REVIEW ARTICLE

Sports Med 2003; 33 (1): 33-46
0112-1642/03/0001-0033/$30.00/0

Effect of Endurance Exercise on Autonomic Control of Heart Rate

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Contents

Abstract

Long-term endurance training significantly influences how the autonomic nervous system controls heart function. Endurance training increases parasympathetic activity and decreases sympathetic activity in the human heart at rest. These two training-induced autonomic effects, coupled with a possible reduction in intrinsic heart rate, decrease resting heart rate. Long-term endurance training also decreases submaximal exercise heart rate by reducing sympathetic activity to the heart. Physiological ageing is associated with a reduction in parasympathetic control of the heart; this decline in parasympathetic activity can be reduced by regular endurance exercise. Some research has indicated that females have increased parasympathetic and decreased sympathetic control of heart rate. These gender-specific autonomic differences probably contribute to a decreased cardiovascular risk and increased longevity observed in females.

1. Autonomic Adaptations to Endurance Exercise

Cardiovascular adaptation to acute exercise involves integration of neural and local factors. Neural factors include: (i) central command; (ii) baroreceptor reflex action; and (iii) neural reflex feedback from contracting muscle. Central command initiates activity in the sympathetic branch of the autonomic nervous system, producing an increased heart rate, increased myocardial contractile force and peripheral vasoconstriction. Central command also initiates a reset in the baroreflex to a higher blood pressure set point. The arterial baroreceptor reflex maintains circulatory homeostasis by responding rapidly to any change in arterial blood pressure. Baroreceptor firing frequency is increased by an increase in blood pressure and decreased by a reduction in blood pressure. Stimulation of baroreceptors results in an increase in efferent cardiac parasympathetic activity and decreases

in sympathetic activity. An increase in baroreceptor impulse frequency inhibits vasoconstrictor action and results in blood vessel vasodilation and a subsequent reduction in blood pressure. Reflex activity from contracting muscle is also activated intramuscularly by mechanoreceptor stimulation (stretch and tension) and chemoreceptors (products of metabolism) in response to muscle contraction. Impulses from these receptors travel centrally through afferent fibres. The central connections of the reflex are still unclear, but the efferent fibres are sympathetic nerve fibres innervating the heart and peripheral blood vessels.

Endurance exercise may be defined as activity of at least 20 minutes duration in which heart rate is elevated to 60–80% of maximum. Endurance exercise is a physiological perturbation that significantly affects autonomic nervous activity. Research has indicated that long-term endurance training increases parasympathetic activity and decreases sympathetic activity directed to the human heart at rest.[1-8] These training-induced autonomic changes, coupled with a possible reduction in intrinsic heart rate, decrease resting heart rate and increase heart rate variability at rest.[3,5,6,9-11] Athletes have a lower resting heart rate, and a more rapid heart rate recovery following exercise due to enhanced parasympathetic activity resulting from long-term endurance training.[6,12] Endurance trained individuals show a reduced sympathetic activity for any given submaximal work-rate, compared with sedentary controls exercising at the same rate.[3,4]

Data from studies of autonomic function indicate that heart rate regulation at rest is dominated by parasympathetic nervous system activity, whereas during exercise the sympathetic nervous system activity is dominant.[3,6,12-15] Up to 100 beats/min, a significant portion of the heart rate increase is produced by parasympathetic withdrawal, however once heart rate rises above 100 beats/min the increase is through enhanced sympathetic activation of cardiac β-adrenergic receptors by noradrenaline.[3,15-17]

Recent studies[18-20] have demonstrated that during low-to-moderate intensity exercise the carotid baroreflex is reset so that the stimulus-response relationship is shifted to operate around the prevailing arterial blood pressure of exercise, without a change in the gain or sensitivity of the baroreflex.[18] It has also been suggested that during dynamic exercise the gain of the baroreflex arc is reduced by inputs from the motor cortex.[19] It is also possible that chronic endurance exercise can alter autonomic function and depress baroreflex control by decreasing baroreceptor sensitivity.[20] Smith et al.[20] found that sensitivity of the arterial baroreflex was significantly reduced in very fit young adults compared with moderately fit young adults.

