Global Journal of Physical Medicine and Rehabilitation

Global Scientific Library

Review Article

Cardiovascular Adaptations to Upright and Supine Prolonged Exercise: Heart Rate Modulation as a Factor of Cardiovascular Drift

Luna Rizzo[1]*, Martin W Thompson[2]

[1]Medical Sciences, Department of Biomedical and Neuromotor, Sciences, University of Bologna, Bologna, Italy
[2]Exercise, Health& Performance, Faculty of Health Sciences, University of Sydney, Sydney, NSW, Australia

Abstract

The interactive effects of posture and exercise on cardiovascular drift remain controversial despite scientists have conducted extensive studies addressed to this topic. For example, changes in posture at rest are associated with significant changes in Left Ventricular (LV) filling and Stroke Volume (SV). A transition from the supine to the upright position produces a decrease in LV end-diastolic pressureand volumeand SV [1,2]. The results of previous studies of the alterations in LV end-diastolic volume during exercise in the supine position have varied [3]. There is general theory that end-systolic volume is smaller during exercise than at rest [3], most scientists have observed an exercise-induced increase in SV, although others have not [1]. LV stroke volume increases markedly during the transition from rest to exercise in the upright position and is almost as great as during supine exercise [1]. It has been found that, after exercise, the Heart Rate (HR) changes showing an initial dramatic decrease followed by a gradual decrease to a steady state level, which maintains itself elevated above the level of pre-exercise baseline for a period relatively extended [4]. Many studies have reported that when generally the body position changes from upright to supine, cardiac sympathetic nerve activity decreases while vagus nerve activity increases[5]. If, during recovery from exercise, a reciprocal change in the activity of the two autonomic nervous system divisions occurs, a decrease of post-exercise HR elevation would be stimulated by the supine position via a prevalence of vagus nerve activity, compared to the upright position. The gravitational force plays an important role on the transition from the supine to the upright position. In fact, the distribution of blood volume in the body is markedly influenced by this force. It has been observed that, adjustments of arterial blood pressure to compensate the postural perturbation, are achieved in a complex manner through a number of reflex mechanisms [6]. Recent studies have reported that, effects of post-exercise include a reduction in arterial blood pressure, are related to body position[4]. However, mechanisms involved in the post-exercise hypotension and why this event occurs, remain unclear so far. This could be addressed to body position differences, i.e. supine and sitting, during the period after exercise. To date, several studies have been conducted on this argument to ascertain and clarify what mechanisms are responsible and involved in the cardiovascular drift responses to posture changes. Certain mechanisms remain still unclear despite few attempts have been made to evaluate different theories on these. The purpose of the present manuscript is to discuss and try to clarify how a change in posture affects the cardiovascular responses during recovery from exercise. This review paper also treats few other topics: circulatory responses affected by general exercise, heart rate influenced by exercise and environmental conditions and its relationship to stroke volume, ventricular function affected by strenuous exercise, hear rate variability.

***Corresponding author:** Luna Rizzo, Medical Sciences, Department of Biomedical and Neuromotor Sciences, University of Bologna, Bologna, Italy, E-mail: luna.rizzo2@unibo.it; luna.rizzo8@gamil.com; luna.rizzo@hotmail.com

Received: August 25, 2017; **Accepted:** October 09, 2017; **Published:** October 11, 2017

Keywords: Exercise position, Autonomic nervous system, Heart rate regulation, Gravitational force, Ventricular filling

Differences in cardiovascular responses between upright and supine exercise position

It has been reported that generally when the body position at rest is changed from standing to supine, cardiac sympathetic nerve activity decreases and the activity of the vagus nerve increases [6,7]. If such a reciprocal change in activity of the two divisions of the autonomic nervous system occurs during recovery from the exercise, a decrease in HR during the post-exercise would be induced in the supine position with the predominance of the vagus nerve activity, compared with the standing position [8].

The distribution of blood volume in the body during the transition from supine to standing position is deeply affected by the gravitational force. The arterial blood pressure adjustment to compensate for the postural perturbation was found to be achieved in a complex manner through reflex mechanisms [6]. Recently, many studies have shown that the cardiovascular responses post-exercise include a reduction in arterial blood pressure, independent of body position [4].

It was determined by Clausen [9], that an increase in Stroke Volume (SV) has to be considered an important cardiovascular response to upright and supine exercise. However, the precise mechanism by which SV changes during upright and supine position in man remains to be detected. A study by Higginbotham *et al.*[10]showed the effects of posture on cardiovascular responses. The study included observations of the Heart Rate (HR) during resting in supine and upright positions. In the supine position HR reached 64 beats/min and exceeded 80 beats/min in three of the 24 subjects.

When the subjects were in an upright position HR increased, diastolic blood pressure increased, pulmonary artery pressures decreased, and systemic and pulmonary vascular resistances increased comparing to when the subjects were in supine position. LV end-diastolic, end-systolic, and SV indexes decreased, so that cardiac index in general decreased even though the small increase in HR occurred [10].

When the subjects assumed an upright position arteriovenous oxygen difference increased to compensate for the decrease in cardiac index. Despite the decrease in LV filling as reflected by lower pulmonary pressures and LV volumes, not many differences were seen in ejection fractions between the supine (0.59) and upright (0.61) positions. Peak ejection rate and LV ejection time both decreased markedly from the supine to the upright position and as a reflex mediated of this, increases

in contractility and HR occur [10]. The pressure and volume responses of the left ventricle during upright exercise in humans differ from exercising in the supine position[10].

