Effects of Over-reaching on Sleep Heart Rate

by

LINDSEY DAHL

A Thesis submitted to the Faculty of Graduate Studies of

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Abstract

Sleep heart rate has been suggested to provide a physiological marker for the detection of fatigue states associated with overtraining (Jeukendrup, Hesselink, Snyder, Kuipers, & Keizer, 1992). The large inter-individual variation in this measure indicates that a group research design may not be the most appropriate methodology for studying the effects of overtraining on the sleep heart rate.

The purpose of this study was to examine the effects of an increased training load on the sleep heart rates of elite triathletes using a single-participant research design to determine their value in the detection of the early stages of overtraining. Training load was expressed as a daily training impulse (TRIMP) and increased 266% during an intervention phase compared to baseline. Sleep heart rate was recorded each night and was not altered during the intervention phase compared to a baseline phase. During the recovery phase, sleep heart rate was decreased compared to both baseline and intervention, suggesting that the participants developed the parasympathetic form of overtraining.

It is concluded that the use of sleep heart rate is altered following over-reaching, but not during the actual process of over-reaching. Therefore, monitoring sleep heart rate after a period of increased training may reveal when an athlete may return to training. Further research involving longer recovery phases and other physiological parameters that supplement sleep heart rate are required in order to determine the ultimate benefits from monitoring sleep heart rate in an over-reaching environment.

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The number of times the heart contracts per minute is referred to as the heart rate and is a major factor contributing to the cardiac output of the cardiovascular system. Cardiac output plays an important role in providing oxygen and nutrient-rich blood to the tissues and organs of the body. In order to meet the changing metabolic demands of the tissues, heart rate is increased or decreased accordingly (Mitchell, 1985). It is through the actions of the autonomic nervous system, circulating hormones and reflex mechanisms that this regulation of heart rate is achieved (Dampney, 1994).

Heart rate monitoring is commonly used to determine the intensity of exercise and provide information of an athletes' training state. During submaximal exercise, heart rate is directly influenced by intensity, which allows for relative ease of monitoring training intensity (Karvonen & Vuorimaa, 1988). In addition, technological advancements have made it easier for athletes and the general public to monitor their training without interruption. Heart rate monitors, the most common form of technology for monitoring heart rate, consist of a transmitter and a receiver and can provide live feedback and/or record data for future review and/or analysis.

In addition to monitoring the intensity of exercise, athletes also monitor heart rates during rest. This enables the individual to measure the effectiveness of their program and determine if any adjustments to their training regimen need to be made. Traditionally, resting heart rate is analyzed before and after an endurance-training program. A decrease in the resting heart rate level after the training program can be one indicator of improved cardiovascular fitness. However, the more fit the individual, the harder it is to produce further cardiovascular improvements. In these individuals, longer and more intense training sessions are required for further improvements to occur.

Training programs for elite athletes regularly include highly intensified periods of training designed to overload the body and to improve performance (Polman & Houlahan, 2004). However, training at an intense level with inappropriate recovery leads to over-reaching, defined as the accumulation of training and/or non-training stress in which performance capacity is compromised and may require days to weeks of recovery (Halson et al., 2002). Continuation of training, while in an over-reached state, can lead to further performance capacity decrements and a condition termed the overtraining syndrome. In this case, the required recovery time before complete restoration of performance capacity is attained increases to several weeks or months. Tracking changes in resting heart rate in the morning has been proposed as a method in the identification of the overtraining syndrome (Dressendorfer, Wade, & Scaff, 1985). The basis of this practice relies on the premise that exercise causes an alteration in the autonomic activity resulting in an increased heart rate, which may persist above baseline levels during the recovery period. Thus an elevation in the morning resting heart rate may suggest that the autonomic system is functioning above normal levels and that recovery is incomplete. However, studies examining resting heart rate during consecutive overloading sessions have been inconsistent (Dressendorfer et al., 1985; Earnest, Jurca, Church, Chicharro, Hoyos, & Lucia, 2004; Halson et al., 2002; Hedelin, Wiklund, Bjerle, & Henriksson-Larson, 2000; Jeukendrup et al., 1992; Portier, Louisy, Laude, Berthelot, & Guezennec, 2001; Uusitalo, Uusitalo, & Rusko, 2000), which may be due to the training load and/or individual variation.

Sleep provides a period of rest and restoration (Adam, 1980) and may provide a more suitable setting for monitoring heart rate (Callister, Callister, Fleck, & Dudley,

1990; Jeukendrup et al., 1992). Studies examining the effects of over-reaching and the overtraining syndrome on sleep heart rate are limited and have yielded inconsistent results (Bosquet, Papelier, Leger, & Legros, 2003; Callister et al., 1990; Jeukendrup et al., 1992; Portier et al., 2001; Stray-Gundersen, Videman, & Snell, 1986). Despite the inconsistent findings, sleep heart rate is still appealing as a diagnostic tool for monitoring recovery status because it is easily attainable, inexpensive and does not require a laboratory setting.

These studies have examined the effects of over-reaching and/or the overtraining syndrome on resting heart rate before and after an overloading intervention. However, overtraining has been suggested to exist on a continuum consisting of acute fatigue, over-reaching and the overtraining syndrome (Fry, Morton, & Keast, 1991). Differentiating among the various forms of overtraining can prove difficult, as the symptoms associated with each overtraining phase tend to overlap with one another. Additionally, tremendous individual variation exists resulting in some symptoms occurring along different points along the continuum and in some instances, not being experienced at all. Therefore, daily monitoring, rather than pre- and post intervention measures, may provide better insight into the time course of potential markers of overtraining. Moreover, variables measured following an overloading intervention, whether they are valuable or not, merely suggest that the athlete has developed the overtraining syndrome which may prove to be too late in addressing overtraining in a meaningful way. Therefore, it is important to study potential markers that may predict the overtraining syndrome. Since over-reaching is a precursor to the overtraining syndrome, following the sleep heart rate as athletes engage in an over-reaching environment may provide valuable information that will allow

coaches and athletes to intervene in an appropriate manner. In order to examine the effects of over-reaching on sleep heart rate it is important to understand how heart rate is controlled and what may affect the mechanisms involved.

REVIEW OF LITERATURE

Autonomic nervous system

The autonomic nervous system functions to maintain homeostasis through the actions of sensory receptors, afferent and efferent nerves, and higher centers located in the central nervous system. The sympathetic and parasympathetic divisions make up the autonomic system. The parasympathetic system functions to conserve and restore energy, while the sympathetic system is called upon during situations of emergency or stress. Each division contains preganglionic and postganglionic neurons, which synapse with each other and go on to influence their intended target organ. Sympathetic preganglionic neurons are located in the thoracic and upper lumbar regions of the spinal cord, while parasympathetic preganglionic neurons are found in the brainstem and sacral region. Sympathetic postganglionic neurons are located in the paravertebral ganglia near the spinal cord, whereas the parasympathetic postganglionic neurons are situated near the target organ. Each division maintains a tone or certain level of activation, which can be adjusted in order to maintain the internal environment of the body.

Autonomic regulation of the heart rate

Control of heart rate is achieved through activity from both autonomic divisions on the sinoatrial node, or cardiac pacemaker, and the conduction velocity through the atrioventricular node. Both divisions are under the control of the cardiovascular control center located in the medulla oblongata and impose a certain amount of tone on the heart during rest. Input from afferent neurons and changes in emotional and physical stimuli can create alterations in the tone resulting in heart rate changes.

Stressful situations activate the sympathetic system, which produces a cardiovascular response consisting of increased heart rate and contractility, dilation of blood vessels in active muscles and constriction of vessels in the viscera and inactive muscles. Stress can present itself during many different mental and physical situations. Positional adjustments from supine to standing (Dixon, Kamath, McCartney, & Fallen, 1992), exercise (Furlan et al., 1993), making a presentation in front of a large audience, and rapid eye movement (REM) sleep (Somers, Dyken, Mark, & Abboud, 1993) are examples of stressors that are associated with elevated sympathetic activity and increased heart rates. The cardiac sympathetic nervous system is initiated by a set of central neurons in the hypothalamus and medulla oblongata (Jansen, Nguyen, Karpitskiy, Mettenleiter, & Loewy, 1995). Once initiated, cardiac preganglionic neurons release acetylcholine, which bind to receptors on the postganglionic neurons. In turn, the postganglionic neurons release norepinephrine, which bind to beta-receptors at the heart increasing heart rate and/or contractility. Cardiac sympathetic fibers from the left side of the body innervate the myocardium and upon stimulation will cause an increase in cardiac contractility, while the fibers on the right side innervate the sinoatrial node and

the atrioventricular node and influence the rate (Berne, 2004). Another stress-response of the sympathetic system results in the stimulation the adrenal medulla, which releases norepinephrine and epinephrine into the circulation. The effects of these hormones on the heart are similar to those of direct neuronal stimulation, except their effects are extended lasting up to 3 minutes after stimulation (Herd, 1991). The fate of unbound circulating catecholamines is excretion through urine. Therefore, plasma catecholamine levels reflect the sympathetic activity at a specific point in time, whereas the amount excreted in the urine reflects the intrinsic sympathetic activity over a longer period.

The parasympathetic system is responsible for decreasing the heart rate. Cardiac parasympathetic preganglionic neurons originate in the brainstem, exit through the vagus nerve and converge on postganglionic neurons near the heart. Both pre- and postganglionic neurons release acetylcholine, which is why the system is sometimes called the cholinergic system (Foss, 1998). Similar to the sympathetic system, the parasympathetic vagi branches into a right and left vagal nerve, which go on to affect different areas of the heart. The right vagi predominantly effects sinoatrial nodal firing rate, while the left vagi slows the conduction time of the action potential through the atrioventricular nodal tissue (Berne, 2004).

During normal rest, the parasympathetic tone is predominant, which has been demonstrated by a large increase in heart rate after pharmacological blockade of the parasympathetic effects with atropine and a slight decrease after sympathetic blockade with propranolol (Katona, McLean, Dighton, & Guz,.1982). Blocking the sympathetic and parasympathetic inputs simultaneously will eliminate any autonomic influence on the heart and will result in what is known as the intrinsic heart rate. The intrinsic heart rate is

approximately 100 beats per minute (bpm) in healthy sedentary individuals and decreases with improved fitness (Lewis, Nylander, Gad, & Areskog, 1980; Katona et al., 1982).

The action of the autonomic nervous system on the heart is influenced by various reflexes that are initiated from the many different stimuli that occur throughout everyday activities. These reflexes affect the activity levels of the sympathetic and parasympathetic systems. The input from these reflexes illustrates the complex neural network involved with producing the heart rate at any given point.

Baroreceptor reflex control of heart rate

The baroreceptor reflex is an important mechanism in the short-term regulation of blood pressure and is achieved through the inclusion of the autonomic nervous system and ultimately heart rate. Baroreceptors are stretch receptors located in the carotid sinuses and aortic arch, which initiate a cardiovascular reflex in response to changes in arterial blood pressure (Eckberg, 1980; Sanders, Mark, & Ferguson, 1989).

The carotid baroreceptors send regular afferent impulses from the carotid sinus through the nerve of Hering to the glossopharyngeal nerve, which carries on to the cardiovascular control center. The aortic baroreceptors send afferent impulses through the vagi to the cardiovascular control center. Changes in arterial blood pressure alter the firing rate of these impulses (Sanders et al., 1989; Carter, Banister, & Blaber, 2003). In addition, it has been shown that the aortic baroreceptors have more influence on the sympathetic nerve activity to the muscles during hypotension, while the carotid baroreceptors have more affect on the heart rate (Sanders et al., 1989).

An increase in blood pressure increases the firing rate from the baroreceptors, which will cause the cardiac sympathetic activity to be inhibited, lowering heart rate and peripheral resistance, which will drive the blood pressure down. Progressive increases of mean arterial pressure over an intermediate range (100-180 mmHg) have been shown to cause an inverse change in heart rate in monkeys (Cornish, Barazanji, Yong, & Gilmore, 1989), which is achieved by an inverse response between the cardiac parasympathetic and sympathetic divisions (Kollai & Koizumi, 1989).

A decrease in blood pressure will decrease the baroreceptor-firing rate and reflexively increase sympathetic outflow to the heart and blood vessels. Feeling light-headed after going from a seated to standing position is a common occurrence which results in a baroreceptor-induced increase in heart rate. Blood pressure is decreased in the upper regions of the body and sensed by the carotid baroreceptors, which initiates a cardiovascular reflex leading to an increase in blood pressure.

Metaboreflex control of heart rate

During exercise, if oxygen delivery to active skeletal muscle does not reach the requirement demands of the tissue, metabolites begin to accumulate. The metabolites initiate a response called the muscle metaboreflex, where afferent neurons signal the increase of sympathetic activity. This increase in sympathetic activity causes an increase in heart rate and mean arterial pressure providing the active muscle tissue adequate blood flow and perfusion pressure (O'Leary, 1993; Ansorge et al., 2002).

Renin-angiotensinogen control of heart rate

Renin is an enzyme synthesized in the kidney and released into the blood in response to decreased sodium chloride concentration, decreased extracellular fluid volume and/or decreased blood pressure. This enzyme cleaves angiotensin I into angiotensinogen II, which acts to increase fluid intake as well as influence the adrenal cortex to increase aldosterone. The result is an increase in sodium reabsorption by the kidney and ultimately increased sodium chloride concentration, extracellular fluid volume and blood pressure. It has been shown that this system effects the short-term control of the cardiovascular system (Akselrod et al., 1981). It appears as if the renin-angiotensin system plays a role in dampening the amplitude of blood pressure fluctuations (Akselrod et al., 1981). Due to the baroreceptor reflex, heart rate fluctuates in response to changes in blood pressure. Blood pressure and heart rate fluctuations have been shown to increase when the renin-angiotensin system is blocked, illustrating its' effects on stabilizing heart rate (Akselrod et al., 1985).

Bainbridge reflex control of heart rate

Increases in blood volume to the right atrium have been shown to increase the heart rate in humans (Boettcher, Zimpfer, & Vatner, 1982). This response, called the Bainbridge reflex, is initiated by stretch receptors located at the junctions of the right and left atria (Nonidez, 1937). The origin of this reflex is also neural, which ultimately effects the efferent impulses from both autonomic divisions to the sinoatrial node (Hakumaki, 1987). The end result is an increased sympathetic activation to the heart and periphery along with a decreased cardiac parasympathetic stimulation.