There is consistent evidence for parasympathetic withdrawal as the primary mediator of increased heart rate to 60% of maximal oxygen uptake ($\dot{V}O_{2max}$), however the characteristics of sympathetic activity have yet to be clarified. An early study by Robinson et al.[15] reported that at low-to-moderate exercise intensity an increase in heart rate was mediated primarily by parasympathetic withdrawal, and that sympathetic activity increased initially at 60% of $\dot{V}O_{2max}$. Arai et al.[21] reported a reduction in parasympathetic activity with increasing cycle ergometer intensity, but no change in sympathetic activity. Yamamoto and Hughson[22] reported parasympathetic activity decreased until the exercise intensity reached 60% of ventilatory threshold (T_{vent}). These authors also reported that sympathetic activity remained unchanged up to 100% of T_{vent} and then increased abruptly at 110% T_{vent}. Nakamura et al.[14] reported that parasympathetic activity decreased significantly at 50% peak oxygen uptake ($\dot{V}O_{2peak}$), while sympathetic activity initially increased at 50–60% $\dot{V}O_{2peak}$, and increased significantly further after 60% $\dot{V}O_{2peak}$.

The influence of autonomic nervous system activity in generating a training-induced bradycardia has been examined indirectly during pharmacological cardiac autonomic blockade. Parasympathetic control is blocked with atropine, while sympa-

thetic activity is blocked with propranolol.[3] The completeness of such autonomic blockade has frequently been questioned, and even when complete, only one branch of autonomic control can be analysed.[12]

Various studies have examined parasympathetic and sympathetic nervous system effects, as well as parasympathetic and sympathetic tonus.[10,23] A parasympathetic effect can be examined by determining the difference between resting heart rate and the heart rate after muscarinic cholinergic receptor blockade with atropine.[24] A sympathetic effect can be studied by determining the difference between resting heart rate and heart rate after β-adrenergic receptor blockade with propranolol.[23] One drawback to receptor blockade is that it becomes difficult to distinguish the direct result of a single blockade action from an indirect comparison.[10] The heart rate resulting from β-adrenergic receptor blockade is due to the direct removal of sympathetic influence on the heart as well as the indirect effect of a now unopposed parasympathetic influence on the heart. A further limitation to experiments using pharmacological cardiac autonomic blockade is that they cannot determine the extent of any change in intrinsic heart rate.

Cardiac autonomic blockade study of humans has reported an increased parasympathetic control of heart rate following endurance training of proper duration and intensity.[5,7] Studies that reported a large increase in $\dot{V}O_{2max}$ (greater than 12 ml/min/kg) after endurance training, have also shown an increase in parasympathetic control of heart rate.[5,7] Studies that have reported a small increase in maximal oxygen uptake after training indicate that parasympathetic control of heart rate is unchanged.[25,26] A cross-sectional study by Katona et al.[27] reported less parasympathetic control of heart rate in rowing athletes compared with sedentary individuals even though the $\dot{V}O_{2max}$ of the rowers was 25 ml/min/kg higher than in sedentary individuals. Conversely, a study by Shin et al.[28] reported that, at rest, parasympathetic activity in athletes was significantly higher than in non-athletes. The inconsistent results of these studies are due to a number of factors. Currently there is no formal training theory that quantitatively and accurately prescribes the pattern, duration and intensity of exercise to elicit a specific physiological adaptation. The training history of an individual will also influence their physiological response to exercise. Research also indicates that there are individual differences in response to exercise.[29]

Endurance training seems to reduce the efferent sympathetic neural outflow to the sinoatrial (SA) node in the heart.[7,30] Human studies that have used cardiac autonomic blockade to investigate the effect of training on autonomic balance, reported a decreased sympathetic control of heart rate following endurance training.[7,25] Smith et al.[7] compared ten endurance-trained runners with ten sedentary controls, and reported that sympathetic influence on heart rate was slightly less in the endurance-trained individuals. One possible mechanism for a decrease in sympathetic nervous activity in a trained individual, is that the reflex heart rate response to myocardial stretch may be attenuated.[31] A change in baroreceptor sensitivity is another possible mechanism contributing to exercise bradycardia.[20] Peripheral adaptation associated with training may also contribute to exercise bradycardia.[32-35] Central, reflex and peripheral adaptation to endurance training probably all contribute to exercise bradycardia.