Higginbotham *et al.*[10]reported that during low levels of upright exercise, SV increases primarily as a consequence of an increase in LV filling pressure and end-diastolic volume (Starling mechanism) with a small contribution from a decreased end-systolic volume. During high levels of upright exercise, LV end-diastolic volume does not increase, despite a further increase in filling pressure; on the contrary, end-diastolic volume decreases, possibly as a result of reduced diastolic filling at high heart rates. SV at high levels of upright exercise is maintained through a progressive decrease in end-systolic volume [10].

Water immersion influence on cardiovascular drift during exercise in upright and supine position

Exercise combined with heat stress conditions in an upright position is associated with decreases in Central Blood Volume (CBV), Central Venous Pressure (CVP) and SV. The Cardiac Output (CO) can be maintained by an increase in HR until it reaches its maximum [11]. Beyond that point, the demand of blood flow to the skin and muscles is compromised. The increase in resting Forearm Blood Flow (FBF) with increasing body temperature shows a tendency towards a plateau in FBF [12].

These responses appear entirely to result from the moving in the blood volume from the central to the cutaneous veins rather than from any temperature induced reduction in inotropic state of the myocardium. It occurs when the vessels of the skin vasodilate, the cutaneous veins fill to a pressure and volume that is determined by the distance below the heart (hydrostatic force) and the compliance which is increased by heating [13]. If this shift of blood to the cutaneous veins causes the changes described above, then they could be prevented by counteracting the hydrostatic shifts of blood volume into veins by the standing position exercise and immersed in water.

Nielsen *et al.*[14] conducted an immersion experiment with eight men (aged between 18 and 31 years, 170 to 186 cm height, 63 to 85 Kg. bodyweight) as subjects. They were all physically active and they were in excellent health conditions. The experiment involved running in a swimming flume [15]. Results obtained from this experiment provided an important insight concerning hydrostatic pressure and cardiovascular responses [14].

Reductions in CBV, CVP [11,16], SV[17], and stabilization in the rate of increase in FBF at an external temperature of 38°C[12,17]. Hydrostatic pressure was hypothesized to counteract the postural and thermal effects in terms of the partitioning of blood volume [18].

Thus, Nielsen and colleagues hypothesized that subjects in a standing position should be able to perform exercise in warm water without showing decreases in CBV, CVP, SV, and MAP [14]. Their experiment results show a higher FBF during immersion, and a tendency to increase in CO and SV during immersion in water at a given external temperature and VO_2[14]. When the subjects were immersed up to the level of xiphoid process Risch and his collaborators showed a restored CBV and CVP in standing men compared with values observed in supine position [18].

In conclusion, the results by Risch*et al.*[18]support the idea that the shift of blood volume to cutaneous veins may contribute to the central circulatory effects of heat conditions during exercise [18]. These effects can counteract the immersion in water. The increase in hydrostatic pressure assists in the maintenance of SV and CO. This indicates that hydrostatic pressure can assist circulatory control and minimize the effect of heat stress precipitating C-V drift. However, this does not negate the effect of a loss of plasma volume contributing to C-V drift 105 [14].

A Research by Rowell [19] investigated the reduction in blood flow to the skin when exercise was performed in an upright position. They were interested to see whether the reduction of blood flow to the skin was also associated with exercise intensity and the percentage of oxygen required by tissues at that precise moment. They used a dye dilution technique to study blood flow.

Effects of dehydration and hyperthermia on blood volume during upright exercise versus supine exercise

The deleterious effects of dehydration and hyperthermia on the cardiovascular function during upright exercise were attenuated by elevation of CBV with supine exercise [20]. The dehydration during prolonged upright exercise in the heat which causes a progressive hyperthermia is associated with reductions in blood flow to the skin and Mean Arterial Blood Pressure (MAP) and increased plasma norepinephrine levels [21,22]. The blood flow to skeletal muscles during exercise also declines significantly [21].

However, the combined effects of hyperthermia and dehydration during upright exercise cause a severe deterioration in cardiovascular function. The effects of dehydration on the reduction of Skin Blood Flow (SBF) are not caused simply by a reduction in plasma volume because Mountain and Coyle [23] have shown that plasma volume expansion during dehydration failed to prevent reductions in FBF or to attenuate hyperthermia. It is likely that this amount of plasma volume expansion didn't have a great effect on the blood central volume even though this prevented hypovolemia and partially restored the SV [23].

The main purpose of the study by Gonzalez-Alonso *et al.*[20] was to determine if marked increases in CBV achieved by supine exercise prevents the reduction in Cutaneous Vascular Conductance (CVC) and MAP and whether it prevents increases in plasma norepinephrine which is characteristic of dehydration and hyperthermia during exercise in an upright position. The most important observations of this study were the reductions in MAP and CVC and increase in plasma norepinephrine concentration, typical of upright exercise when subjects are dehydrated and hyperthermic. These changes were totally absented during supine exercise despite identical conditions of dehydration and hyperthermia.

However, when subjects were dehydrated and hyperthermic in supine exercise compared with upright exercise it was restored two thirds of the reduction in SV and prevented one third of the increase in HR [20]. Saltin and Stenberg [24]found that supine exercise prevents or reduces the dehydration effects on SV and HR during exercise in the sitting position. In fact, they tested two subjects before when they were euhydrated at the beginning of exercise and after again when they were dehydrated after 195 minutes exercise duration.