The increased right atrial pressure leads to an increased cardiac output, which translates into an increased arterial pressure (Vatner, Boettcher, Heyndrickx, & McRitchie, 1975). As mentioned above, the normal response of the baroreceptor reflex to an increase in blood pressure would be a decrease in heart rate. However, during situations of volume expansion, the baroreflex sensitivity (BRS) is reduced, which makes the receptors less responsive to changes in blood pressure (Cornish et al., 1989). Therefore, the Bainbridge reflex predominates during volume expansion and the heart rate increases despite a rise in blood pressure, which may serve as a protective mechanism to avoid blood pooling and backing up (Hakumaki, 1987).

Respiratory sinus arrhythmia

Heart rate fluctuates rhythmically during breathing. This respiratory sinus arrhythmia occurs as a result of an increased sympathetic tone during inspiration and increased vagal activity during expiration (Dampney, 1994; Eckberg, 1983; Katona & Jih 1975; Katona, Poitras, Barnett, & Terry, 1970). Due to the short latency of the vagal effects respiratory sinus arrhythmia is accomplished almost entirely by changes in parasympathetic activity (Akselrod et al., 1985; Katona et al., 1970).

Reflex and central factors are involved with producing the respiratory sinus arrhythmia (Berne, 2004). Inspiration reflexively increases the heart rate by activating stretch receptors in the lungs, which send impulses to the cardiovascular center. In addition, a Bainbridge reflex is initiated during inspiration when a transient increase in venous return occurs due to a drop in the intrathoracic pressure. Heart rate will then be

decreased by the baroreceptor reflex after a short time delay, as the increased volume of blood will elevate left ventricular pressure and, ultimately, arterial blood pressure.

Measurement of autonomic activity

Early studies examining the effects of training on autonomic function involve pharmacological blockade (Katona et al., 1982; Lewis et al., 1980). The parasympathetic activity can be observed with administration of a muscarinic receptor blocker, and the sympathetic activity with a cholinergic receptor blocker. With the use of a mathematical model, comparing the resting heart rates with the resulting heart rate after blockade will yield a value that reflects the contribution of each autonomic division (Katona et al., 1982). However, the resulting heart rate may be misleading since it reflects the heart rate that is totally removed of one of the autonomic divisions (Carter et al., 2003).

Analysis of heart rate variability (HRV) has more recently been used to provide indices of the autonomic system on the heart rate. HRV is the variation in time between beats (R-R interval) and provides a non-invasive marker of the cardiac autonomic control on the sinoatrial node (Goldberger, 1999). HRV data can be analyzed in the time domain and/or the frequency domain of the power spectrum (Achten & Jeukendrup, 2003; Aubert, Seps, & Beckers, 2003). Measuring HRV in the time domain requires plotting the R-R interval against time from which statistical parameters can be computed. Analysis in the time domain is relatively simple, however, it is unable to separate the parasympathetic control from the sympathetic (Aubert et al., 2003; Dixon et al., 1992). Power spectral analysis breaks the fluctuating time-dependent R-R intervals into its frequency components. This method of HRV analysis has shown that the

parasympathetic and sympathetic activity can be expressed at specific frequencies (Akselrod et al., 1981). The power spectrum can be divided into a very low-frequency (VLF), a low-frequency (LF) and a high-frequency (HF) component. The occurrence of the VLF component is unclear (Carter et al., 2003), whereas the HF component is mediated by the parasympathetic system, while the LF component has been shown to be mediated by both sympathetic and parasympathetic modulation (Akselrod et al., 1981).

Acute effects of exercise on heart rate and heart rate regulation

Exercise provides a stimulus that places a large demand on the metabolic pathways to generate energy. The responsibility of the cardiovascular system is to provide the exercising muscles with the appropriate amounts of oxygen and nutrients to generate enough energy. Therefore, heart rate is increased during exercise and is directly related to the intensity of the exercise. This exercise-induced elevation in heart rate is mainly mediated by the autonomic nervous system (Arai et al., 1989), which is initiated by the central command centers and metaboreflex mechanisms (Mitchell, 1985).

At low to mild intensities (<100 bpm) heart rate is increased by the progressive withdrawal of the parasympathetic tone (Robinson, Epstein, Beiser, & Braunwald, 1966; Victor, Seals, & Mark, 1987). At intensities greater than this, further increases in heart rate are a result of more parasympathetic withdrawal along with increased sympathetic activation (Arai et al., 1989; Casadei, Cochrane, Johnston, Conway, & Sleight, 1995; Dixon et al., 1992; Robinson et al., 1966; Victor et al., 1987). Nearing maximal intensity, the parasympathetic tone is removed and the heart rate is mediated by the sympathetic system (Tulppo, Makikallio, Seppanen, Laukkanen, & Huikuri, 1998).

At the cessation of exercise, stimulus from the cardiovascular control center in the brain and afferent impulses from reflex mechanisms are reduced resulting in a rapidly decreasing heart rate (Carter et al., 2005) before settling into a slow decline to pre-exercise levels (Arai et al., 1989; Furlan et al., 1993; Maehlum, Grandmontagne, Newsholme, & Sejersted, 1986). The length of time required to reach pre-exercising levels depends on the length and intensity of the exercise performed (Maehlum et al., 1986). After control participants exercised for only 15-minutes at 50% of their maximal power output, the LF:HF ratio, a sympathetic indicator, was similar to exercise levels 15-minutes following the exercise (Dixon et al., 1992). When exercise is performed at a greater intensity until exhaustion, such as a maximal treadmill test, the LF:HF ratio has been shown to be elevated as long as 24-hours (Furlan et al., 1993).

Hydration status also influences the autonomic activity on the heart (Carter et al., 2005). The HF power was increased during rest and recovery from exercise while in a hypohydrated state. However, heart rate variability and heart rate increased indicating a blunted response of the autonomic nervous system and a cardiac instability (Carter et al., 2005). This study also demonstrated that heat stress, while hypohydrated, exacerbated this response.

Exercise also stimulates the sympathoadrenal system, which causes an elevation in plasma and urinary catecholamines (Pequignot, Peyrin, Mayet, & Flandrois, 1979). The presence of increased levels of circulating catecholamines may contribute to the elevations in heart rate during the recovery period. Elevated urinary norepinephrine excretion was evident following 15-minutes of exercise at 80% VO_{2max}, while exhaustive exercise at the same intensity caused an increase in urinary epinephrine and

norepinephrine. The levels of norepinephrine were still higher than pre-exercising levels after 2-hours of recovery, which may serve a purpose in glycogen resynthesis since catecholamines are involved in lipid mobilization during the post-exercise oxygen consumption period (Pequignot et al., 1979).

Plasma catecholamine levels are at their lowest during rest (Dela, Mikines, Von Linstow, & Galbo, 1992). However, an intense exercise bout may elevate the resting plasma catecholamine level above baseline, indicating that the sympathetic system is still active (Sagnol et al., 1989; Sagnol et al., 1990).

During an extremely stressful 24-hour ultra-marathon, the plasma norepinephrine was increased immediately following the race, which persisted over the next 24-hours of recovery (Sagnol et al., 1989). A year later, the same group came to the same conclusion when conjugated norepinephrine remained elevated 24-hours after a triathlon (Sagnol et al., 1990; Achten & Jeukendrup, 2003). Conjugated norepinephrine has a longer half-life than free norepinephrine, which may be a better indicator of sustained sympatho-adrenal stimulation (Sagnol et al., 1990).

Chronic effects of exercise on heart rate and heart rate regulation

It is well known that endurance training results in lower resting and exercising heart rate known as athletic bradycardia (Catai et al., 2002; Dela et al., 1992; Dixon et al., 1992; Furlan et al., 1993; Goldsmith, Bigger, Steinman, & Fleiss, 1992; Iellamo et al., 2002; Katona et al., 1982; Lewis et al., 1980; Yamamoto, Miyachi, Saitoh, Yoshioka, & Onodera, 2001). Along with enabling the heart to not work as hard during submaximal exercise, athletic bradycardia increases long-term HRV and parasympathetic indices,

suggesting that long-term exercise could provide a cardio protective function in susceptible individuals (Carter et al., 2003; Somers, Conway, Johnston, & Sleight, 1991; Ueno & Moritani, 2003). Theoretically, athletic bradycardia can be achieved through increased parasympathetic activity, decreased sympathetic activity, decreased intrinsic heart rate or any combination of these factors (Katona et al., 1982). Studies that have looked at the effects of training on the autonomic divisions have been carried out through both pharmacological blocking and HRV analysis.

During exercise, following endurance training, heart rate is lower at a given submaximal workload since cardiac output is maintained by a greater stroke volume. In addition, sympathetic activity is decreased at the same submaximal workloads, along with a greater parasympathetic drive (Ekblom, Kilbom, & Soltysiak, 1973). The sympathetic activity is reduced at submaximal workloads as a result of a lower metaboreflex activation since the muscles are more efficient at generating energy and producing less metabolites (Mostoufi-Moab, Widmaier, Cornett, Gray, & Sinoway, 1998). In addition, the lower sympathetic activity decreases the amount of circulation catecholamines at a given workload, which contributes to a lower heart rate response (Cousineau et al., 1977).

Following exercise, the recovery of the resting sympathovagal balance, measured by the LF:HF ratio, after a 15-minute bout of exercise is restored quicker in trained participants compared to sedentary controls (Dixon et al., 1992). After exercise, the heart rate is gradually decreased mainly through the restoration of the resting parasympathetic tone (Arai et al., 1989). Together, these suggest that endurance training enhances vagal

activity, which returns the heart rate to pre-exercising levels more quickly during recovery.

At rest, studies have shown inconsistent results for the cause leading to athletic bradycardia. Comparisons between trained and untrained participants have displayed that lowered intrinsic heart rates are the sole cause in the lower heart rates (Katona et al., 1982; Lewis et al., 1980). The lower intrinsic heart rate is likely due to cardiac enlargement commonly seen in endurance trained athletes. Under normal conditions, a stretch to the atrium will invoke a Bainbridge reflex causing the sinoatrial node to increase its firing rate. However, when the volume of the heart is increased through training, the chronic stretch may inhibit the sympathetic response and lower the intrinsic heart rate (Lewis et al., 1980). It has also been proposed that a biochemical adaptation of the myocardium may possibly lead to a lower intrinsic heart rate, where the heart becomes more efficient in energy generation and utilization following training (Katona et al., 1982). A longitudinal study came to a similar conclusion, reporting a lower intrinsic heart rate following an endurance training program in young (21 years) and middle-aged (53 years) participants after both groups showed no change in the autonomic system despite lowered heart rates (Catai et al., 2002).

Other studies have demonstrated that athletic bradycardia is a reflection of an autonomic adaptation. At rest, there is a parasympathetic predominance in the sympathovagal balance. Heart rate variability analysis has shown that training decreases heart rate due to an increase in the parasympathetic tone (Dixon et al., 1992; Iellamo et al., 2002; Yamamoto et al., 2001). This has been demonstrated by an elevation of the HF region and a decrease of the LF region of the power spectrum. A possible explanation for

the increase in the parasympathetic tone may be from the increase in blood volume that follows training (Convertino, Mack, & Nadel, 1991). The increased blood volume causes an elevation in the pressure on artery walls, which in-turn causes a greater activation of the baroreceptor impulses and an increase in parasympathetic tone.

Indeed, the tone of the baroreceptor impulse may be greater following training; however, this does not always translate into a greater sensitivity and management of blood pressure. In borderline hypertensive participants, baroreceptor sensitivity has been shown to be increased following 6 months of endurance training (Somers et al., 1991). However, in normotensive sedentary participants, no change was seen after 5 months of aerobic training (Loimaala, Huikuri, Oja, Pasanen, & Vuori, 2000). Moreover, the baroreceptor response to steady-state hypotensive stress is attenuated in endurance trained participants, which may be attributed to sympathovagal imbalance caused by an increased parasympathetic tone (Smith, Graitzer, Hudson, & Raven, 1988; Smith, Hudson, Graitzer, & Raven, 1988).

Interestingly, it has also been shown that athletes at the peak of their training may have a more complex neural integration compared to detrained athletes (Furlan et al., 1993). Participants training at a high intensity achieved similar bradycardia as the detrained athletes while demonstrating an elevation in LF power. This suggests that the sympathetic system may remain elevated following the heavy training session performed during the day. This type of response may serve to prepare the athletes for competition by enabling themselves to engage their sympathetic system more efficiently during their sport/event.

Plasma-free levels of catecholamines reflect the activity of the sympathetic system as it relates to acute stress (Lehmann, Foster, Dickhuth, & Gastmann, 1998). Exercise is a form of stress associated with an elevation in plasma-free catecholamine concentration (Dela et al., 1992). Following effective endurance training, plasma levels of catecholamines are lower at the same absolute workload (Lehmann et al., 1988; Lehmann, Baumgartl, Wiesenack, Seidel, Baumann, Fischer et al., 1992). In other words, exercise at the same workload will be less stressful after training.

Sleep

Sleep architecture

Sleep can be described as is a period of rest and restoration, where important cognitive, reparative and restorative processes occur in order to maintain health and homeostasis (Murali, Svatikova, & Somers, 2003). Electroencephalographic recordings of brain wave activity show that the brain is continuously active during sleep and that different patterns exist giving rise to the stages of sleep (Rechtschaffen & Kales, 1968). Two distinct states of sleep exist, non-rapid eye movement (NREM) and REM. NREM sleep is further divided into stages I, II, III and IV. Stages I and II are considered light sleep, whereas the deeper third and fourth stages are usually combined and referred to as slow wave sleep (SWS).

The construction of sleep, or sleep architecture, maintains a cyclical pattern beginning with stages I and II followed by SWS and finally REM sleep (Penzel, Kantelhardt, Lo, Voigt, & Vogelmeier, 2003). A typical cycle lasts from the start of one REM period to the start of the next which usually lasts approximately 90 to 100 minutes

(Miller & Horvath, 1976) and may be repeated up to six times per night (O'Connor & Youngstedt 1995; Penzel et al., 2003). Approximately 45% of sleep occurs in stage II, while 20% of sleep is slow wave sleep, 20% REM sleep and 15% stage I sleep (Miller & Horvath, 1976). The order of appearance of the sleep stages is fairly consistent, however, the time spent in each stage is not identical across cycles. The greatest amount of time spent in SWS is greater during the early portion of sleep, whereas the time spent in REM sleep is greatest in the later portion (Cajochen, Pischke, Aeschbach, & Borbely, 1994; Degaute, van de Borne, Linkowski, & Van Cauter, 1991; O'Connor & Youngstedt, 1995).