The effect of exercise on autonomic activity may also be determined from bed rest and detraining studies, in which participants undertake no physical activity for an extended period.[36] A study by Hughson et al.[37] reported a significant reduction in parasympathetic activity, and a non-significant increase in sympathetic activity, after 28 days of head-down bed rest. Physiological ageing is also associated with a reduced parasympathetic control of the heart.[21,38] This decline in parasympathetic activity with age may be strongly influenced by an age-related decrease in fitness level.[39] As outlined previously, endurance training increases parasympathetic control, and the endurance-trained athlete has an increased para-

sympathetic control compared with a sedentary individual.

1.1 Spectral Analysis

Spectral analysis of the electrocardiogram (ECG) has been used since the late 1960s. The technique reduces a heart rate time series to its constituent frequency components and quantifies the relative power (squared amplitude) of these components.[40,41] Power spectral density analysis provides information on how power (variance) distributes as a function of frequency.[42] Power spectral density methods are classified as parametric or nonparametric. Fast Fourier transform analysis is the most common nonparametric method. Fast Fourier transform analysis breaks down the variation of R-R interval sets into component sine waves of differing amplitude and frequency.[43] Amplitude is then displayed as a function of frequency and the power (cumulative variance) is calculated for different frequency ranges. The advantages of nonparametric methods, such as the fast Fourier transform, are the simplicity of the algorithm used and the high processing speed.[40] An autoregressive algorithm is an example of a parametric method, which yields centre frequencies and absolute power of component frequencies.[44] The advantages of the parametric method include: an accurate estimation of power spectral density even with a small number of sample sequences, smooth spectral components that may be distinguished independently of pre-selected frequency bands and automatic calculation of low and high-frequency power components with identification of the central frequency of each component.[40,42] The disadvantages of parametric methods are that they are more complex, and it is difficult to verify the mode.[40,42] In general however, if proper algorithms are used, nonparametric and parametric methods will produce comparable results.[42]

During the last two decades, spectral analysis of the ECG has been used to determine the harmonic composition of the heart rate signal and provide insight into autonomic cardiovascular control.[45-48] Some researchers have also concluded that underlying the harmonic component of heart rate variability, is a fractal component that may yield further insight into cardiovascular dynamics.[49-56]

1.2 Heart Rate Variability

Heart rate variability (HRV) has been recognised as a powerful tool for the estimation of cardiac autonomic modulations.[24,42,47] Heart rate variability is a term used to describe variations in both instantaneous heart rate and R-R interval sets.[42] There are regular fluctuations in heart rate, which are primarily due to the changing level of both parasympathetic and sympathetic neural control of the heart.[14,21,57] Heart rate fluctuation may therefore be considered an output variable of a feedback network that is continuously monitored and regulated by the autonomic nervous system.[58] Measurements of HRV during short time periods (5 minutes) are stable and may be regarded as characteristic of an individual.[45,48] HRV has been associated with three major physiological factors: oscillatory fluctuations in blood pressure, frequency oscillations due to thermal regulation and respiration.[59] Recent evidence also suggests that genetic factors may explain a substantial proportion of the variance in HRV.[60,61]

Analysis of the time course of an ECG signal demonstrates that HRV may be recorded as harmonic oscillations.[21,22,43,58,59,62] Sayers[59] was able to separate spontaneous fluctuations in heart rate into three separate bandwidths. Sayers[59] attributed low-frequency (<0.05Hz) oscillations to thermal regulation, mid frequency (0.1Hz) fluctuations to arterial baroreceptor modulation and high frequency (>0.15Hz) fluctuations to respiratory sinus arrhythmia. Chess et al.[62] examined the effect of selective blockade on either branch of the autonomic nervous system on decerebrate cats, and determined that high frequency fluctuations are mediated entirely by parasympathetic activity. Akselrod et al.[63] investigated the effect of selective and combined autonomic blockade and angiotensin-converting enzyme inhibition on a conscious dog. These investigators determined the role of parasympathetic activity in mediating heart rate