They found 18% decline in SV during exercise in both supine and sitting positions. However, Saltin and Stenberg [24] observed that the CO was maintained during upright treadmill or bicycle ergometer 180 min prolonged exercise in contrast to the study by Gonzalez-Alonso *et al.* [20]which elicited a more severe hyperthermic stress. Nevertheless, Saltin and Stenberg [24]observed the MAP to be lower when subjects were dehydrated during upright prolonged exercise. The main observation in the study by Saltin and Stenberg [24]is that the supine position causes a significant increase in venous return, CBV and LV end-diastolic volume compared with upright exercise.

The observation that supine exercise even in dehydrated and hyperthermic subjects restored the forced vital capacity (FVC) and CVC and lowered plasma norepinephrine to levels observed during exercise in euhydrated subjects emphasizes the important effect of the CBV on baroreflex control of the skin and nervous system. Conversely, it appears that reductions in CVC and the increase in concentration of norepinephrine observed during upright exercise in dehydrated and hyperthermic subjects are caused by reductions in central blood volume.

MAP is also maintained at hydrated levels in these studies during supine exercise in dehydrated subjects, but declined significantly during upright exercise in dehydrated subjects[25].However, the reduced MAP with dehydration during upright exercise was also associated with significant increases in plasma norepinephrine and epinephrine (50-95%) and reductions in SBF of the forearm and FVC [20].

It has recently been shown that lowering of SV with dehydration during upright exercise is related to the combined influences of hypovolemia concomitant to losses of plasma volume induced by hyperthermia and dehydration [26]. During upright exercise a more lowered CBV and reduced MAP with dehydration and hyperthermia probably results in the unloading of both high and low-pressure baroreceptors [27]. The elevation of plasma norepinephrine associated with dehydration during upright exercise and the plasma norepinephrine restoration during supine exercise despite the dehydration supports the notion that cutaneous vasoconstriction is involved in vasoconstriction of the skin induced by dehydration [28].

In summary, these findings suggest that the decline in cutaneous vascular conductance and increased concentrations of plasma norepinephrine with dehydration during upright exercise, independently of hyperthermia, are associated with a reduction in CBV and lower arterial blood pressure. Also, increased CBV associated with exercise in the supine exercise reversed most of the reduction in SV experienced with dehydrated whereas the persistent reduction in SV was associated with an elevated HR and hyperthermia.

They observed moderate to high intensity exercise performed by endurance-trained men cycled in an upright standing position resulted in a reduction of blood flow to the splanchnic region of the body. Initially, the splanchnic blood flow was reduced by approximately 2 liters, but when exercise intensity was increased splanchnic blood flow was reduced further. This study further showed a relationship between the percentages of VO_{2ma}x per kilogram of body weight (required during the exercise) and flow of blood to the skin. An experiment by Rowell *et al.*[11]was conducted on 17 men who undertook exercise at different speeds, to achieve a percentage of VO2max from 26% to 96%. When the pace of exercise was reduced, and thus a reduced percentage of VO2max (range from 40 to 65 ml, not more than) men showed a high SBF. When the pace of exercise, and consequently the percentage of VO2max increased, the SBF decreased.

Thanks to the research methods using dyes to detect such evidence, it was possible to obtain images of the SBF in normal subjects during moderate and severe exercise on a motor-driven treadmill in an upright position. It was noted that the position of the body during the course of exercise was not considered a very influential factor on the reduction of SBF during exercise [11].

Effects of exercise on circulatory responses

The cardiovascular system is challenged by a bout of exercise with the need to increase blood flow to the exercising muscles at a rate that matches the metabolic requirements. According to the law of the heart described by Patterson and Starling [29]in 1914, the stretching of the myocardium produces an increase in the contraction of the cardiac muscle and a consequent improvement in SV.

These observations were consistent with the thesis of Guyton [30], that the primary cause of increased CO is believed to be the local vasodilation in skeletal muscle.Guyton *et al.*[31] observed in animal studies an increase in the effectiveness of the heart as a pump that by itself cannot increase the CO more than a small percentage, unless some simultaneous effect takes place in the peripheral circulatory system at the same time to translocate the blood from peripheral vessels to the heart.

The depressed SV was conventionally explained by a decrease in systemic venous return and cardiac filling in response to decreases in blood volume from dehydration or increased demands for blood flow to the skin (for thermoregulation). Increase in HR was interpreted as a compensatory mechanism to maintain the CO as the SV falls. More likely from these data the increase in HR is the primary cardiovascular response to maintain CO and hence exercise (a result of an increased sympathetic drive) with a secondary reduction in SV which is based on a shorter cardiac filling time.

Fritzsche *et al.*[32] and showed. that the latter explanation in fact is the correct one. They found that beta blockade during such exercise prevented the expected increase in HR and SV did not change. At the same time the cutaneous blood flow rose normally, and no differences were seen in CO compared with those in control subjects. These more recent discoveries suggest that the accepted explanation for drift (cardiovascular insult by a fall in blood central volume) is incorrect.