Sleep architecture following exercise

The functional role of sleep is generally assumed to serve as a period of recovery and restoration for the body and the brain from the activities encountered during the waking period (Adam, 1980). Therefore, it is not surprising that the architecture of a normal nights' sleep may be altered when exercise is performed during the day. Studies have shown that the magnitude of the alterations and the stages that are affected depend on the intensity and duration of the exercise, the level of fitness of the participants and the time of day that the exercise is performed.

The sleep period that is most analyzed following daytime exercise is SWS. This period is considered the deepest phase of sleep and it has been suggested that most of the recovery from the daily energy expenditure occurs during this period (Shapiro, Bortz, Mitchell, Bartel, & Jooste, 1981). Studies lending support to this concept have shown that the amount of sleep time spent in SWS is increased after a day consisting of exercise (Griffin & Trinder, 1978; Hague, Gilbert, Burgess, Ferguson, & Dawson, 2003; Shapiro,

Griesel, Bartel, & Jooste, 1975; Shapiro et al., 1981). Moreover, the increased time in SWS sleep appears to be accompanied by a decrease in REM sleep (Shapiro et al., 1975; Shapiro et al., 1981)

The increase in SWS and decrease in REM sleep in two highly trained participants has been shown to be related to the intensity and duration of the exercise performed (Shapiro et al., 1975). Furthermore, when the exercise becomes extreme, such as in a 92 km marathon, SWS is increased the night of the exercise and remains increased during the following night when no exercise is performed during the day (Shapiro et al., 1981). The increase in SWS during the first night was achieved predominantly by an increase in stage IV and then displayed a balance between stage III and IV the next night. The results of these studies are supported by the reversal of the time spent in SWS and REM sleep when regular exercisers engage in a sedentary day (Hague et al., 2003).

Level of fitness also plays a role in the effect of exercise on the amount of SWS encountered. Comparing fit and unfit participants following a sedentary day revealed that fit participants have higher levels of SWS, which was due mostly to greater stage III sleep (Griffin & Trinder, 1978). Both groups completed a 7.3 km run during the evening, which resulted in an increase in SWS time for the fit participants and no change in the unfit subjects. It may be possible that the unaccustomed exercise in the unfit subjects attributed to discomfort during the sleep period reducing the time spent in SWS (Griffin & Trinder, 1978).

Another possible reason may be that total SWS sleep may not change however there may be a redistribution of SWS duration per each cycle. When moderate exercise was performed in the afternoon by healthy participants, the first half of sleep showed an

increase in stage III sleep and was most evident during the period preceding the first REM episode, whereas a non-significant decrease occurred in the second half. When the same exercise was performed in the morning, stage II sleep decreased and was accompanied by a slight, non-significant, increase in stage IV sleep. It was concluded that when moderate exercise is performed early in the day, the remainder of the waking day is sufficient for full recovery, while recovery processes may be present during the early portion of sleep if the exercise is performed later in the day (Horne & Porter, 1975).

It has also been shown that SWS durations were significantly increased during the period preceding the first rapid eye movement episode after performing 80-minutes of hand dynamometer exercise (Browman, 1980). Despite the inability of the static exercise performed by the participants to change the whole-night SWS level, the time of day that it was performed (2 hours before bedtime) may be responsible for the effects of the exercise to be present during the early part of the night.

Although it is still unclear if the REM stage has a functional role in the body's recovery process, it has been shown to decrease in response to exercise allowing for greater time spent in SWS (Shapiro et al., 1975; Shapiro et al., 1981; Torsvall, Akerstedt, & Lindbeck, 1984; Walker et al., 1978). However, in other cases the REM period is unchanged (Browman, 1980; Buguet, Roussel, Angus, Sabiston, & Radomski, 1980). This may be attributed to the lower intensity of exercise performed in the latter studies.

Sleep heart rate

The general trend over an entire night is for the sleep heart rate to decrease (Aldredge & Welch, 1973; Cajochen et al., 1994; Snyder, Hobson, Morrison, & Frank, 1964). Heart rate has been shown to progressively drop 5-10% from sleep onset to wake-up (Snyder et al., 1964). Looking more closely at the sleep stages show that the decrease in heart rate found over an entire night is more than just a gradual decline from start to finish.

Average heart rate during the waking state and each of the sleep states are not equal. Heart rate during NREM is lower than the resting heart rate in the waking state (George & Kryger, 1985; Khatri & Freis, 1967). The highest average heart rate during the sleep period is found during REM sleep (Khatri & Freis, 1967) and is similar to the heart rate during the resting waking state (Somers et al., 1993). When broken down into its' cycles, sleep heart rate and the average heart rate for each stage within a cycle decreases or remains stable across successive cycles (Aldredge & Welch, 1973). Therefore, the lowest mean heart rate values for each stage are most likely to occur later in the sleep period.

During each cycle of sleep, heart rate gradually declines through the NREM stages before increasing during REM sleep (Aldredge & Welch, 1973). The heart rate is consistently lower during NREM sleep before and after REM sleep. Mean heart rate during the 20 minutes before and after REM sleep has been shown to be lower 89% and 87% of the time, respectively (Snyder et al., 1964; Versace, Mozzato, De Min Tona, Cavallero, & Stegagno, 2003). Additionally, the dynamics of the transition from NREM

to REM sleep reveal that heart rate increases abruptly and rapidly, while the transition back to NREM sleep is more gradual (Cajochen et al., 1994).

Cardiac autonomic control during sleep

The contributions of the parasympathetic and sympathetic systems during sleep depend on the stage of sleep. The increase in heart rate during REM sleep can be attributed to a slight increase in sympathetic activity, but more importantly to a withdrawal of parasympathetic activity (Zemaityte, Varoneckas, & Sokolov, 1984). During NREM sleep, sympathetic activity is at its' lowest while parasympathetic activity is at its' greatest (Monti, Medigue, Nedelcoux, & Escourrou, 2002; Versace et al., 2003). The progressive decrease in heart rate that occurs as NREM sleep progresses from stage I to IV is produced by a progressive increase in parasympathetic activity and a decrease in sympathetic input (Zemaityte et al., 1984). Additionally, parasympathetic activity has also been found to be greater during the last sleep cycle (Monti et al., 2002), which seems appropriate since the lowest mean heart rate for each stage normally occurs during the last cycle.

Sleep heart rate response to acute exercise

Following exercise, the heart rate initially decreases rapidly, which is followed by a slow decline towards baseline levels (Arai et al., 1989; Furlan et al., 1993; Maehlum et al., 1986). The length of time for the heart rate to reach pre-exercising levels depends on the intensity of the exercise and the individuals' level of fitness (O'Connor, Crowley, Gardner, & Skinner, 1993). Following a 15-20 km run in the afternoon, trained

participants showed that heart rates before and after sleep were elevated 5 and 3 bpm, respectively (Torsvall et al., 1984). Furthermore, when the training load was increased the next day to either a 30 or 42 km run, the heart rate increased another 7 bpm before bed and 3 bpm upon rising (Torsvall et al., 1984). These results imply that heart rates may have been elevated throughout the night as a result of daytime exercise and the magnitude of this elevation is affected by amount of exertion of the exercise. Studies that have examined the effect of a single exercise bout on sleep heart rate have produced equivocal results (Bunnell, Bevier, & Horvath, 1983; Bunnell, Bevier, & Horvath, 1985; Mischler et al., 2003; O'Connor et al., 1993; Roussel & Buguet, 1982; Walker et al., 1978), which may be due to the participants fitness levels, the extent of the exercise stimulus and the time of day the exercise was performed.

In untrained individuals, light exercise (2.4 km jog at a self-regulated pace) was able to produce a strong trend ($P = 0.02$) for the mean sleep heart rates measured at hourly intervals to be elevated (Walker et al., 1978). In the same study, sleep heart rates in trained runners did not change after completing a 10.2 km run at a greater intensity (Walker et al., 1978). Slow wave sleep was also unaltered in this group indicating that complete recovery may have been achieved during the waking hours.

Contrasting the results in the untrained participants, 30 minutes of cycling at 75% VO_{2max} produced no change in sleep heart rate (O'Connor et al., 1993). These results may have not reached significance because the time frame from which the participants could choose to exercise was very large (between 6 am and 5 pm). If the participants chose to exercise in the morning, then recovery from exercise of this intensity may have been completed during the day. Likewise, no change in sleep heart rate occurred during

the night after a single bout of exercise of the same length and intensity after the participants improved their level of fitness following a 12-week training period.

In moderately active participants, single exercise sessions that maximally tax the cardiovascular system have been shown to increase the sleep heart rate (Bunnell et al., 1983; Bunnell et al., 1985). When energy expenditure is relatively high but total energy expenditure is low, such as a standard treadmill exercise bout to exhaustion, the sleep heart rate may only be elevated during the early portion of the night (Bunnell et al., 1983). The duration of the elevation during the night can be increased when the exercise increases the total energy expenditure by lowering the relative energy expenditure, such as exercising at 50 – 70% VO_{2max} until exhaustion (Bunnell et al., 1985). In addition, further analysis revealed that females and participants with the lowest fitness levels had higher sleep heart rates (Bunnell et al., 1983).

Therefore, it appears that sleep heart rate may be increased if exercise is performed during the day and the energy expenditure required to complete the exercise is high. However, this may not be true for all individuals, therefore the time of day and the level of fitness of the individual should also be considered.

An acute exercise training bout can alter the activity of the sympathetic system, which in turn may change the morning urinary catecholamine excretion (Bunnell et al., 1983; Bunnell et al., 1985).

When exercise is performed to exhaustion, using a relatively large energy expenditure, moderately active participants have shown that the morning excretion of epinephrine is decreased from baseline, whereas norepinephrine is increased (Bunnell et al., 1985). In addition, the largest increases occurred in the least-fit participants.

However, norepinephrine excretion is not increased when exercise requiring much lower energy expenditure is performed (Bunnell et al., 1983). This observation suggests that the relatively low energy expenditure of this test, performed in the afternoon, was likely not adequate enough to produce an increase in sympathetic activity during the night. Therefore, when an exercise bout is intense and long enough, as is the case in most overloading sessions, an elevation in the sympathetic tone may be present throughout the night such that an increase in nocturnal catecholamine excretion may occur.

Sleep heart rate response to chronic exercise

It is well documented that aerobic training leads to lower resting and exercising heart rates. Studies examining the effects of training on the sleeping heart rate have produced equivocal results. Training programs and the participants' level of fitness should be considered when evaluating the heart rate response.

In sedentary men, one study showed that sleep heart rate decreased faster and reached plateau earlier following a 12-week endurance program (O'Connor et al., 1993), whereas another study of similar duration showed no change (Sedgwick, Craig, Crouch, & Dowling, 1974). In the former study, exercise sessions were performed three times a week, while two per week exercise sessions were used in latter study. The extra day per week, in combination with slightly more intense training sessions, may be responsible for a greater improvement in cardiovascular fitness and the change in the sleeping heart rate dynamics during the early portion of the night. Another study reported a small decrease of 1.9 bpm in sleep heart rate, measured as the lowest heart rate attained for at least 1-hour, following a 20-week endurance program (Wilmore et al., 1996). In this study, the

participants showed a 16% improvement in VO_{2max} at the end of the training period. Further improvements in aerobic capacity have produced larger sleep heart rate decreases (Pichot et al., 2002). This study reported a 9.7 bpm decrease in sleep heart rate and a 20% improvement in their VO_{2max} in sedentary individuals following a more intense training program lasting eight weeks.

In the above studies, all of the participants who were subjected to the training programs were initially sedentary. In physically active female participants, moderate training does not seem to be appropriate to significantly alter sleep heart rate. The subjects were split into a control group and an exercise group, which underwent a 5-week moderate aerobic training program. The training program produced a training effect of lowered submaximal exercising heart rates, however did not significantly decrease sleep heart rate. Therefore, in physically active females, a 5-week program of moderate intensity (1-hour at 80% HR_{max}, 3-times per week) may not be long enough to induce a heart rate change during the night (Pigozzi et al., 2001).

Exercise effects on cardiac autonomic control during sleep

It is known that during an exercise bout there is a withdrawal of the parasympathetic activity and an increase in the sympathetic (Arai et al., 1989; Dixon et al., 1992). It is possible for the sympathetic activity to remain elevated up to 24-hours after a single bout of maximal exercise (Furlan et al., 1993). Therefore, a possibility exists that the sympathetic activity is active above normal resting levels and may persist throughout the night leading to an elevated heart rate.

Endurance training lowers resting heart rate, which may be partially explained by an increase in parasympathetic activity. During sleep, cross-sectional studies comparing trained and untrained participants have also revealed significant differences in the HRV power spectrum pertaining to the parasympathetic system (Goldsmith et al., 1992). Both HF and LF components were significantly higher during sleep in the trained participants. Although the LF component is suggested to reflect both sympathetic and parasympathetic input (Pigozzi et al., 2001), the activity of the sympathetic system is low during periods of rest and sleep and does not contribute as much to this component during sleep, specifically NREM sleep.

Moderately trained athletes (4-6 hours of aerobic activity per week) have also been shown to possess greater parasympathetic indices during SWS leading to lower sleep heart rates compared to sedentary individuals (Buchheit, Simon, Piquard, Ehrhart, & Brandenberger, 2004). However, a group of participants categorized as being highly trained (more than 18 hours of aerobic activity per week), showed similar parasympathetic indices as the sedentary participants despite significantly lower sleeping heart rate. This may be a result of a more complex neural interaction seen in highly trained athletes (Dixon et al., 1992).

Longitudinal studies have produced inconclusive results regarding changes in HRV indices during sleep following an endurance training program. Sedentary participants increased HRV and parasympathetic tone at the end of an intense 8-week training program (Pichot et al., 2002). Meanwhile, a 3-month training program effective in lowering sleep heart rate with sedentary male participants produced no significant changes in any of the parasympathetic or sympathetic indices of the HRV (Catai et al.,

2002). Based on these results, the authors associated the decreased sleeping heart rate with a decreased intrinsic heart rate, which has been reported in trained athletes during the awake resting state (Katona et al., 1982). Another study reported no difference in the LF:HF ratio, a sympathetic indicator, between awake and sleep states in healthy female participants who trained for 5-weeks (Pigozzi et al., 2001). The normal response would be a decrease in the LF component and an increase in the HF component, lowering the LF:HF ratio, when transitioning from wake to the early stages of NREM sleep. The fact that no difference was seen suggests a persistent sympathetic stimulation. The authors allude to the possibility that the lingering effects of an exercise training session could be responsible for the increased sympathetic tone seen during the night in their participants.