fluctuations to be above 0.15Hz, and the importance of sympathetic activity and renin-angiotensin at fluctuations below 0.15Hz.[63] Berger et al.[58] applied a frequency-modulated pulse train to either the right vagus or the cardiac sympathetic nerve in anaesthetised dogs, and computed the transfer function between nerve stimulation rate and the resulting atrial rate. They found that the SA node acts as a low pass filter to fluctuations in both the sympathetic and parasympathetic tone. Sympathetic fluctuations have a lower corner frequency than the parasympathetic fluctuations and a delay of approximately 1.7 seconds.[58]

Spectral analysis, pharmacological blockade and direct neural stimulation all indicate that heart rate harmonic oscillations are concentrated in at least two distinct spectral regions.[21,22,43,49,58,59,63-67] Results from these studies lead to the conclusion that the high frequency (HF) region is between 0.15 and 0.50Hz, and it is mediated solely by the parasympathetic nervous system, with respiration being the primary rhythmic stimulus.[21,43,63,68-70] The low frequency region (LF) is between 0.04 and 0.15Hz and is mediated by both the parasympathetic and sympathetic nervous system.[21,63,68,69,71] Thus the estimation of sympathetic activity from low frequency power is more problematic than estimation of parasympathetic activity from high frequency power.[43] Low frequency heart rate fluctuations may also be related to baroreflex activity, temperature regulation and cardiovascular stresses such as haemorrhage and congestive heart failure.[21] The low frequency region includes a peak centred near 0.1Hz which is referred to as Mayer wave activity, believed to represent variation in blood pressure caused by altered peripheral vascular tone.[68] There are also very-low-frequency (VLF) components (≤0.04Hz) with time periods lasting several hours, which may affect the spectral analysis of a signal recorded for only a short period of time.[22] The VLF component is not attributed to any physiological mechanism and remains subject to debate. The major constituent of the VLF component is thought to be non-harmonic or fractal in nature.[42]

The accepted measurement of HF, LF and VLF power components is in absolute values of power (ms^2).[42] LF and HF components may also be measured in normalised units in order to represent the relative value of each power component as a proportion of total power.[67,68] Representation of LF and HF in normalised units indicates a balanced behaviour of the autonomic nervous system. Sympathovagal balance is simply the ratio of absolute LF to absolute HF power.[44] The concept of sympathovagal balance has been promoted by some researchers, but lacks a physiological base.[44] There is no physiological evidence that sympathetic and parasympathetic nervous activity constantly interact and are balanced. However, it is true that both sympathetic and parasympathetic motoneurons respond to interrelated neural influences.[44] It should therefore be noted that the LF and HF components of heart rate variability provide a measure of the degree of autonomic fluctuation rather than a level of autonomic tone.[42]

Current HRV research is divided into several areas. There are studies which refine existing, and explore new, technical modes of ECG processing and new HRV assessment methods.[52,57,72-74] There is research attempting to develop improved understanding and interpretation of HRV.[54,75-77] There are numerous studies that are using HRV analysis for clinical and practical use.[51,53,78-80] Some recent technical studies have concentrated on the non-linear aspects of HRV, and the physiologic factors that influence HRV.[46,52,73,74] Current studies indicate that HRV may soon be a powerful tool for predicting and monitoring coronary heart disease and monitoring reinnervation after heart transplant surgery.[46,79,81]

1.3 Effect of Exercise on Heart Rate Variability

Several studies that have used spectral analysis to investigate the effect of chronic endurance exercise on autonomic nervous system activity have demonstrated some inconclusive results.[2-4,82,83] However, most spectral analysis studies support the theory that endurance training increases HRV,

increases parasympathetic activity and thus contributes to a training bradycardia.[2-4,82]