Relationship between heart rate and stroke volume during prolonged exercise

During prolonged exercise the decline in SV is affected by an increase in HR. Studies by Fritzsche *et al.*[32]showed that the decline in SV during prolonged incremental cycle-ergometer exercise protocol is associated by an increase in HR and - or by an increase in SBF[32]. An experiment conducted by Coyle and Fritzsche *et al.*[32]proved their theory. They led an experiment with seven active men who pedaled for 60 minutes at 57% of peak oxygen consumption in a neutral environment (27 C, over 40% relative humidity). These men received an oral dose of beta adrenoceptors blocker atenolol before exercise.

From the first 15 minutes of exercise SV and HR were similar. From 15 minutes to 55 minutes of exercise had noticed a decrease of 13% in SV which was associated with an increase in HR to 11%. Decline in SV was not associated with an increase in CBF [32]. From 5 to 15 minutes of exercise was noted an increase in CBF, but this remained stable from 20 to 60 minutes of exercise. However, from 5 to 15 minutes of exercise when the increase in HR was prevented by ethanol although the decline in SV was prevented as well. The SV was stable during exercise in both treatments [32].

In conclusion, the experiment shows important evidence. During cycle-ergometer prolonged exercise in neutral environment the decline in SV is associated to the increase in HR and it is not influenced by CBF [32]. At the same time a rise in HR maintains nearly constant CO [19]. Increased HR causes the reduction in the right ventricle filling time [33], and it decreases ventricular end-diastolic volume and therefore it decreases SV [34].

The increase in HR during prolonged cycling exercise is prevented when the intensity of exercise or the environmental stress is lower [35], or when the subjects are trained, euhydrated, heat-acclimated, and they familiar with the mode of exercise that they are using [35]. As it occurs during static exercise, a progressive increase in motor unit recruitment may lead to a progressive increase in HR or an increase in VO2max which leads to an increase in HR, that was observed in these studies [36]. It has been observed in these studies that the increase in HR may depend on several factors.

The contribution of endurance exercise acclimation of the body to the heat, familiarize to the type of exercise running, they can mitigate HR during prolonged exercise. Instead, the influence of the perceived fatigue and the core temperature can increase HR. Some researchers have found a direct relationship between core temperature and HR [37].

Effects of exercise and environmental conditions on heart rate

In the nineteenth century, it became clear that the heart generated electricity. The first systematic approach to the heart from electrical point of view was taken by Augustus Desiré Waller, at St Mary's

Hospital in Paddington, London. In 1911, he could appreciate the first clinical applications derived from its work. The step forward was made by Willem Einthoven with his galvanometer (built already in 1903), which was much more precise galvanometer used by Waller. Einthoven assigned the letters P, Q, R, S and T to the various waves and described the electrocardiographic of many cardiovascular diseases. For this discovery, he was awarded the Nobel Prize for Medicine in 1924. Einthoven was the first to propose using this method for the diagnosis of heart diseases.

The spectral analysis of Electrocardiogram (ECG) had been used since 1960. This analysis was used to determine the harmonic composition of the signal pulse rate of the heart and to allow a better understanding of cardiovascular autonomic control of heart. The harmonic component of HR variability is a very important component because this could provide further information on the cardiovascular dynamics in general.

As seen so farin the past**,**methods have been discovered and today they are used in cardiovascular and HR study. With regard to studies conducted years ago, today new theories can be asserted. One of these theories shows that the exercise training may decrease the risk of cardiovascular mortality and cardiac death. A regular exercise physical training is enlarged to modify autonomic nervous system balance [38]. A recent experimental study intended to assess how the effects of training exercise were acting in vagal activity. At the same time, this study also provided information on changes in electrical activity of the heart [38].

The exercise training also accelerates the physiological recovery of sympathetic nervous system. A study by Furlan investigated the effect of dynamic exercise in short- and long-term on neural control of HR [32]. In conclusion, the long-term training strongly affects autonomic control of HR. The endurance training causes an increase in Heart Rate Variability (HRV); in fact, it causes an increase in parasympathetic activity and it decreases the sympathetic activity in heart people at rest.

There are several physiological factors that affect the HR during exercise. These factors include: cardiovascular drift processes, hydration status, environment temperature (heat, cold), and altitude. Not only physiological factors may influence the relationship between HR and other parameters related to the exercise. The environment has much influence on HR. For example, the environmental temperature can have a strong influence on the relationship between HR and VO_{2max}. The environmental temperature influence (hot or cold) on the responses to exercise was studied in detail. In almost of the studies where exercise was performed during heat conditions, it was showing an increase in HR [38,22].

In an exercise carried out in heat conditions there is a rise in core temperature and an increase in HR even if it is keeping always the same exercise intensity. The cost of oxygen required by the body is exactly the same when the exercise is performed in altitude or when it is performed at sea level [39].However, the percentage of oxygen decreases by 30% in altitude environment. For this reason, when the exercise is performed at altitude the muscles require a greater percentage of oxygen. This is to compensate for the low amount of oxygen that there is in altitude environment. It has been shown that when submaximal exercise is performed at altitude SV increases because of an increase in HR [39].

The rate of the heart increased normally in every subject during mild exercise intensity, but there was a less speeding in three of the four subjects during higher levels of work intensity.The cardiac acceleration was reduced and it was abolished in two subjects in response to mild exercise intensity. Parasympathetic activity is reduced during mild and moderate running exercise, and after *1½ hour of training on treadmill the parasympathetic activity is disabled* [38].Following blockade of the sympathetic efferents (double blockade) the resting HR was above the normal control level in all subjects, but during mild exercise the HR was increased slightly in all subjects. During higher level of work HR had a small increase [38].