Overtraining

The term overtraining has been used generically to describe decreased performance in response to an imbalance between stress and recovery (Achten & Jeukendrup, 2003; Fry et al., 1991). The stress imposed on the body may come from physical, psychological and/or social sources. Due to the negative effects on performance, it is thought to be avoided at all costs in an athletic environment. However, the state of overtraining is dynamic and exists on a continuum with both positive and negative components (Kentta & Hassmen, 1998). Training consisting of high volume and intensity is necessary for performance improvements. The downfall of this type of training is that performance suffers if it is done incorrectly. Symptoms that differentiate between positive or negative overtraining are difficult to identify across individuals.

Despite the difficulty, markers that relate to the states of overtraining have been extensively studied.

The overtraining continuum

A common training strategy is based on the overload principle, where adaptation and supercompensation occur during an adequate recovery period in response to an exercise stress that has altered the body's homeostatic environment (Fry et al., 1991). Proper periodization of training should be designed to allow for sufficient time spent overloading and resting in order to achieve optimal performance. It is the stress of the overloading session that stimulates the adaptation and supercompensation processes that occur during rest. When done properly, the athlete gains the ability to train harder during subsequent training sessions, resulting in improved physical performance. This adaptive response is the basis for the positive component of overtraining.

Negative overtraining is associated with short or long-term performance decrements depending on the athletes' status on the continuum. Negative overtraining consists of over-reaching and the overtraining syndrome (Kentta & Hassmen, 1998).

Over-reaching occurs when several overloading sessions are performed without enough recovery periods. This will result in performance decrements that requires a few days longer than normal of recovery before any supercompensation is observed (Gleeson, 2002). If an athlete does not recover from their training session within 72 hours, they are most likely in an over-reached state (Kentta & Hassmen, 1998). In some cases, over-reaching is deliberately used to promote larger adaptations and improvements. Therefore, proper scheduling of over-reaching can ultimately lead to positive

performance gains. However, if the athlete continues to train while over-reached, a chronic form of negative overtraining, called the overtraining syndrome, may develop (Halson et al., 2002; Pichot et al., 2002; Urhausen & Kindermann, 2002).

The overtraining syndrome is more severe than over-reaching and is characterized by sustained decreases in performance. Highly motivated, elite athletes are most susceptible to developing the overtraining syndrome because they are more likely to train harder, rather than rest, to combat any decreases in performance (Fry et al., 1991; Kentta & Hassmen, 1998). This type of thinking causes a vicious cycle resulting in further decreases in performance. Once an athlete develops the overtraining syndrome, the best way to regain the prior physical condition is rest, however, it requires a significantly longer period than the over-reached state and does not produce any performance improvements (Fry et al., 1991).

Developing markers that detect the early stages of negative overtraining is of valuable importance to coaches and athletes. However, the establishment of reliable, specific and sensitive markers has been difficult (Kuipers, 1998). The characteristics that define the different states of overtraining are difficult to differentiate from each other, which has made it difficult to determine if specific markers are related to normal training fatigue, over-reaching or the overtraining syndrome (Kentta & Hassmen, 1998). In addition, the symptoms associated with negative overtraining are numerous (Fry et al., 1991) and highly individual (Uusitalo et al., 2000).

Autonomic hypothesis of negative overtraining

Some symptoms of negative overtraining have led to the theory of an underlying autonomic imbalance (Lehmann et al, 1998). Both divisions of the autonomic system have been said to be effected when an athlete is negatively overtrained giving rise to the sympathetic and parasympathetic forms of overtraining (Achten & Jeukendrup, 2003; Aubert et al., 2003; Kuipers, 1998; Lehmann et al., 1998). The sympathetic type may reflect an autonomic imbalance caused by training that is too intense combined with elevated psycho-emotional stress (Lehmann et al., 1998) and occurs during the early stages in the development of the overtraining syndrome (Kuipers, 1998). The sympathetic type mainly affects athletes in speed and strength sports (Kuipers, 1998), and causes an increase in sympathetic tone characterized by increased nocturnal catecholamine excretion, increased plasma catecholamine levels, increased resting and exercising heart rate, and sleep disturbances.

The parasympathetic type is caused by large volumes of training, usually observed in endurance athletes, and is thought to occur in the later, more severe, stages of the overtraining syndrome (Kuipers, 1998). Characteristics suggestive of the parasympathetic type of negative overtraining consist of a decreased sympathetic tone with a greater parasympathetic predominance, which result in low resting and exercising heart rates, progressive anemia, and early fatigue. In each form of overtraining, performance is decreased and undesirably long recovery periods are required.

Autonomic markers of negative overtraining

Evidence of an autonomic imbalance can be expressed by alterations in catecholamine levels, heart rate variability and heart rate values. Heart rate and catecholamine levels are a reflection of the state of the autonomic system. However, heart rate is more easily attained than catecholamine levels, which makes it attractive to athletes.

Plasma and Urinary Catecholamines

Monitoring the resting plasma catecholamine levels over a 6-month training program has been shown to be a possible diagnostic tool in the detection of negative overtraining (Hooper, Mackinnon, Howard, Gordon, & Bachmann, 1995). In elite swimmers, resting plasma norepinephrine levels were increased during a period of intense training, decreased practice performance and increased ratings of fatigue (Hooper et al., 1995). Additionally, these athletes did not improve their performance during a major competition, following a 3- to 5-day taper period. This suggests that these athletes may have embarked on a state of sympathetic negative overtraining during their intense training period and did not allow for enough recovery time before their competition. Untrained individuals who cycled and ran (5-hours/day) for four consecutive days, showed increases in the morning plasma norepinephrine concentration on each day compared to baseline (Mischler et al., 2003). The peak value was attained after the second day of exercise and then decreased on the last two nights, which the authors indicated may be due to a blunted catecholamine response caused by volume expansion (Helyar, Green, Zappe, & Sutton, 1997). In contrast, no change in resting levels of

plasma norepinephrine or epinephrine occurred in elite canoeists who increased their training load by 50% for 6 consecutive days (Hedelin, Kentta, Wiklund, Bjerle, & Henriksson-Larsen, 2000). Therefore, it appears that the length of the training program and the athletes' level of fitness are determinants of levels of plasma catecholamines and whether a training stimulus will alter them or not. In terms of monitoring training status it would seem that a rise in exercising and resting levels of plasma catecholamines occurs in response to an exhaustive stress that has decreased the sensitivity of target organs (Lehmann, Baumgartl et al., 1992). With inadequate rest, this may lead to an exhaustion of the sympathetic function on the adrenomedullary system and ultimately to the parasympathetic type of negative overtraining (Lehmann et al., 1988; Lehmann, Schnee, Scheu, Stockhausen, & Bachl, 1992).

Following Olympic track and road cyclists during the 6-months prior to the 1988 Summer Games revealed that during the last month, when athletes are typically engaged in peak training, morning catecholamine excretion was significantly reduced (Lehmann, Baumgartl et al., 1992; Lehmann, Schnee et al., 1992). The norepinephrine levels decreased 41% and 35% in the track and road cyclists, respectively. The track cyclists did not utilize a recovery period prior to the Games and did not perform well. However, the road cyclists showed large increases above baseline levels of both norepinephrine and epinephrine during an appropriate recovery period. This team was successful and won two medals at the Games demonstrating that deliberate over-reaching can lead to a desirable adaptation after an adequate recovery period.

Middle and long-distance runners showed similar morning catecholamine responses after their training volume doubled over a 3-week period (Lehmann, Baumgartl

et al., 1992). Norepinephrine decreased 48% by the end of the training, while epinephrine decreased by 52%. Performance indices, measured as aerobic, anaerobic and 4mmol lactate thresholds, became stagnate after the training, while the total running distance during an incremental test decreased. A year later, many of the same participants underwent another 3-week training period, this time doubling the distance during the intense portion of training (i.e. speed-endurance, high-speed and interval training) (Lehmann, Baumgartl et al., 1992). This program was effective in improving performance and improvements were seen in running velocity at 4mmol of lactate and in total running distance during the incremental test. Moreover, the participants showed only a moderate decrease in morning catecholamine excretion.

Soccer players, who also showed decreasing performance at the 4mmol lactate threshold and total running distance also had an evident decrease in morning catecholamine excretion (Lehmann, Schnee et al., 1992). The authors concluded that decreases of basal catecholamine excretion depends on the training state of the athlete, not the sport or event that they are involved in (Lehmann, Schnee et al., 1992). Therefore, during periods of training that contain high volume levels, there seems to be a decrease in morning catecholamine excretion by approximately 50% or more that appears to occur during times of compromised performance. This may provide evidence of an exhausted intrinsic sympathetic activation leading to an imbalance of a greater parasympathetic predominance.

Heart Rate Variability

The analysis of HRV has been used to provide insight into the autonomic nervous control of the heart during and after exercise (Aubert et al., 2003). As a monitoring tool, HRV has been proposed as a marker in the determination of the early stages of negative overtraining (Pichot et al., 2002). However, the use of HRV analysis during and after a heavy training protocol has yielded conflicting results in terms of its value.

The different results may be due to the training load employed and the fitness levels of the participants. Performance capacity was decreased after 6-days of overload training in elite canoeists, suggesting that they were over-reached (Hedelin et al., 2000). Total training volume over the 6-days was 13 hours, which equaled a 50% increase for these athletes. Despite the performance decrement, HRV parameters measured during supine rest and after a 70° passive tilt test revealed no change. The length of the overloading period may not have been long enough to induce changes in the autonomic control in these trained participants during this study. Using a longer training program designed to negatively overtrain female endurance athletes, another study was able to demonstrate an increase in sympathetic activity (Uusitalo et al., 2000). The training ceased when the athlete met the pre-determined overtraining criteria or was physically and/or mentally exhausted, which took between six and nine weeks. Training increased from 6 hours per week to 12. Five of the nine participants reached the criteria and were likely close to developing the overtraining syndrome. Since the other participants did not exhibit the overtraining criteria, they may have only been over-reached. In either case, the LF component was increased during supine rest following the training, whereas the HF component was unchanged, suggesting an enhanced cardiac sympathetic activation.

Another study showed that when elite rowers increased their training load by 75%, parasympathetic modulation measured after three and six months increased (Iellamo et al., 2002). However, during the next three months training load increased by 100%, which produced a shift in the cardiac modulation towards the sympathetic system.

The studies above lean towards the idea that there is a sympathetic shift in the sympathovagal balance as training status progresses along the overtraining-response continuum. However, it is difficult to conclude that the more debilitating overtraining syndrome developed in the participants. A case study involving a junior national cross-country skier who displayed symptoms of the overtraining syndrome for several months suggest that there may be another shift in the sympathovagal balance back towards a parasympathetic predominance (Hedelin, Wiklund et al., 2000). The participant had an increased HF component during supine rest in the negatively overtrained state, which returned to lower values after a 2-month recovery period. In addition, this athlete showed a decreased LF component after a 70° tilt, a maneuver that stresses the sympathetic system. After a lengthy recovery period the HRV components were restored.

Heart rate variability during sleep has also been examined following an intensified training period. Three-weeks of progressively increasing training volume by 100% did not produce any changes in HRV indices during sleep in endurance trained males despite performance decreases (Bosquet et al., 2003). However, another study found an altered cardiac autonomic modulation in over-reached participants (Pichot et al., 2002). This study commenced with an initial 8-week intensive training program in which previously sedentary individuals showed a normal training response of improved fitness, increased HRV and a parasympathetic predominance. After a 4-week overload period

there were no further improvements in fitness or parasympathetic indices. However, there was a concurrent shift towards a sympathetic predominance.

Therefore, it appears that HRV measures may be altered during negative overtraining, which depends on the athletes' status on the overtraining-response continuum. It could be argued that the early stages of negative overtraining causes a shift towards a sympathetic predominance during rest and sleep as evidenced by an increased LF component. More severe stages of negative overtraining have shown that the LF component is decreased, suggesting that the sympathetic system may be exhausted and that the parasympathetic system predominates.

Heart Rate

Resting and exercising heart rates are influenced mainly by the autonomic nervous system and its effects on the adrenal medulla. Heart rate variability analysis indicates that the autonomic system may be altered during and following intensified training. Therefore, it could be presumed that the heart rate may also be altered when an individual is overtrained.

During exercise, the heart rate increases linearly with increasing workloads (Zavorsky, 2000). However, the submaximal and maximal heart rates have been shown to be decreased when an athlete is over-reached (Costill et al., 1988; Hedelin, Kentta et al., 2000) as well as engaged in the parasympathetic type of negative overtraining (Costill et al., 1988; Hedelin, Kentta et al., 2000; Lehmann, Baumgartl et al., 1992; Snyder, Kuipers, Cheng, Servais, & Fransen, 1995; Uusitalo et al., 2000). Under normal circumstances a lower submaximal response is viewed as an increased performance

capacity (Uusitalo et al., 2000). However, these studies reveal that performance is compromised suggesting that negative overtraining has occurred. Possible explanations for the decreased submaximal heart rates seen in these athletes may be a decreased sympathetic activation (Uusitalo et al., 2000), a decrease in β-adrenoreceptor density and/or sensitivity (Lehmann, Baumgartl et al., 1992) or hypervolemia leading to increased stroke volume (Hedelin, Kentta et al., 2000; Snyder et al. 1995). The decreased maximal heart rate values should be approached with some caution, as peripheral factors may be responsible for the termination of a maximal test before the true heart rate maximum is reached (Hedelin, Kentta et al., 2000).

Monitoring heart rates during resting conditions is common practice among athletes who are concerned with negative overtraining. Studies examining the effects of chronic exposure to high levels of intense training on morning resting heart rates have been conflicting. Multi-day races provide unique situations since maximal effort is repeated over consecutive days. It should be noted that races often involve elevated levels of mental stress, which may also affect the autonomic response. Nonetheless, valuable information of the physiological demands of repeated high intensity can be discovered. During a 3-week stage race, professional cyclists displayed a non-significant decreasing trend in morning resting heart rates measured on day 10 and day 17 of the race (Earnest et al., 2004). Marathon runners developed a significant decrease in the heart rates measured in the morning during the first 8 days of a 20-day 500-km road race (Dressendorfer et al., 1985). However, by day 20, heart rates increased 10 bpm from the value on day 8 (Dressendorfer et al., 1985). Interestingly, despite all being endurance trained marathon runner there was a large range of morning resting heart rates between

the lowest and highest value for each day (approximately 25 bpm). This may reflect a difference in fitness levels between participants and therefore the races may very well produce different responses among individuals.