Spectral analysis studies indicate that as acute exercise intensity increases, there is a change in cardiovascular control response.[84] HRV, and total spectral power of HRV, both decrease exponentially as exercise intensity increases. This is due to a progressive decrease in HF power (parasympathetic activity).[2,16,18,85-88] Parasympathetic activity is reduced during light and moderate exercise,[88,89] and is suppressed up to 1 hour after 30 minutes of intense treadmill running.[82] LF power remains constant[86] or decreases[16,18] during exercise. Perini et al.[88] reported that supine cycle ergometry at 28% of maximal oxygen uptake and seated cycle ergometry at 50% of maximal oxygen uptake, shift LF peak power towards lower frequencies with a reduction in power. Brenner et al.[12] reported that 30 minutes of cycle ergometry at 50% of maximum oxygen uptake decreased both low and high frequency power. There was a shift in the LF peak power to a lower value, and a shift in the HF peak to a higher value. Parasympathetic and sympathetic indicators returned to pre-exercise values, 15 minutes after exercise ceased.[12]

A study by Furlan et al.[82] investigated the long- and short-term effect of heavy dynamic exercise on neural control of heart rate. These investigators compared healthy controls with a group of trained swimmers during a detraining period and a group of trained swimmers at the peak of their training programme. The detrained swimmers showed a slight bradycardia with a predominant HF component, suggestive of a prevailing parasympathetic tone. The trained swimmers demonstrated a resting bradycardia together with a high low frequency component, suggesting a more complex neural interaction modulating heart rate.[82] Maximal dynamic exercise performed by controls induced an increase in the LF component, and a decrease in the HF component, which was still present 24 hours after exercise, suggesting a persistent sympathetic activation. From these results Furlan et al.[82] concluded that cardiac sympathetic activation, induced by heavy dynamic exercise, may explain the coexistence of training induced bradycardia and signs of increased sympathetic activity in trained athletes.

A study by Dixon et al.[2] examined the cardiac autonomic responses before and after steady state exercise by long distance runners and sedentary controls. They found that the HF parasympathetic component was higher, and the LF component was significantly lower, in athletes compared with controls. The researchers also found that that the LF : HF ratio was restored more rapidly following exercise in the athletes compared with the sedentary controls.[2] However, both groups demonstrated a similar attenuation of all spectral components during steady-state exercise. From these results Dixon et al.[2] concluded that endurance training modifies heart rate control in whole, or in part, through autonomic control mechanisms.

Further longitudinal studies are required to fully document cardiovascular adaptation to endurance exercise. Longitudinal studies are very difficult to manage however, due to the difficulty in quantifying endurance training, and accounting for individual variability. Spectral analysis appears to be a good non-invasive tool to examine endurance training-induced changes in autonomic control of heart rate. Improved understanding of the information provided by spectral analysis, coupled with improved spectral analysis technique, will provide greater insight into the autonomic adaptations associated with endurance training.

2. Effect of Age on Autonomic Adaptations

Research studies[90-93] indicate that in adult humans the cardiovascular system deteriorates with age. This deterioration is attributed to structural and functional change in the heart and blood vessels, coupled with a reduced inotropic and chronotropic response to a catecholamine stimulus.[90-93] The age-related decline in cardiovascular performance is more apparent during exercise than at rest.[90,93] Physiological indicators associated with cardiovascular ageing include a reduced, maximal heart rate, ejection fraction and maximal cardiac

output during exercise.[90,93-96] Reduction in maximal heart rate, and left ventricular performance, under maximal exercise stress is considered the most significant reason for reduced cardiac output in the older individual.[92,97,98]

Heart rate variability increases with gestational age and during early post-natal life, but a decline in autonomic function is already discernible after 10 years of age.[48,54,99-101] Physiological ageing is associated with a reduction in parasympathetic control of heart rate.[48,102-104] This decline in parasympathetic control with age may be partly due to a decrease in physical fitness with age.[3,105] This observed reduction in parasympathetic control is accompanied by a decline in respiratory sinus arrhythmia and a reduction in the bradycardia response to the Valsalva manoeuvre.[38] There is also a reduction in LF heart rate fluctuations in elderly individuals due to an age-related decline in cardiac responsiveness to sympathetic activation.[38,106] Thus, the older individual has a reduced range of parasympathetic and sympathetic response to physiological stress, which could have important medical implications. In these older individuals this decline in HRV may be modified by regular endurance exercise, which will increase parasympathetic activity and reduce sympathetic activity at rest and during exercise.[4,101,106,107]