Impairment of the left ventricular function in athletes after strenuous prolonged exercise

Findings are indicative of a significant impairment in diastolic function of left ventriclein athlete's runners competing in ultramarathon running events (200 kilometers or 24 hours).According to a study by Niemela *et al.*[40], running almost continuously for 24 hours is an extreme form of physical stress that produced abnormalities in LV diastolic function.

The delay in mitral valve opening, the reduced peak speed of dimension increase in the prediastole and the prolongation of the duration of the fill were indicators of a compromised LV function [41]. The reduced posterior wall may have been as a consequence of altered behavior of the myocardium.However, the correlation between the distance and abnormalities in diastolic behavior does not necessarily indicate a causal effect. Abnormalities in diastolic function, however, were better related to the distance completed than to the alteration of the systolic function. The reversibility of the altered LV diastolic behavior was shown by measurements made on 6 athletes during the recovery of 2-3 days after a race.

The mechanisms that affect the LV filling has still not been fully understood [42]. Independent of these mechanisms, however, the abnormalities of resting LV diastolic function combined with the compromised systolic function are probably more deleterious to CO during exercise in diastole which is too short and this is caused by high heart rates. Cardiac filling could also be seriously compromised unless a significant dehydration is prevented, because the filling is partly dependent on loading conditions [43]. Faced with thousands of participants each year in various ultramarathons it is also important to note a clinically significant effect of myocardial ischemia on LV filling [44].

In patients with increased LV afterload in hypertension but without thickening of the posterior wall have also been reported to show abnormalities in isovolumic relaxation [45]. Ventricular filling in running athletes during the early diastole was previously attributed to their physical training [43]. Thus, running continuously for 24 hours appears to result in reversible abnormalities in resting LV filling and relaxation.

The relationship between systolic and diastolic volume is depressed during the last part of a 24 h race [46]. Coyle [47] reported that during prolonged cycling exercise the decrease in SV is provoked by the increase in HR which reduces the time for filling of the heart. The increase in HR may be a compensation for a reduction in cardiac function independent of changes in preload [48], which has been termed exercise-induced cardiac fatigue [49]. Furthermore, both a depressed systolic function and diastolic filling are noticed after an "Ironman" triathlon [50].

Prolonged endurance exercise may also provoke an increase in serum levels of the myofibrillar protein cardiac troponin T, but whether that indicates myocardial damage remains unknown [51]. However, LV systolic function may be compromised after exercise of 1-6 hours [52]. Goodman *et al.*[53] reported that 150 min of cycling at 70% VO2max resulted in a 12% increase in HR and a small decline in SV with no decrease in systolic performance. Though Lucia *et al.*[54] did not find any others change in LV systolic function after 2.5-4 h exercise. It may be in that study the exercise duration was not long enough to provoke any changes in contractile function of left ventricle [49].

LV diastolic filling was reduced after 4 h of exercise and there was a correlation between changes in E: A ratio (early/late diastolic flow

velocity) and VO_{2max}, with a greater reduction in those with the higher VO_{2max}[54]. It was suggested that the depression of E:A ratio may be associated to disturbance of intracellular Ca2+ metabolism post exercise LV function and cTnT in recreational marathon runners [55]. A reduction in E:A was taken to represent diastolic dysfunction, although it may be influenced by HR, which is related to the peak filling rate and inversely rated to the time to peak filling as by alterations in systolic function or loading conditions [53].

Four hours of cycling resulted in a progressive increase in HR despite maintained preload to the heart and it was not related to its systolic function or to myocardial damage [49]. There appeared to be no impact of VO_{2max} on the rise in HR or changes in LV contractile function. In contrast, the biphasic filling of the left ventricle indicated that, especially in those subjects with a high VO_{2max}, exercise resulted in a reduced global diastolic function[49].

The normal LV response to submaximal exercise in the upright position is associated to increases in both HR and CO [53]. Increases in SV are brought about largely by increased LV filling [56], particularly during exercise performed on a bicycle ergometer at a low to moderate intensity. Beyond this point, further increases in CO are brought about by continued increases in HR, and a decrease in End-Systolic Volume (ESV) secondary to increased contractility [56]. During prolonged running, an upward drift in HR and a decline in SV, as it was mentioned previous, characterize the cardiovascular response [19].

These responses are thought to be largely the result of gradual reductions in ventricular filling pressures and systemic blood pressure. The responses reflect a gradual decline in central blood volume, probably occurring secondary to a redistribution of blood volume to the venous capacitance vessels of the cutaneous circulation through peripheral vasodilatation [47].

Some investigations by Niemela *et al.*[46], have showed an impairment of LV function following prolonged exercise in ultramarathon runners. It is possible that a decline in ventricular output involves a depression in the contractile state. However, despite reports of a transient decline in systolic function by Niemela *et al.*[46] and diastolic filling characteristics by Niemela *et al.*[40]several studies have failed to report a decline in LV performance [57].

Studies by Nadel et al (58) reporting diminished LV function during prolonged exercise such as marathon running failed to control or monitor fluid intake, which is known to be important during prolonged effort and may contribute indirectly to the decline in LV filling pressures and SV. To date, there is very little information describing serial changes in LV function during prolonged exercise under controlled conditions, and it remains unclear if a true decline in LV systolic performance actually contributes to a reduction in SV and/or a reduction in cardiovascular performance [53].