Studies involving a training intervention, rather than actual races, also display the same heterogeneity in morning resting heart rate response to overtraining. No change in morning resting heart rate during and after a two-week high intensity interval training program in competitive cyclists has been reported (Jeukendrup et al., 1992), whereas slight decreases (Hedelin, Wiklund et al., 2000) or increases (Pelayo, Mujika, Sidney, & Chatard, 1996) have also been observed. The fact that no change was seen in the morning heart rate was interesting, seeing as an elevation in heart rate during sleep was present in these subjects (Jeukendrup et al., 1992). However, the mean sleep heart rate values also showed a large range across individuals (approximately 20 bpm). Nonetheless, the authors suggested that monitoring heart rates during sleep might provide a more sensitive marker for the determination of negative overtraining in athletes.

After effective training, sleep heart rate may be lower compared to pre-training levels (Iellamo et al., 2002; Pichot et al., 2002). During intensified training programs designed to negatively overtrain participants, sleep heart rates are shown to be elevated (Roussel & Buguet, 1982; Stray-Gundersen et al., 1986; Jeukendrup et al., 1992; Iellamo et al., 2002; Mischler et al., 2003) or unchanged (Bosquet et al., 2003; Callister et al, 1990; Pichot et al., 2002), which likely depends on the training stimulus.

Prolonged exercise (5 – 7 hours per day) at low to moderate exercise (35 – 65% VO_{2max}) during 4 to 6 consecutive days has shown to elevate sleep heart rates during the first night and remain significantly above baseline levels each exercise day (Mischler et

al., 2003; Roussel & Buguet, 1982). Performing 5 hours of moderate intensity exercise for 4-days, sleep heart rate was highest on night 2 and decreased slightly during the following 2 days (Mischler et al., 2003). Despite the elevated sleep heart rates the circadian rhythm was not altered in this measure (Mischler et al., 2003; Roussel & Buguet, 1982). Performance was not monitored in either study, thus it is difficult to know if any form of negative overtraining was achieved. Due to the length of the training sessions, these participants may have been in an over-reached state.

In slightly longer protocols, the sleeping heart rate response has been conflicting. The addition of 4 hard interval sessions to the normal training of recreational runners (VO_{2max} 4.9L/min) over a 2-week period caused the sleep heart rate to be increased by 6 bpm (Stray-Gundersen et al., 1986). However, six experienced runners (max aerobic speed 19.40 km/hr) were classified as being negatively overtrained after 3-weeks and did not show any significant changes in sleep heart rate (Bosquet et al., 2003). The training volume in this study consisted of increasing long, slow, continuous running by 33, 66, and 100% during weeks 1, 2, and 3, respectively.

Similar results after an overtraining period were found in sedentary participants (Pichot et al., 2002). Following an 8-week intensive training program sleep heart rate was lower than pre-training values. The next 4-weeks were designed to overload the participants, which resulted in a stagnation of the sleep heart rates. During 1-week of recovery sleep heart rate decreased significantly reflecting a positive training adaptation and, possibly, a state of over-reaching in these participants. In junior national rowers, increasing training volume to 75% over 6-months decreased sleep heart rate (Iellamo et al., 2002). However, when training volume was further increased to 100% over the next

3 months, sleep heart rate increased 5 bpm compared to baseline values and 11 bpm compared to the preceding value attained after the 6-months of training.

The response of the heart rate during rest suggests that over-reaching causes an increase in heart rate that is immediate and persistent. The persistence may lead to an exhaustion of the sympathetic system, as evidenced by the HRV analysis and catecholamine levels, which may produce decreases in heart rate. However, the response appears to be highly individual.

Rationale for the study

Athletes and coaches can benefit greatly from a reliable marker of negative overtraining. Extensive research has been conducted in the area of overtraining. However, despite all the efforts, the development of markers of overtraining that are reliable and easily attained has been challenging. This can be attributed to the high degree of variability in responses across individuals. Moreover, group research designs have been used almost exclusively in this area, which measure a physiological parameter before and after the implementation of an overtraining intervention. This type of research design poses some problems for athletes in an applied setting. First, realizing that an athlete is overtrained based on a change in a pre-determined marker, may provide information that is too late for the athlete and coach to utilize and make the necessary adjustments to training (i.e. the marker would only confirm that the athlete was overtrained). Second, the results say nothing about the response of the marker as the athlete progresses throughout the training intervention. Third, markers developed in a tightly controlled laboratory may not show similar responses during real life training

situations. Lastly, finding enough participants to make a definitive conclusion who will overtrain themselves is difficult to attain.

If a marker is used to detect over-reaching or the overtraining syndrome, understanding its' day-to-day fluctuations as they occur during the training process are important. This provides information of changes that may occur in the transition from acute fatigue to over-reaching and the overtraining syndrome. Therefore, a marker could predict the overtraining syndrome rather than merely verifying that it has occurred.

A variable that has been commonly monitored during training is the resting heart rate. However, due to the large inter-individual variation expressed in this value, studies examining the effects of intense training on resting heart rate have been inconclusive. Since outside influences are minimized during sleep, it has been suggested that the average heart rate during sleep may be the more appropriate marker (Jeukendrup et al, 1992). It has been shown that sleep heart rate may be increased after a day consisting of regular exercise; however, research on the effects of over-reaching on sleep heart rate is limited and inconsistent, especially using a single-subject research design.

The purpose of this study was to examine the sleep heart rate response and subjective feelings relating to performance and recovery as training load is dramatically increased in order to provide a predictor of over-reaching and/or the early phases of the overtraining syndrome.

Research question

What is the time course of the sleep heart rate during a nine day period of intensified training designed to over-reach elite endurance athletes in an applied conditioning setting?

METHOD

A Retrospective Analysis

Data from the Canadian Sport Centre Manitoba (CSCM) database was utilized for this study. The CSCM provides sport science & medicine support to high performance athletic training groups. As part of this support, regular monitoring and assessment of the athlete's response to training is conducted to enhance the coach/athletes ability to make informed decisions regarding the effectiveness of their training process. This study examined data collected during a five-week period from a group of triathletes training with the Manitoba National Triathlon Centre.

Ethical Approval

Prior to commencement of the study, ethical approval for the experimental protocol was granted by the Education/Nursing Research Ethics Board of the University of Manitoba (Appendix A).

Participants

Three male and two female trained triathletes (n=5) were recruited for this study. All participants were members of the Manitoba National Triathlon Centre and have had extensive experience with training and physiological testing. Physical characteristics are shown in Table 1. Prior to the analysis of the data, all participants gave informed consent to allow their data to be used (Appendix B).

Table 1

Physical characteristics of participants

Participant	Age (yr)	Mass (kg)	Height (cm)	Peak Watts	VO_{2max} (ml/kg/min)
Females					
P1	19	64.6	168.0	357	
P2	20	57.7	164.1	317	60.1
Males					
P3	21	72.4	189.2	425	75.6
P4	16	59.0	179.9	394	79.6
P5	18	76.3	188.1	453	71.0

Note. Peak watts attained from cycle ergometer test. VO_{2max} attained from treadmill test.

Research Design

As part of their normal training schedule, the participants underwent three 9-day cycles (phases) of training that were structured as normal, over-reach, and normal training cycles.

A single-subject reversal (withdrawal) ABA design was used to examine the effects of over-reaching on the dependent variables of the study. An ABA design follows

the structure of baseline (first phase A), intervention (phase B) and withdrawal of the intervention (second phase A). Since the participants trained together and followed the same program, each participant received the intervention at the same time.

Measures

Dependent variables

The dependent variables used were the mean sleep heart rate found over the whole, first three hours and last three hours of the sleep period. The sleep heart rate data was collected by a device called an Actiheart (Cambridge Neurotechnology Ltd, Papworth, UK). The data collected was downloaded with computer software (Actiheart 2.0) and then exported to a Microsoft excel spreadsheet (Microsoft ® Excel 2002) for analysis. The information downloaded and exported contained heart rate and activity levels as they occur in respect to time. The sleep period was determined by visual inspection of the data set from each night, which began 30 minutes after activity levels ceased and ended 30 minutes before the activity levels noticeably increased. These points were also used in the calculation of the mean sleep heart rates for the first and last three hours.

Independent variable

The independent variable was the daily training load expressed as a training impulse (TRIMP) score (Foster et al., 2001). Heart rate training zones were calculated in relation to the measured mode-specific maximal heart rates; expressed as percentages of the individuals' maximum heart rate for each mode of exercise. The TRIMP score was

determined from the time spent in each training zone. The time in each training zone was then multiplied by a factor and summated to yield a total TRIMP value for that training session. The TRIMP values attained from all the exercise performed during the day, excluding swimming, was added together to give a daily TRIMP value. Heart rate training zones and multiplying factors are shown in Table 2.

Table 2

Heart rate training zones and accompanying multiplier

Zone	$\%HR_{max}$	Multiplier
1	50-65	1
2	65-75	2
3	75-85	3
4	86-92	4
5	92-100	5

Additional Measures

Participants performed a maximal treadmill and cycle ergometer test 4 and 5 days, respectively, before the commencement of the baseline phase. These tests were used to determine the maximal heart rates for each mode of exercise as well as some physiological characteristics of the participants.

The participants participated in three pre-scheduled 3000 meter races during the entire study period. Times from 3000 meter races were used to determine performance.

Since the races were already scheduled into their training plan they did not interfere with the phases of the study. The first race was held 8 days before the baseline phase, the second race was on the third day of the baseline phase, and the third race was 3 days after the recovery phase.

A Recovery-Performance Survey (Appendix C) was also completed each morning over the course of the study. The survey uses a Likert-scale with values ranging from 0 (completely disagree) to 5 (completely agree) indicating how the participants felt about their recovery from their training.

Study Phases

Baseline

Training in this period consisted of moderate intensity where participants completed their normal scheduled training. Participants were instrumented with a Polar S610i heart rate monitor, which was used to record all training sessions. The recordings from the training sessions were used to calculate the amount of time spent in each of the heart rate training zones from which daily TRIMP values were calculated. Participants were also instrumented with an Actiheart (Cambridge Neurotechnology Ltd, Papworth, UK), which was used to record heart rate during all sleep periods. The recordings from the sleep periods were used to determine the mean sleep heart rates outlined in the dependent variables section.

Intervention

Training for the intervention phase took place at a training camp held in Tucson, Arizona. The training load was determined by the coach with the intention of increasing the daily TRIMP score substantially from the baseline period. Participants wore the Actiheart and Polar heart rate monitors during each sleep period and every training session, respectively.

Recovery

During the recovery phase, participants reduced their training load to less than that in the baseline period. Participants continued to wear the Actiheart and Polar heart rate monitors during the sleep and training periods.

Equipment

1. Actiheart (Cambridge Neurotechnology Ltd, Papworth, UK): The Actiheart is a device used to monitor heart rate and movement, which has been shown to be reliable and valid for measuring movement and heart rate in humans at rest (Brage, Brage, Franks, Ekelund, & Wareham, 2005). The unit contains a main component and a smaller component that are connected by a 100mm wire. Both components attach to two electrodes placed on the chest. The total weight of the unit is 8g. The main component also contains the accelerometer for detecting movement.
2. Polar S610i heart rate monitor (Polar Electro, Oy, Finland): The Polar S610i heart rate monitor consists of a transmitter worn around the chest and a receiver worn as a

wristwatch. This monitor can provide live feedback and/or record data at 5 second intervals for future review and/or analysis.

3. AEI Moxus Metabolic Cart: Respiratory oxygen and carbon dioxide analyzer used in the determination of VO2max during the maximal treadmill test.
4. Lode "Excalibur" cycle ergometer: An electro-magnetically braked cycle ergometer used during the maximal bike test.

Data Analysis

Data was analyzed according to the guidelines for visual inspection (Hrycaiko & Martin, 1996). Each sleep heart rate value (whole night, first and last 3 hours) was averaged over their respective time period to give one data point for each night. The average scores from both the recovery and performance sections of the Recovery-Performance Survey also provided one data point each for every day. Each data point was then presented graphically for scientific visual inspection. In addition, the TRIMP value was summated from all training completed during the day and averaged among the participants to provide a data point for each day. This was also presented graphically to provide a reference for the amount of training during the analysis of the dependent variables. The guidelines used for determining whether the independent variable was responsible for any observed changes were:

1. A stable baseline or a baseline trend in a direction opposite to that predicted for the effects of the treatment.
2. The greater the number of times that an effect is replicated both within and across participants.

3. The fewer the number of overlapping data points between baseline and treatment phases.
4. The sooner the effect occurs following the introduction of the treatment.
5. The larger the size of the effect in comparison to baseline.
6. The results are consistent with existing data and accepted theory.

The values for the sleep heart rate and recovery-performance survey were also averaged for each phase for each participant. These values were displayed in a table format and analyzed with the same single subject criteria for visual inspection.

Interobserver Reliability Assessment of the Dependent Variable

An interobserver reliability (IOR) assessment is used in single subject designs to be confident that the data on the dependent variable are reliable. These assessments are carried out by two trained observers, who independently record the dependent variable. Computation of the IOR assessment involves dividing the smaller total by the larger total and multiplying that number by 100 giving a percentage. IOR assessments above 80% are considered acceptable (Hrycaiko & Martin, 1996).

Since the dependent variable, sleep heart rate, in this study is an objective measure, which does not require subjective scoring; an IOR assessment was not used. Sleep heart rate was recorded via an Actiheart, which has been shown to provide reliable and valid values in humans at rest (Brage, et al., 2005).

Social Validity

Social validity questionnaires are used to determine the practical importance of a behavioral change from the perspective of the participants. Social validity was assessed through the use of a questionnaire (Appendix C), which was completed by the participants after the study period. Social validity questionnaires are designed to address three issues of practical importance (Hrycaiko & Martin, 1996). These are the issues as they pertain to this study:

1. To what extent is the importance of sleep heart rate monitoring for treatment programs for the participant or society?
2. Are the particular procedures used acceptable to the participant, especially when alternative procedures might be available to accomplish the same results?
3. Are the participants satisfied with the results obtained?

Procedural Reliability

A procedural reliability assessment is performed to ensure that the treatment was applied as intended and as described. Since the study was a retrospective analysis, the data had already been collected as part of the normal training process as set up by the coach. In other words, the procedure was pre-determined in the form of a training strategy implemented by the coach. Therefore, a procedural reliability assessment could not be performed for this study.

RESULTS

Maximal Tests

Maximal heart rates from the maximal cycle ergometer and treadmill tests are reported in Table 3. Maximal heart rate data was collected from all tests but one. Due to a technical error, the maximal treadmill test for P1 was terminated early. Consequently, maximal heart rate achieved during the bike test for this subject was used to determine the heart rate zones for the run training sessions. P2, P4 and P5, achieved similar maximal heart rates for each mode of exercise. P3 had a discrepancy of 12 bpm between the bike and run tests.