In general there is no significant change in resting heart rate with age, except that its variability is reduced.[103,108] During exercise, an older individual has a lower heart rate for a given submaximal work rate. The increase in heart rate during exercise is a function of the relative, rather than the absolute, exercise intensity. The mechanism(s) responsible for a lower exercise heart rate in the older individual is not completely explained by a lower peak heart rate, as the percentage increase in heart rate within the operating range is also lower. The decrease in exercise heart rate with age is mainly due to less withdrawal of cardiac parasympathetic tone and a diminished β-adrenergic responsiveness.[109] The diminished β-adrenergic responsiveness in the older individual appears to contribute to an attenuated left ventricular contractile response to exercise, despite greater β-adrenergic stimulation.[109] Current research supports this, since administration of atropine, which inhibits the parasympathetic effect on the heart, produces a smaller increase in heart rate in the older individual compared with younger individual under resting conditions.[109] Spectral analysis studies also demonstrate that the older individual has less HF power in the heart rate variability spectra, which indicates reduced parasympathetic control.[4] These age-related differences do not adversely affect submaximal exercise performance in the older individual. However, a lower peak exercise heart rate and reduced left ventricular contractile capability will reduce maximal cardiac output, oxygen consumption and peak exercise capacity.[109]

In the older individual, endurance training will induce a lower heart rate during rest and submaximal work rates.[97,107] The decrease in resting and exercise heart rate is similar to that observed in the younger individual.[109] The training-induced resting bradycardia in the older individual is partially due to an increase in parasympathetic activity.[45,50] As discussed previously in this review, there is also a training-induced intrinsic heart rate change that may differ between older males and females.

Heart rate and blood pressure variability has been used to investigate the progressive impairment of the cardiovascular control system with age.[102,110] A healthy physiological control system will have a high level of complexity and variability, while an ageing or diseased system will be less complex and show less variability. This loss of complexity with ageing leads to an impaired ability to adapt to physiologic stress.[41,52,54] Kaplan et al.[108] analysed continuous non-invasive blood pressure and heart rate in 16 young (21–35 years of age) and 18 older (62–90 years of age) adults. The results of this study indicated that the older individual had a lower heart rate and blood pressure complexity (variability).

A cross-sectional study by Gregoire et al.[4] used coarse graining spectral analysis to examine

autonomic control of heart rate, at rest and during exercise, in young (18–30 years of age) and middle-aged (40–55 years of age) participants. Each age group consisted of endurance-trained individuals and sedentary controls. Gregoire et al.[4] reported that there was a significant shift to increased parasympathetic and reduced sympathetic indicators at rest, in trained versus untrained, middle-aged participants. There were no differences in parasympathetic and sympathetic indicators at rest in young participants. Gregoire et al.[4] also reported that during exercise parasympathetic indicators decreased and sympathetic indicators increased, however spectral analysis techniques were unable to differentiate between age groups.

Davy et al.[111] studied HRV and carotid baroreflex sensitivity in 12 older (61 ± 2 years of age) and 11 younger (28 ± 1 years of age) sedentary females with physically active older and younger females. They reported that HRV and baroreflex sensitivity decline similarly with age in healthy sedentary and physically active females. However, physically active females show an increased level of HRV and baroreflex sensitivity compared with their sedentary peers, regardless of age.[81,111]

At rest, older individuals have an elevated arterial plasma noradrenaline concentration/spillover rate and increased muscle sympathetic nerve activity.[112] During brief exercise of increasing intensity, the plasma noradrenaline concentration response is consistently greater in older humans, however during prolonged exercise the increase in plasma noradrenaline concentration/spillover rate is not present.[112] The augmented noradrenaline spillover rate may be an attempt to maintain a capacity for β-adrenergic stimulation of heart rate and ventricular contractility in the face of an age-associated receptor impairment.[112]