Heart rate variability

Heart Rate Variability (HRV) has been recognized as the most powerful tool for estimating cardiac modulations of the heart [34]. HRV is a term used to describe changes in both of the following cases: changes that occur in the instantaneous HR and changes as variations that can be represented by a set of intervals during trends in HR [34]. There are regular fluctuations in HR which are mainly generated by the change in the control level of sympathetic and parasympathetic nervous system that acts on the heart [34].

The fluctuation in HR could therefore be considered as a variable or as a stimulus output, which is part of a network of feedback that is constantly in action. This feedback is regulated and controlled by the autonomic nervous system continuously [59]. Chess et al, showed evidence very important about the control that the autonomic nervous system implemented in HRV. In fact, they carried out an experiment where they implemented a selective blockade on autonomic nervous system in cats. Following the experiment, they determined that the high-frequency fluctuations in HRV were entirely mediated by parasympathetic nervous system [59].

Evidence demonstrated by Akselrod et al.(60) clearly explaining the importance of the parasympathetic and sympathetic nervous system activity in HRV. Indeed, Akselrod and his staff made a major discovery. They investigated the effect of selective and combined blockade in autonomic nervous system, and the inhibitory action of angiotensin converting enzyme in conscious dogs. This study indicated that the activity of the parasympathetic nervous system mediated fluctuations that were above 0:15 in HR. The activity of sympathetic nervous system and renin angiotensin mediated fluctuations that were under 0:15 Hz [60].

An experiment [61] was carried out on dogs. This experiment consisted of a daily exercise training that the dogs had to perform for six weeks. Following that exercise the dogs were placed in their cage where they were resting. The experiment shows that after the physical workout, the HVR increases by 74% and all animals survive to the next test that requires the achievement of ischemia process.

In conclusion, the present review offers an overview on the work of expert scientists who have investigated how changes in posture influence differences among cardiovascular responses during recovery from exercise. For instance, with the reliable technique of Doppler echocardiography, in a study conducted by Johnson and colleagues (1990) was observed a dramatic change (increase) in SV during recovery in the supine position, compared to change in SV (limited increase) during recovery in the upright position, following upright cycling exercise. Papers of these researchers contribute to clarify mechanisms involved in the haemodynamic responses with respect to SV, HR, SBF, MAP, CO, CVC, CBV and FVC, offering an interpretation of the integrated control functions of circulatory system, analyzing different responses and variables of the cardiovascular functional capacity. Despite this, to the present, some of these responses have not been fully understood, but very much discussed. It would be necessary to conduct new studies on that topic, considering also research findings over the past few years and provide not only an overview of the current state of knowledge, but also a new prospective for the mechanisms governing the complex phenomenon of haemodynamic responses during prolonged intense exercise.

Acknowledgements

This review paper was supported by Associate Professor Martin William Thompson, Pro- Dean of the Faculty of Health Sciences based at the University of Sydney. I thank Professor Martin Thompson who provided insight and expertise that greatly assisted the research and for sharing his pearls of wisdom with me during the course of this manuscript.

References

1. Bevegard S. Studies on the regulation of the circulation in man. Acta Physiol Scand. 1962; 57: 1-36.
2. Rushmer RF. Postural effects on the baselines of ventricular performance. Circulation. 1959; 20: 897-905.
3. Crawford MH, White DH, Amon KW. Echocardiographic evaluation of left ventricular size and performance during handgrip and supine and upright bicycle exercise. Circulation. 1979; 59: 1188-1196.
4. Hagberg JM, Montain SJ, Martin WH. Blood pressure and hemodynamic responses after exercise in older hypertensives. J Appl Physiol. 1987; 63: 270-276.