Table 3

Maximal heart rate values from maximal cycle ergometer and treadmill tests

	Cycle Ergometer Test	Treadmill Test
Participant	HR_{max} (bpm)	HR_{max} (bpm)
P1	195	T
P2	194	192
P3	172	184
P4	184	184
P5	198	195

Note. bpm = beats per minute, T = terminated early

Performance Measure – 3000 m race

Each participant competed in a 3000 m running race before and after the intervention phase (Table 4). Comparing Race 1 with Race 2 illustrates that most

participants (P4 did not participate in Race 2) had made an improvement in their 3000 m race performance before the intervention phase. Results from Race 3 demonstrate a performance decrease in comparison to the times from the previous two races.

Table 4

Performance results from 3000m races

	Race		
Partcipant	1	2	3
P1	11.35	11.13	11.39
P2	11.68	11.18	11.73
P3	DNF	9.21	DNF
P4	9.17	DNP	9.21
P5	9.22	9.24	DNF

Note. The values represent race times in minutes.
DNF = Did not finish, DNP = Did not participate

Training Load

Four of the five participants completed each phase of the training program. One participant (P5) developed an illness that affected the volume of training during the intervention phase. For this reason, subsequent analysis will be conducted on participants P1 – P4, and will be referred to as the experimental group, unless otherwise specified.

Group Average Results

Total training volume increased from an average of 10.98 hours during the nine day baseline phase to 36.48 hours in the intervention period (Figure 1). Total phase TRIMP value from baseline to intervention increased 266% from 1261.70 to 4623.02 (Figure 2). This equates to an average daily TRIMP value of 140.19 during baseline and 513.67 during intervention (Figure 3). The recovery phase produced the lowest training load with a total training volume of 9.77 hours (Figure 1), total phase TRIMP value of 996.02 (Figure 2), and an average daily TRIMP value of 110.67 (Figure 3).

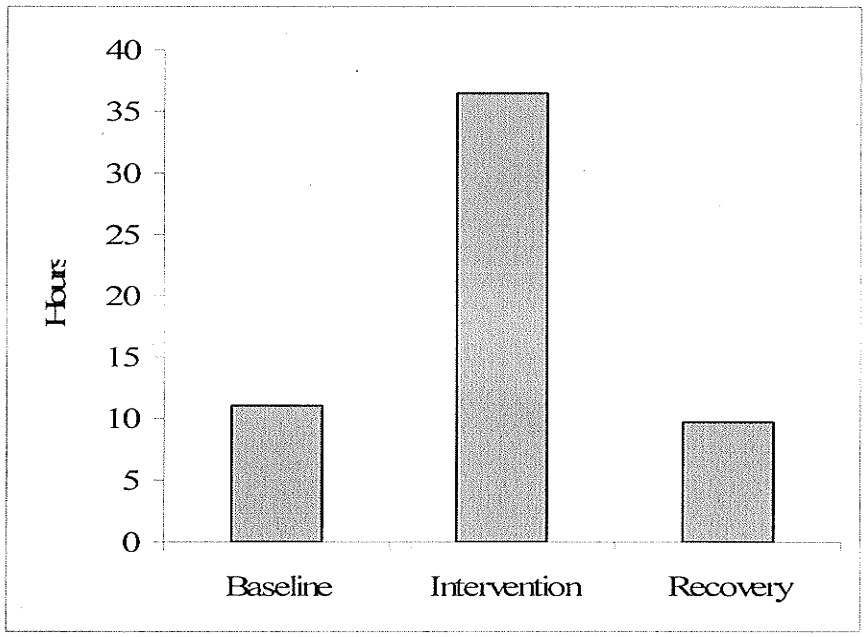

Figure 1. Total training time during each phase

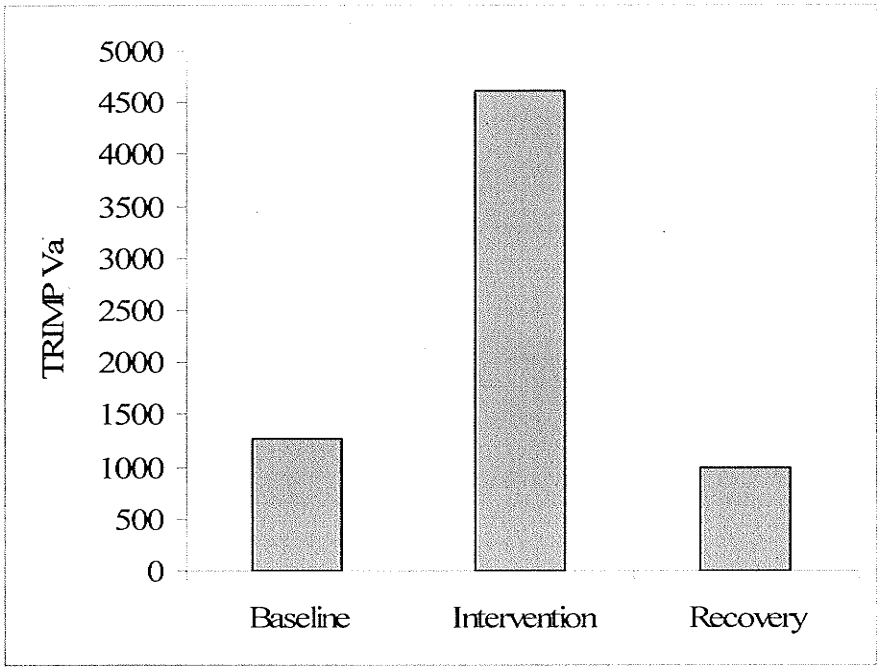

Figure 2. Total TRIMP value during each phase

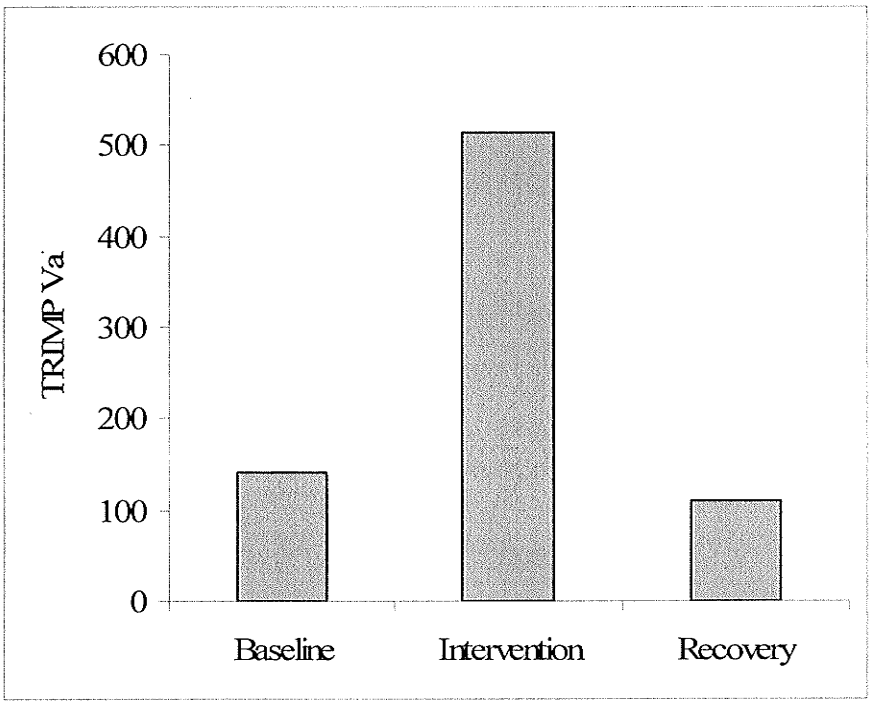

Figure 3. Daily average TRIMP value during each phase

The average total time training for each heart rate zone for each phase is shown in Table 5. The most training occurred in Zone 1 during the baseline and recovery phases, while the greatest percentage of time during the intervention phase was spent in Zone 2. Compared to the baseline phase, the intervention phase was characterized by substantial increases in total training time in Zones 1, 2, 3 and 4. The greatest relative increase in the intervention was in Zone 3, which increased 617.69 percent. A small decrease of 29.29% occurred in zone 5. During the recovery phase the time in zones 3, 4, and 5 decreased compared to the baseline phase. Meanwhile, time in Zones 1 and 2 were similar to baseline levels.

Table 5

Mean time accumulated in each training zone over each phase

Zone	Baseline (min)	Intervention (min)	Recovery (min)
1	311.63	659.08	309.78
2	197.54	815.54	199.17
3	76.13	546.38	43.13
4	40.50	144.34	11.67
5	32.92	23.28	22.37

Individual Participant Results

Total TRIMP and average daily TRIMP values per phase are shown in Table 6 and Table 7, respectively. Each participant increased their TRIMP values from the baseline phase to the intervention phase. The lowest increase was 214.62% (P2) and the highest was 333.22% (P3) compared to their baseline value. During the recovery phase, P2, P3, and P4 decreased their TRIMP value below their baseline values. P1 showed a similar value between baseline and recovery phases.

Table 6

Total TRIMP value for each phase

Participant	Baseline (min)	Intervention (min)	Recovery (min)
P1	975.90	4023.15	1004.02
P2	1436.49	4519.55	1069.30
P3	1319.01	5714.15	789.54
P4	1315.38	4235.24	1121.20

Table 7

Average daily TRIMP value for each phase

Participant	Baseline (min)	Intervention (min)	Recovery (min)
P1	108.43	447.02	111.56
P2	159.61	502.17	118.81
P3	146.56	634.91	87.73
P4	146.15	470.58	124.58

Percentages of change in TRIMP value for each training zone from the baseline phase to the intervention phase are shown in Table 8. The greatest percentage of increase occurred in Zone 3 for all participants each subject. The percent change during Zone 3 ranged from 439.38% (P2) to 892.25% (P3). Each participant also showed slight decreases in zone 5. This value ranged from 7.05% (P3) to 100% (P1).

Table 8

Percent TRIMP change from baseline to intervention phases

	Zone				
Participant	1	2	3	4	5
P1	164.67	307.46	634.25	348.23	-100.00
P2	55.37	279.66	439.38	75.96	-52.66
P3	139.17	363.27	892.25	714.34	-7.05
P4	110.74	301.35	647.91	67.49	-14.94

Sleep Heart Rate

Whole night mean sleep heart rates for each phase are shown in Table 9. A slight increase of approximately 2 bpm was shown across 3 participants (P2, P3, and P4). P1 showed no change in mean sleep heart rate during the intervention phase. During the recovery phase, all participants had lower mean sleep heart rates compared to the intervention phase. These values were also lower than the baseline values in 3 of the

subjects (P1, P2, P3), while one participant, P4, had a value similar to baseline. P2 had the greatest difference between the two phases, which was 6.5 bpm.

Table 9

Whole night mean sleep heart rate

Participant	Baseline	Intervention	Recovery
P1	49.6	49.5	45.8
P2	61.7	63.0	56.5
P3	50.1	52.7	49.5
P4	41.9	44.9	42.3

Note. The values represent heart rate in beats per minute.

During each phase, each participant had higher sleep heart rates during the first 3 hours of sleep compared to the last 3 hours (Table 10, Table 11). Similar findings to that for the whole night mean sleep heart rate were expressed during both the early and latter portions of sleep.

Table 10

Mean heart rate during the first 3 hours of sleep

Participant	Baseline	Intervention	Recovery
P1	50.6	50.1	46.3
P2	61.8	63.8	56.8
P3	52.2	55.2	50.1
P4	43.8	45.5	42.7

Note. The values represent heart rate in beats per minute.

Table 11

Mean heart rate during the last 3 hours of sleep

Participant	Baseline	Intervention	Recovery
P1	48.4	49.0	45.0
P2	61.6	63.2	56.5
P3	48.1	50.0	48.6
P4	41.9	43.9	41.9

Note. The values represent heart rate in beats per minute.

Based on the guidelines for visual inspection of the data, the mean sleep heart rate over the entire sleep period did not show any change from the baseline phase to the intervention phase for the experimental group (Figure 4).

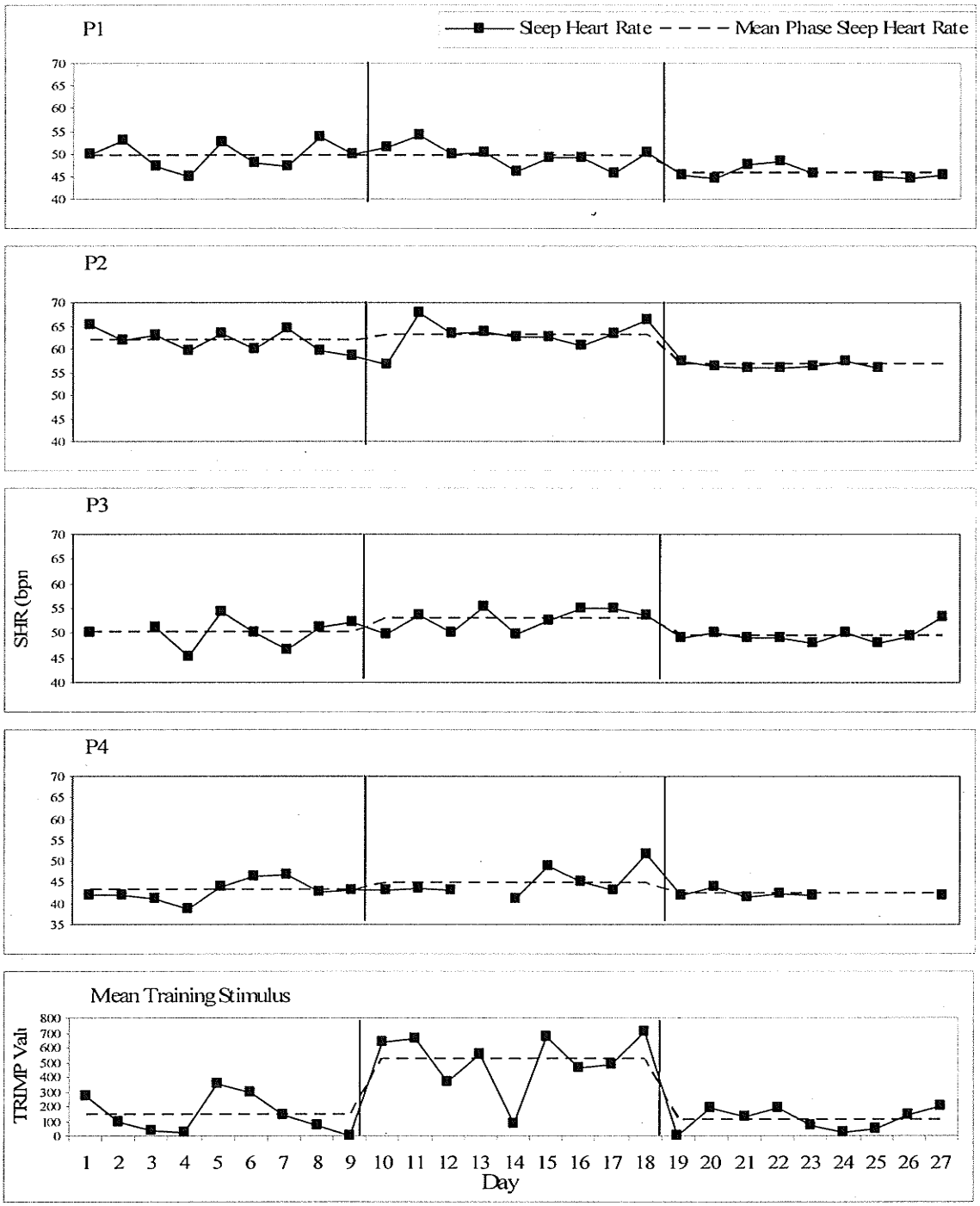

Figure 4. Time course of whole night sleep heart rate

There is a certain degree of fluctuation with this measure and appears to fluctuate fairly consistently with the training impulse. During the baseline phase concurrent decreases in TRIMP value and sleep heart rate occurred during day 1 to day 4 in each participant. The lowest sleeping heart rate in the baseline period also occurred on the night in which no exercise was performed (day 4). Returning to exercise on day 5, each of participants' sleep heart rate increased in comparison to day 4. Taking this day-to-day fluctuation into account, all participants displayed a stable sleep heart rate during the last few data points (i.e. nights) of the baseline phase.