The augmented plasma noradrenaline concentration observed in the older individual during exercise may play an important role in integrative circulatory control.[112] According to Rowell,[113] arterial blood pressure is the key regulated function during exercise. Research shows that for the same relative exercise intensity, cardiac output is lower in the older individual.[97] Thus, to produce the same arterial blood pressure response with a lower cardiac output, the older individual must maintain systemic vascular resistance at a higher level than a young individual.[97] Therefore a higher level of sympathetic neural outflow and/or synaptic noradrenaline concentration would be required to increase systemic vascular resistance.[112]

Age-related cardiac impairment probably has little effect on the ability of the healthy older human to perform submaximal exercise at the same relative exercise intensity as the young individual. The capacity to perform prolonged submaximal exercise is well preserved in the older individual. Research indicates that in older humans endurance training increases heart rate variability, lowers resting and submaximal heart rate and reduces heart rate and plasma noradrenaline concentration at the same absolute submaximal work rate.[102,107,112]

3. Effect of Gender on Autonomic Adaptations

Females have the same qualitative cardiovascular pattern of training response as males, but generally do not reach a similar absolute level of $\dot{V}O_{2max}$.[114-118] However, the variation in cardiovascular adaptation, between males and females, to endurance exercise training has not been thoroughly investigated. The main reason for the lack of research evidence is the fact that the majority of studies examining cardiovascular adaptation to endurance training have been completed on males. The developmental difference in females, attributed to the gender difference in steroid hormones, suggests that females may follow a different adaptation sequence to endurance exercise than males. Female characteristics that may affect adaptation to endurance training include: a difference in autonomic control, metabolism and hormone levels, as well as a smaller lean body mass, blood volume and heart size compared with males.[92,119]

Few studies have examined gender differences in autonomic control, with even fewer investigating the effect of gender-related adapta-

tions in autonomic control following endurance exercise. Some HRV studies have indicated that females may have increased parasympathetic and decreased 'sympathetic control of heart rate.[4,32,103,120,121] Although a decline in HRV occurs in both sexes, it may occur earlier in men.[48] Females may also have an increased overall complexity of heart rate dynamics compared with males.[103] These gender-specific autonomic differences may contribute to the decreased cardiovascular risk and increased longevity in females.[32,103]

A cross-sectional study by Gregoire et al.[4] investigated the effect of gender and physical training on HRV at rest and exercise. These investigators reported that both trained and untrained young (18–30 years of age) and middle-aged (40–55 years of age) females had significantly greater HRV than males. Young untrained, middle-aged trained and untrained females had significantly lower sympathetic activity at rest compared with their respective male counterparts. Furthermore, young untrained and middle-aged trained females had significantly higher parasympathetic activity at rest compared with their respective male counterparts.[4]

A study by Ryan et al.[103] examined HRV in healthy young (20–39 years of age), middle aged (40–64 years of age) and elderly (65–90 years of age) males (n = 40) and females (n = 27). They reported that resting mean heart rate did not differ between age and gender groups. HF power (parasympathetic activity), and high/low frequency power ratio, significantly decreased with age in both males and females. HF power was significantly greater in females than males. Overall complexity of heart rate dynamics decreased with age and was higher in females than males.[103]

A large cross-sectional study by Kuo et al.[122] examined the effect of age and gender on autonomic control of heart rate. These authors studied the effect of gender on sympathetic and parasympathetic control of heart rate in middle aged individuals and on the subsequent ageing process. They recorded HRV in healthy, normal populations of females (n = 598) and males (n = 472) ranging in age from 40–79 years. Females had higher parasympathetic activity in the 40–49 year age group, compared with males. Males showed increased sympathetic activity in the 40–59 year age group, compared with females. There were no significant gender differences in HRV measurements after 60 years of age. Kuo et al.[122] reported that the mean age of menopause in the female group was 47.7 years and thus the disappearance of parasympathetic dominance in females older than 50 years is probably due to a change in female sex hormones.