5. Robinson BF, Epstein SE, Beiser GD, Braunwald E. Control of heart rate by the autonomic nervous system: studies in man on the interrelation between baroreceptor mechanisms and exercise. Circ Res. 1966; 19: 400-411.
6. Rowell LB. Human cardiovascular control. New York: Oxford University Press. 1993; pp: 520.
7. Robinson BF, Epstein SE, Beiser GD, Braunwald E. Control of heart rate by the autonomic nervous system: studies in man on the interrelation between baroreceptor mechanisms and exercise. Circ Res. 1966; 19: 400-11.
8. Takahashi T, Okada A, Saitoh T, Hayano J, Miyamoto Y. Difference in human cardiovascular response between upright and supine recovery from upright cycle exercise. Eur J Appl Physiol. 2000; 81: 233-239.
9. Clausen JP, Trap-Jensen J. Heart rate and arterial blood pressure during exercise in patients with angina pectoris. Effects of training and of nitroglycerin. Circulation. 1976; 53: 436-442.
10. Higginbotham MB, Morris KG, Williams RS, McHale PA, Coleman RE, Cobb FR. Regulation of stroke volume during submaximal and maximal upright exercise in normal man. Circ Res. 1986; 58: 281-291.
11. Rowell LB, Marx HJ, Bruce RA, Conn RD, Kusumi F. Reductions in cardiac output, central blood volume, and stroke volume with thermal stress in normal men during exercise. J Clinic Invest. 1966; 45: 1801-1816.
12. Brengelmann GL, Johnson JM, Hermansen L, Rowell LB. Altered control of skin blood flow during exercise at high internal temperatures. J Appl Physiol J Appl Physiol Respir Environ Exerc Physiol. 1977; 43: 790-794.
13. Henry JP. The significance of the loss of blood volume into the limbs during pressure breathing. J Av Med. 1951; 22: 31-38.
14. Nielsen B, Rowell LB, Bonde-Petersen F. Cardiovascular responses to heat stress and blood volume displacements during exercise in man. Eur J Appl Physiol Occup Physiol. 1984; 52: 370-374.
15. Astrand PO, Englesson S. A swimming flume. J Appl Physiol. 1972; 33: 514.
16. Rowell LB, Murray JA, Brengelmann GL, Kraning KK. Human cardiovascular adjustments to rapid changes in skin temperature during exercise. Circ Res. 1969; 24: 711-724.
17. Nadel ER, Wenger BC, Roberts MF, Stolwijk JAJ, Cafarelli E. Physiological defenses against hyperthermia of exercise. Ann NY Acad Sci 1977; 301: 98-109.
18. Risch WD, Koubenec HJ, Beckmann U, Lange S, Gauer OH. The effect of graded immersion on heart volume, central venous pressure, pulmonary blood distribution, and heart rate in man. Pflügers Archiv Eur J Physiol. 1978; 374: 115-118.
19. Rowell LB. Human circulation: regulation during physical stress. New York: Oxford University Press. 1986; pp: 416.
20. Gonzalez-Alonso J, Mora-Rodriguez R, Coyle EF. Supine exercise restores arterial blood pressure and skin blood flow despite dehydration and hyperthermia. Am J Physiol. 1999; 277: 576-583.
21. González-Alonso J, Calbet JAL, Nielsen B. Muscle blood flow is reduced with dehydration during prolonged exercise in humans. J Physiol. 1998; 513: 895-905.
22. Gonzalez-Alonso J, Mora-Rodriguez R, Below PR, Coyle EF. Dehydration reduces cardiac output and increases systemic and cutaneous vascular resistance during exercise. J Appl Physiol. 1995; 79: 1487-1496.
23. Montain SJ, Coyle EF. Fluid ingestion during exercise increases skin blood flow independent of increases in blood volume. J Appl Physiol. 1992; 73: 903-910.
24. Saltin B, Stenberg J. Circulatory response to prolonged severe exercise. J Appl Physiol. 1964; 19: 833-838.
25. González-Alonso J, Calbet JAL, Nielsen B. Metabolic and thermodynamic responses to dehydration-induced reductions in muscle blood flow in exercising humans. J Physiol. 1999; 520: 577-589.
26. Mitchell JB, Voss KW. The influence of volume on gastric emptying and fluid balance during prolonged exercise. Med Sci Sports Exerc. 1991; 23: 314-319.
27. Mack GW, Thompson CA, Doerr DF, Nadel ER, Convertino VA. Diminished baroreflex control of forearm vascular resistance following training. Med Sci Sports Exerc. 1991; 23: 1367-1374.
28. Mora-Rodriguez R, Gonzalez-Alonso J, Below PR, Coyle EF. Plasma catecholamines and hyperglycemia influence thermoregulation during prolonged exercise in the heat. J Physiol. 1996; 491: 529-540.
29. Patterson SW, Starling EH. On the mechanical factors which determine the output of the ventricles. J Physiol. 1914; 48: 357-379.
30. Guyton AC. Regulation of cardiac output. New Engl J Med. 1967; 277: 805-812.
31. Guyton AC, Douglas BH, Langston JB, Richardson TQ, Abernathy B. Instantaneous increase in mean circulatory pressure and cardiac output at onset of muscular activity. Circ Res. 1962; 11: 431-441.
32. Fritzsche RG, Switzer TW, Hodgkinson BJ, Coyle EF. Stroke volume decline during prolonged exercise is influenced by the increase in heart rate. J Appl Physiol. 1999; 86: 799-805.
33. Turkevich D, Micco A, Reeves JT. Noninvasive measurement of the decrease in left ventricular filling time during maximal exercise in normal subjects. Am J Card. 1988; 62: 650-652.
34. Lazoglu AH, Glace B, Gleim GW, Coplan NL. Exercise and heart rate variability. Am Heart J. 1996; 131: 825-827.
35. Shaffrath JD, Adams WC. Effects of airflow and work load on cardiovascular drift and skin blood flow. J Appl Physiol Respir Environ Exerc Physiol. 1984; 56: 1411-1417.
36. Schibye B, Mitchell JH, Payne FC, Saltin B. Blood pressure and heart rate response to static exercise in relation to electromyographic activity and force development. Acta Physiol Scand. 1981; 113: 61-66.
37. Montain SJ, Coyle EF. Influence of graded dehydration on hyperthermia and cardiovascular drift during exercise. J Appl Physiol. 1992; 73: 1340-1350.
38. Furlan R, Piazza S, Dell'orto S, Gentile E, Cerutti S, Pagani M, et al. Early and late effects of exercise and athletic training on neural mechanisms controlling heart rate. Cardiovasc Res. 1994; 27: 482-488.
39. Klausen K, Dill DB, Horvath SM. Exercise at ambient and high oxygen pressure at high altitude and at sea level. J App Physiol. 1970; 29: 456-463.
40. Niemela K, Palatsi I, Ikaheimo M, Airaksinen J, Takkunen J. Impaired left ventricular diastolic function in athletes after utterly strenuous prolonged exercise. Int J Sports Med. 1987; 8: 61-65.
41. Gibson DG, Brown DJ. Measurement of instantaneous left ventricular dimension and filling rate in man, using echocardiography. Brit Heart J. 1973; 35: 1141-1149.