During the intervention phase, there is no consistent trend in sleep heart rate values. This is reflected in the low number of times that any effect (increasing or decreasing) is replicated within and across participants. Comparing the individual data points during the intervention phase with the average sleep heart rate during the baseline period shows that an effect did not occur. P1 showed sleep heart rate values above and below its' baseline mean. P2 showed values mostly around this participants baseline mean, with only two data points higher and one lower. P3 showed values that were either at his baseline mean or slightly above. P4 only expressed any deviation from his baseline mean near the end of the intervention phase, and even then only two points were higher. These observations provide evidence of multiple overlapping points between the baseline and intervention phases demonstrated by each participant. The fluctuation in sleep heart rate during the intervention phase also appeared to vary with the changes in TRIMP values. However, despite much greater increases in TRIMP value it did not result in elevated sleep heart rates.

Immediately at the onset of the intervention phase, no participant showed any change in sleep heart rate. P2 showed a substantial increase on day 2 of the intervention phase compared to day 1 (56 bpm vs. 67 bpm). However sleep heart rate returned to baseline levels on the third night and did not increase again until the last night of the intervention period. All other participants showed similar sleep heart rates as baseline at the initiation of the intervention.

All participants sleep heart rate decreased during the first night of the recovery phase compared to their last night of the intervention phase. This response was immediate and long lasting. The sleep heart rates for P1 and P2 were lower than the baseline average value throughout the entire recovery phase. Meanwhile, participants P3 and P4 had sleep heart rate values that were on or just below their baseline average values. Only P2 had no overlapping data points between either of the two previous phases. All other participants had values that overlapped values in their baseline and intervention phases. However, all participants had an attenuated day-to-day fluctuation producing a more stable day-to-day sleep heart rate.

These observations give confidence that no effect on whole night sleep heart rate was observed as the participants transitioned from the baseline phase to the intervention phase. However, sleep heart rate did significantly decrease when the participants entered the recovery phase.

When sleep was confined to the first and last three hours, the average sleep heart rate illustrated similar observations as the whole night values when following the guidelines for any treatment effects (Figures 5 and 6).

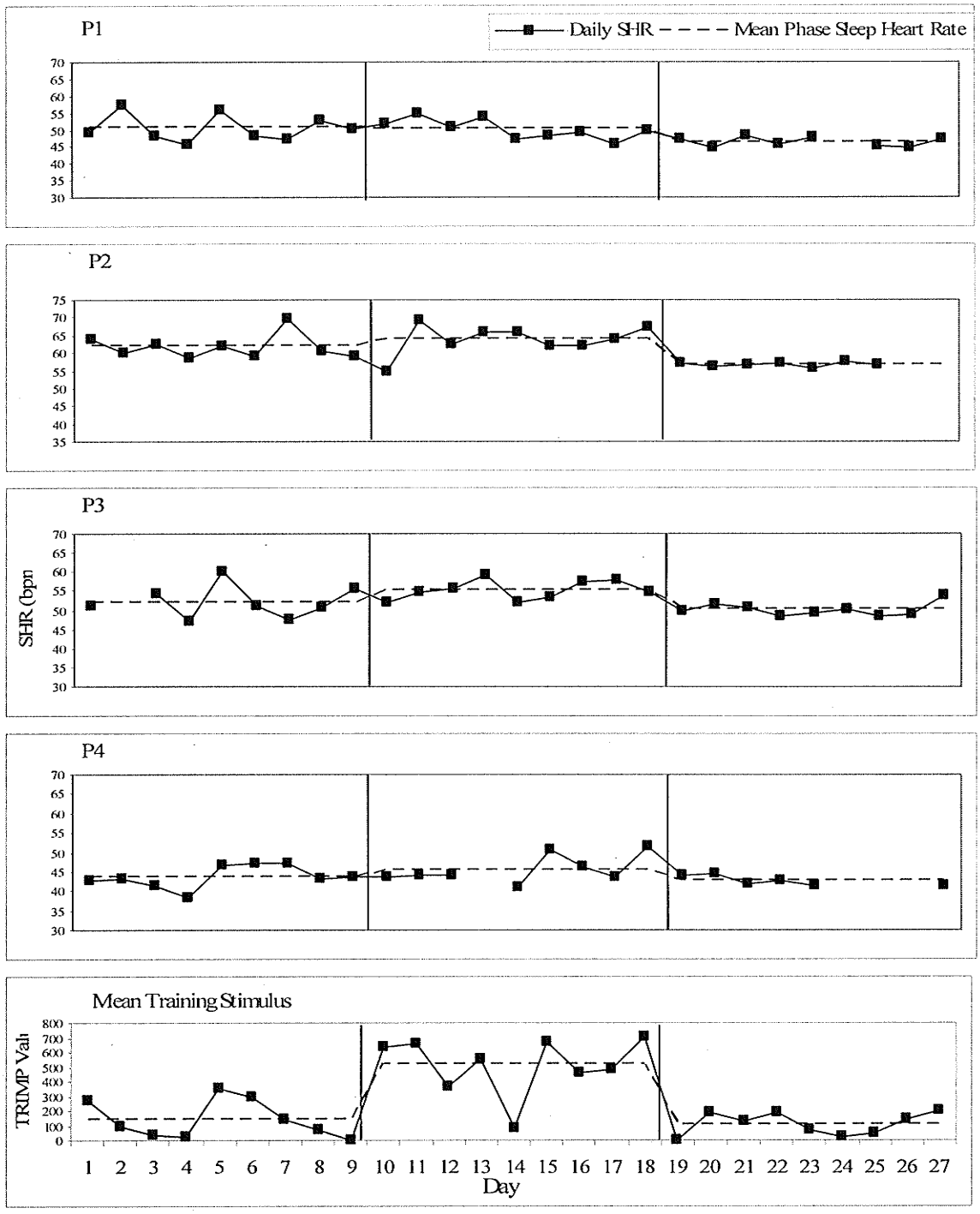

Figure 5. Time course of heart rate during the first 3 hours of sleep

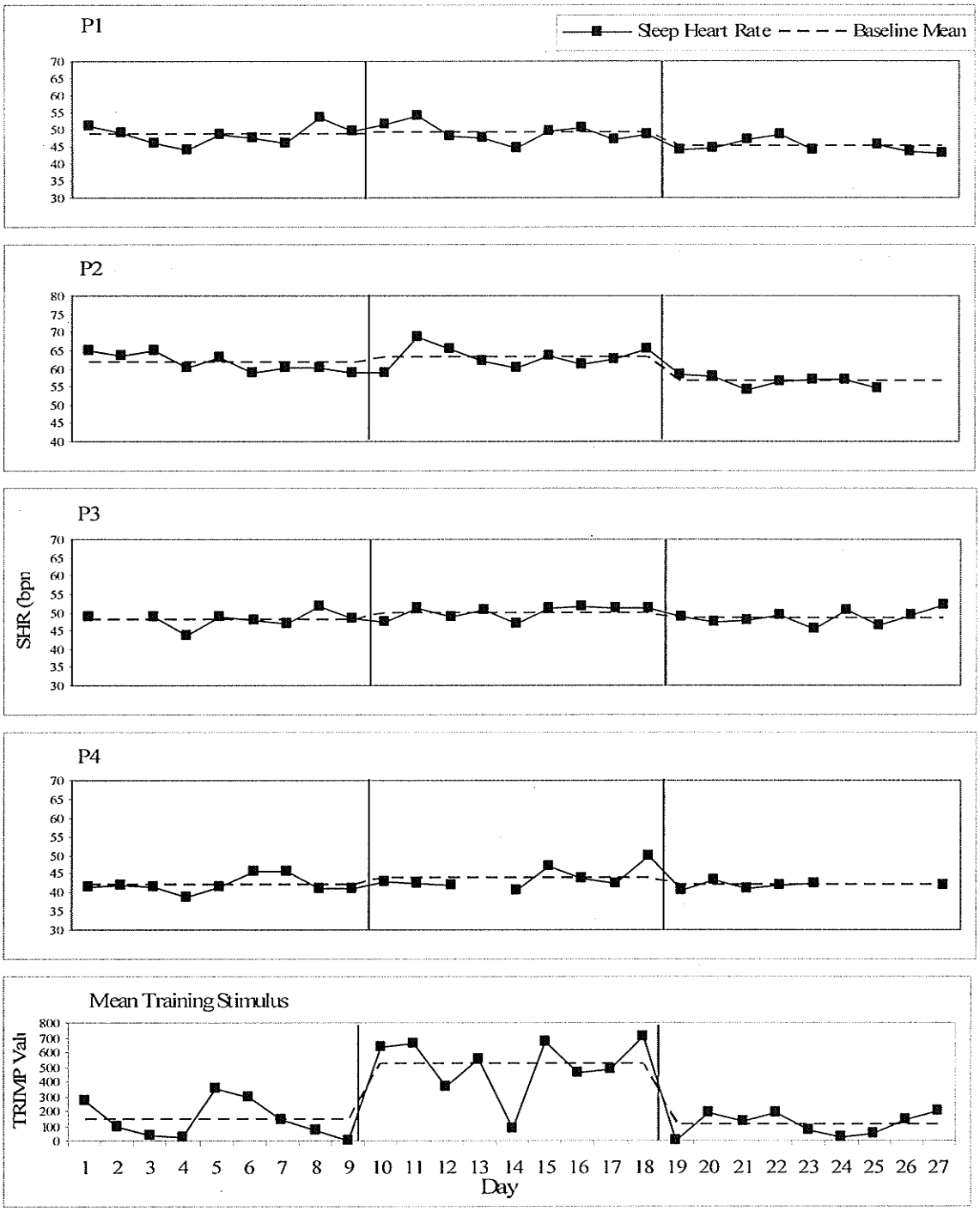

Figure 6. Time course of heart rate during the last 3 hours of sleep

The participant who developed an illness (P5) showed significant increases in sleep heart rate during the intervention phase (Figure 7). The baseline was stable until the last night. Sleep heart rate was increased on that night and steadily increased during the intervention until day 5 where it peaked. No exercise was performed on day 5 and the sleep heart rate progressively decreased during the remainder of the intervention period.

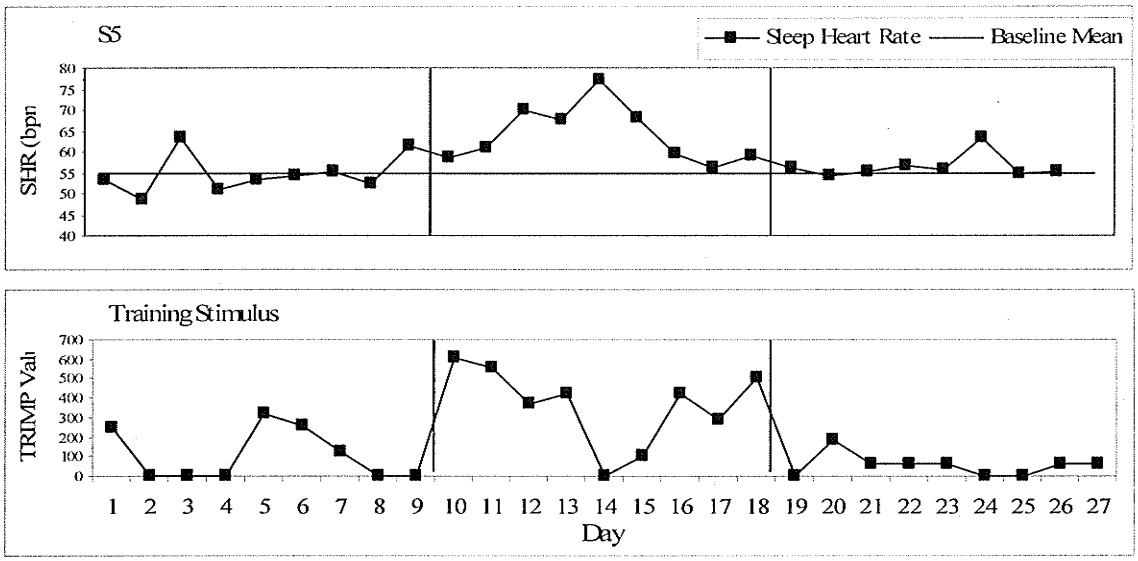

Figure 7. Whole night sleep heart rate and TRIMP value for P5

Table 12 reports the magnitude of the fluctuations that occurred in each period. During the baseline phase, each participant showed at least a 6.8 bpm range between the minimal and maximal sleeping heart rate values attained in the whole period. During the intervention phase, P2 and P4 increased their range at a magnitude of 4.5 and 2.9 bpm, respectively. P1 and P3 decreased their range at a magnitude of 0.4 and 3.5 bpm. During the recovery period, sleeping heart rates became more stabilized as each participant had lower ranges compared to both baseline and intervention phases. The

magnitude of decrease was 4.4, 4.9, 3.7, and 5.3 bpm for participants P1, P2, P3, and P4, respectively.