Huikuri et al.[121] studied baroreflex sensitivity and HRV in healthy, middle-aged females (n = 186; 50 ± 6 years of age) and males (n = 188; 50 ± 6 years of age). Baroreflex sensitivity, which was measured from the overshoot phase of the Valsalva manoeuvre, was significantly lower in females than in males. Females showed decreased LF HRV power and increased HF power, compared with males. Huikuri et al.[121] concluded that baroreflex responsiveness is attenuated, while parasympathetic activity is augmented, in females compared with males. Laitinen et al.[123] also found that age and gender have a significant impact on baroreflex sensitivity, with 24% of females over 40 years of age showing markedly depressed baroreflex sensitivity.

The results from these studies indicate that there are gender differences in autonomic cardiovascular regulation. In general, males have a greater sympathetic influence on the heart while females have greater parasympathetic control of heart rate. This enhanced parasympathetic control of heart rate may provide greater protection for females during periods of cardiac stress.[32] Despite these gender differences, both males and females will produce positive autonomic control changes to the heart following endurance training. Current research also indicates that there are individual differences in response to endurance exercise.[29]

4. Endurance Exercise and Cardiac Disease

Changes in autonomic control of the heart may be used to predict the onset of some diseases. Numerous studies, using a variety of methods, have determined that low HRV is a powerful predictor of all cause mortality.[79,121,124] Low HRV is considered a risk factor for coronary heart disease.[125] Research has shown that cardiovascular disease is often characterised by reduced parasympathetic activity and increased sympathetic activity.[3,32,78,124,126-129]

Numerous studies have reviewed how physical inactivity is an important risk factor for cardiovascular disease.[80,130] Various studies have indicated that enhanced parasympathetic activity prevents ventricular fibrillation during exercise, and that endurance training provides protection from sudden death by enhancing cardiovascular autonomic function.[104,131-133] Regular endurance exercise is clinically relevant in lowering blood pressure in hypertensive patients and increasing baroreflex gain in patients with ischaemic heart disease.[87,124] Coronary heart disease and cardiac sudden death incidence increases after menopause, but the incidence is lower in physically active females compared with less active females of the same age.[125] Endurance training increases parasympathetic activity in patients with cardiac disease.[132,133] Malfatto et al.[132] studied the effect of an 8 week endurance training programme on 22 patients recovering from a myocardial infarction. The investigators found that endurance training increased parasympathetic activity at rest. It therefore seems possible that endurance exercise may be used therapeutically to improve autonomic activity in those at risk of cardiovascular disease.

5. Conclusion

Long-term endurance training significantly affects autonomic control of the heart. Endurance training increases HRV, increases parasympathetic activity and decreases sympathetic activity in the human heart at rest. These training-induced autonomic changes, coupled with a possible reduction in intrinsic heart rate, will decrease resting heart rate. Athletes have a lower resting heart rate and a more rapid recovery of heart rate following exercise, due to enhanced parasympathetic activity produced by long-term endurance training. Endurance-trained individuals have a decreased submaximal exercise heart rate due to reduced sympathetic activity for a given submaximal work-rate.

Physiological ageing is associated with a reduction in parasympathetic control of the heart that is partly due to a decrease in physical fitness with age. The older individual has a decreased cardiovascular complexity that is displayed by a reduced range of both parasympathetic and sympathetic response to physiological stress. This inadequacy has important medical implications. The decline in HRV with age can be modified by regular endurance exercise, which will increase parasympathetic activity and reduce sympathetic activity at rest and during exercise

Some research has indicated that females may have increased parasympathetic and decreased sympathetic control of heart rate. Baroreflex sensitivity has been found to be significantly lower in females than in males. Females may also have an increased overall complexity of heart rate dynamics compared with males. These gender-specific autonomic differences may contribute to decreased cardiovascular risk and increased longevity in females. Numerous studies, using a variety of methods, have determined that low HRV is a powerful predictor of all cause mortality, and is considered a risk factor for coronary heart disease. It is possible therefore that endurance exercise may be used as therapy to improve autonomic activity in high-risk individuals.

Acknowledgements

The authors have provided no information on conflicts of interest directly relevant to the content of this review. This research was supported by the Heart and Stroke Foundation of British Columbia and the Yukon.

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