Citation: Rizzo L, Thompson MW. Cardiovascular Adaptations to Upright and Supine Prolonged Exercise: Heart Rate Modulation as a Factor of Cardiovascular Drift. J Phy Med Rehab 2017; 1:102.

42. Rankin JS, Olsen CO. The diastolic filling of the left ventricle. Eur Heart J. 1980; 1: 95-105.

43. Colan SD, Borow KM, Neumann A. Effects of loading conditions and contractile state (methoxamine and dobutamine) on left ventricular early diastolic function in normal subjects. Am J Card. 1985; 55: 790-796.

44. Poliner LR, Farber SH, Glaeser DH, Nylaan L, Verani MS, Roberts R. Alteration of diastolic filling rate during exercise radionuclide angiography: a highly sensitive technique for detection of coronary artery disease. Circulation. 1984; 70: 942-950.

45. Shapiro LM, Mckenna WJ. Left ventricular hypertrophy. Relation of structure to diastolic function in hypertension. Brit Heart J. 1984; 51: 637-642.

46. Niemela KO, Palatsi IJ, Ikaheimo MJ, Takkunen JT, Vuori JJ. Evidence of impaired left ventricular performance after an uninterrupted competitive 24 hour run. Circulation. 1984; 70: 350-356.

47. Ekelund LG. Circulatory and respiratory adaptation during prolonged exercise. Acta Physiol Scand. 1967; 292: 1-38.

48. Mcgavock JM, Warburton DER, Taylor D, Welsh RC, Quinney HA, Haykowsky MJ. The effects of prolonged strenuous exercise on left ventricular function: a brief review. Heart & Lung: J Crit Care. 2002; 31: 279-292.

49. Dawson EA, Shave R, George K, Whyte G, Ball D, Gaze D, et al. Cardiac drift during prolonged exercise with echocardiographic evidence of reduced diastolic function of the heart. Eur J Appl Physiol. 2005; 94: 305-309.

50. Whyte GP, George K, Sharma S, Lumley S, Gates P, Prasad K, et al. Cardiac fatigue following prolonged endurance exercise of differing distances. Med Sci Sports Exerc. 2000; 32: 1067-1072.

51. Mair J, Wohlfarter T, Koller A, Mayr M, Artner-Dworzak E, Puschendorf B. Serum cardiac troponinT after extraordinary endurance exercise. The Lancet. 1992; 340: 1048.

52. Manier G, Wickers F, Lomenech AM, Cazorla G, Roudaut R. Echocardiographic assessment of myocardial performance after prolonged strenuous exercise. Eur Heart J. 1991; 12: 1183-1188.

53. Goodman JM, Mclaughlin PR, Liu PP. Left ventricular performance during prolonged exercise: absence of systolic dysfunction. Clin Sci. 2001; 100: 529-37.

54. Lucia A, Serratosa L, Saborido A, Pardo J, Boraita A, Moran M, et al. Short-term effects of marathon running: no evidence of cardiac dysfunction. Med Sci Sports Exerc. 1999; 31: 1414-1421.

55. Shave R, Dawson E, Whyte G, George K, Gaze D, Collinson P. Altered cardiac function and minimal cardiac damage during prolonged exercise. Med Sci Sports Exerc. 2004; 36: 1098-1103.

56. Poliner LR, Dehmer GJ, Lewis SE, Parkey RW, Blomqvist CG, Willerson JT. Left ventricular performance in normal subjects: a comparison of the responses to exercise in the upright and supine positions. J Am Heart Ass. 1980; 62: 528-534.

57. Upton MT, Rerych SK, Roeback JR, Newman GE, Douglas Jr JM, Wallace AG, et al. Effect of brief and prolonged exercise on left ventricular function. Am J Card. 1980; 45: 1154-1160.

58. Nadel ER, Wenger BC, Roberts MF, Stolwijk JAJ, Cafarelli E. Physiological defenses against hyperthermia of exercise. Ann NY Acad Sci. 1977; 301: 98-109.

59. Chess GF, Tam RM. & Calaresu FR. Influence of cardiac neural inputs on rhythmic variations of heart period in the cat. American Journal of Physiology-Legacy Content. 1975; 228: 775-780.

60. Akselrod S, Gordon D, Madwed JB, Snidman NC, Shannon DC, Cohen RJ. Hemodynamic regulation: investigation by spectral analysis. Am J Physiol Heart Circ Physiol. 1985; 249: 867-875.

61. Hull Jr SS, Vanoli E, Adamson PB, Verrier RL, Foreman RD, Schwartz PJ. Exercise training confers anticipatory protection from sudden death during acute myocardial ischemia. Circulation. 1994; 89: 548-552.

Citation: Rizzo L, Thompson MW. Cardiovascular Adaptations to Upright and Supine Prolonged Exercise: Heart Rate Modulation as a Factor of Cardiovascular Drift. J Phy Med Rehab 2017; 1:102.