regulating mechanism exerted by the ANS on cardiovascular function in athletes, needed for better understanding of this phenomenon.

The question remains whether aerobic exercising helps in maintaining and developing cardiovascular fitness in a general population,[209] or whether it can be used as a predictor of mortality,[81] or whether physical activity has beneficial effects on the cardiovascular risk profile,[210] or in other words, is lifelong exercising cardioprotective? The fact is, physical activity has a great impact on quality-of-life improvement in all of those involved in athletic activities.

Finally, study of cardiovascular variability (HRV and BPV) is a potentially powerful method as a basic scientific tool for better understanding the regulation and control of the cardiovascular system. From a practical point of view it remains to be determined if it can also be used as a predictor of athletic condition[211] and of athletic achievements.

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