These results demonstrate that there is not a consistent change as the participants transitioned from the baseline phase to the intervention phase. However, there appears to be a decreasing change in the amount of fluctuation after the intervention phase as demonstrated throughout the baseline phase.

Table 12

Whole night maximal and minimal sleep heart rates observed during each phase

	Phase								
	Baseline			Intervention			Recovery		
Participant	Min	Max	Difference	Min	Max	Difference	Min	Max	Difference
P1	45.1	53.6	8.5	45.8	53.9	8.1	44.4	48.5	4.1
P2	58.6	65.4	6.8	56.6	67.9	11.3	55.8	57.5	1.7
P3	45.3	54.4	9.1	49.7	55.3	5.6	47.9	53.3	5.4
P4	38.8	46.6	7.8	40.9	51.6	10.7	41.6	44.1	2.5

Note. Values are represented as heart rate in beats per minute.

Minimal and maximal sleeping heart rate ranges during the first and last three hours demonstrated similar responses as the whole night sleep heart rate ranges (Tables 13 and 14). The participants who showed an increased range during the intervention phase were the same as those when referring to the whole night data. Likewise, the participants who had decreased ranges during the intervention were the same under all three time frames. In addition, all subjects demonstrated decreased ranges during the recovery phase compared to the baseline and intervention phases.

Table 13

First 3 hours maximal and minimal sleep heart rates observed during each phase

	Phase								
	Baseline			Intervention			Recovery		
Participant	Min	Max	Difference	Min	Max	Difference	Min	Max	Difference
P1	45.6	57.3	11.7	45.7	54.8	9.1	44.5	48.3	3.8
P2	58.8	69.5	10.7	55.0	69.1	14.1	55.8	57.6	1.8
P3	47.2	60.0	12.8	51.9	59.2	7.3	48.6	53.9	5.3
P4	38.5	47.3	8.8	40.8	51.3	10.5	41.2	44.6	3.4

Note. Values are represented as heart rate in beats per minute.

Table 14

Last 3 hours maximal and minimal sleep heart rates observed during each phase

	Phase								
	Baseline			Intervention			Recovery		
Participant	Min	Max	Difference	Min	Max	Difference	Min	Max	Difference
P1	44.1	53.7	9.6	44.5	53.9	9.4	43.1	48.5	5.4
P2	58.6	65.2	6.6	58.9	68.9	9.0	54.3	58.6	4.3
P3	43.4	51.6	8.2	47.0	51.5	4.5	45.5	51.9	6.4
P4	38.6	45.6	7.0	40.7	49.8	9.1	40.4	43.4	3.0

Note. Values are represented as heart rate in beats per minute.

Recovery-Performance Survey

Table 15 shows the average scores to the recovery questions and performance questions from the recovery-performance survey for each participant. P2 and P4 showed a decrease in feelings of recovery during the intervention phase. Scores for these participants rebounded back to baseline levels during the recovery phase. The other two participants (P1, P3) had similar scores from baseline to intervention. P3 had a lower score during the recovery phase compared to both the baseline and intervention phase. Feelings of performance showed a decrease in all participants during the recovery phase compared to the baseline and intervention phases. During the intervention phase, only P2 reported a decrease in performance scores.

Table 15

Mean recovery scores from recovery-performance survey

	Recovery		
Participant	Baseline	Intervention	Recovery
P1	17.33	17.67	17.50
P2	20.38	17.00	19.13
P3	21.63	20.63	18.00
P4	18.00	15.80	17.00

Table 16

Mean performance scores from recovery-performance survey

	Performance		
Participant	Baseline	Intervention	Recovery
P1	6.33	10.00	3.25
P2	9.25	7.00	6.63
P3	8.63	9.88	7.75
P4	8.00	9.60	7.60

Figure 8 shows the daily responses from each participant to the questions referring to their feelings of recovery. Due to a lack of data points from P1 in the baseline and intervention phases, it is difficult to make a confident conclusion. However, this participant was showing an increasing trend during the early portion of the intervention phase. At the beginning of the recovery phase, her feelings of recovery were quite low before increasing throughout the remainder of the phase. P2 showed high feelings of recovery during the early portion of the baseline phase before progressively decreasing at the end of the phase. This low level persisted throughout the intervention phase and increased back to baseline levels during the recovery phase. P3 showed a similar response during the baseline and intervention phases as P2. However, values never reached baseline levels during the recovery phase. There was also a lack of data points for P4, however it appears as if there was a decrease in feelings of recovery during the intervention phase and a rebound during the recovery phase.

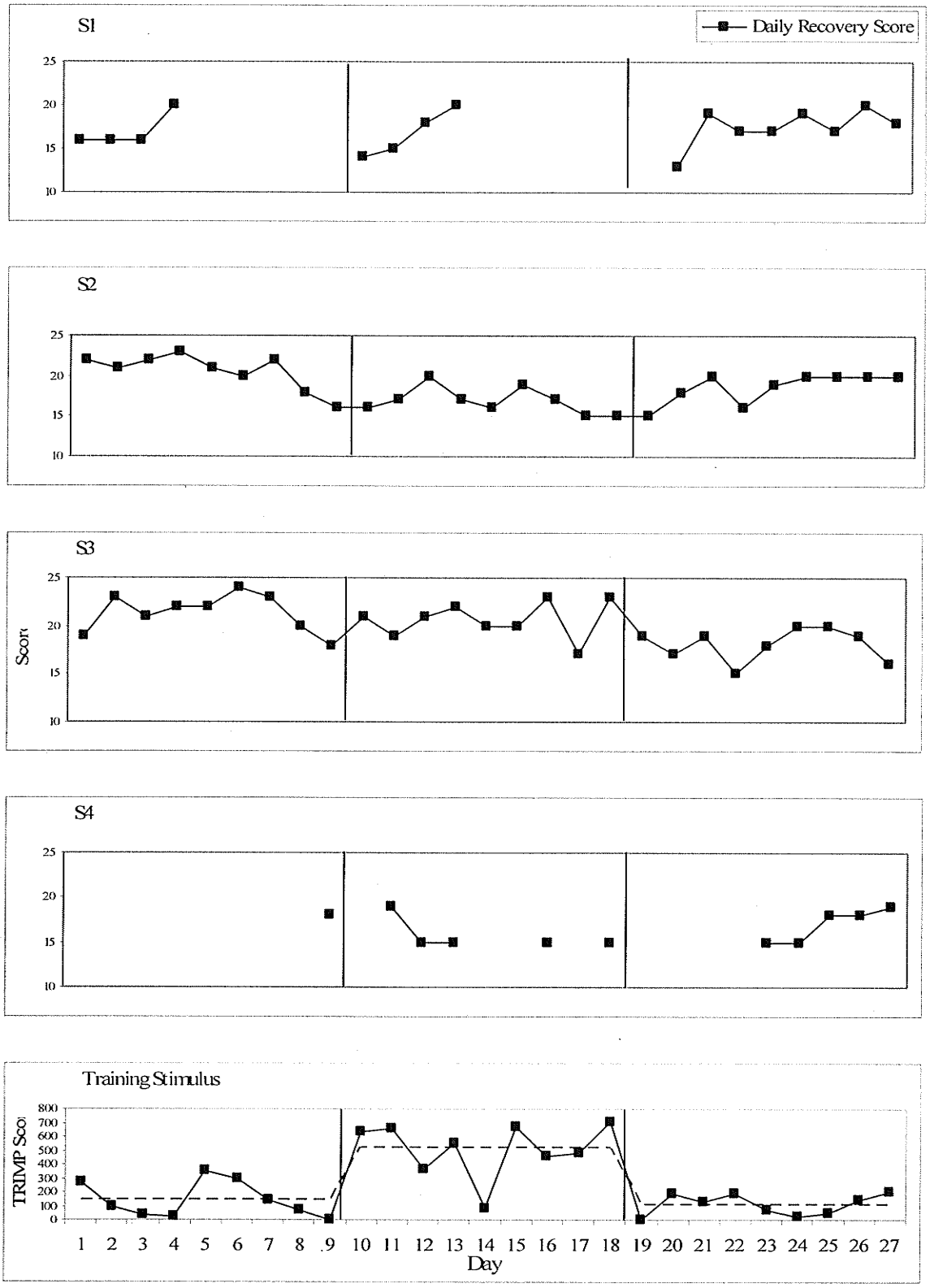

Figure 8. Time course of recovery scores from the recovery-performance survey

Figure 9 shows the daily responses to the questions referring to feelings of performance for each participant. Only P2 reported an increasing trend in their feelings of performance by the end of the study. P1, P3 and P4 all had progressively decreasing feelings of performance through the recovery phase. Interestingly, these subjects also had increased feelings about their performance during the intervention phase, while P2 had a progressive decrease.

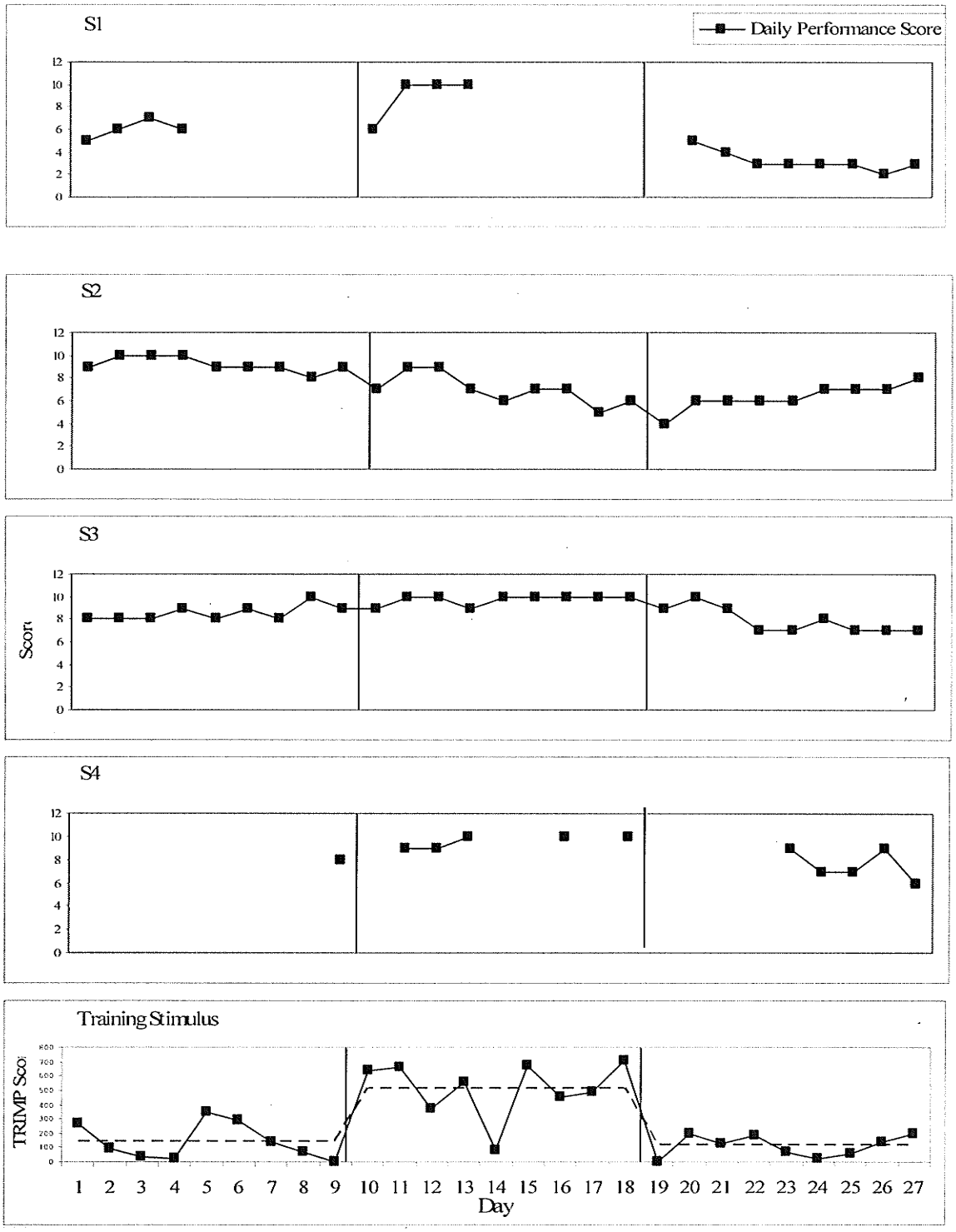

Figure 9. Time course of performance scores from the recovery-performance survey

Social Validity

Results from the social validation questionnaire are shown in Table 17. The general agreement from the group was a feeling that the monitoring of physiological components was important in training. Monitoring sleep heart rate did not require a great deal of energy in most participants and all felt comfortable utilizing this practice if the results proved beneficial. The subjects liked the idea of being able to compare their own results in order to understand their recovery and how their training was affecting their sleep. Both female participants expressed some discomfort with the monitoring device being used at night.

Table 17

Responses to the social validity questionnaire

Social Validity Questionnaire Component	P1	P2	P3	P4
Does monitoring physiological responses provide important information when determining if you are overtraining?	3	4	4	
Did you feel that the monitoring sleep heart rate on a daily basis required a lot of time and energy?	4	3	1	
If any changes were observed in your sleep heart rate during this study, would you be comfortable using this practice on your own to supplement your training?	4	4	5	
Do you feel that monitoring sleep heart rate would benefit other athletes?	4	4	5	

Note. 1 = Completely Disagree, 2 = Somewhat Disagree, 3 = Not Really Sure, 4 = Somewhat Agree, 5 = Completely Agree

DISCUSSION

The overtraining syndrome occurs in elite athletes who develop an imbalance between stress (physical, psychological and/or emotional) and recovery. Athletes use the training principle of overload in order to stress certain physiological characteristics important for enhancing performance in their particular sport/event. Recovery from the stress is only achieved during the interval between overloading sessions. When the body is not given enough time to recover from the training before another overloading session begins than an imbalance occurs. This process carried out over multiple overloading sessions creates a fatigue state that lies somewhere along the overtraining continuum with negative consequences. Detecting markers of the overtraining syndrome have been extensively studied (Bosquet et al., 2003; Callister et al., 1990; Dressendorfer et al., 1985; Lehmann, Baumgartl et al., 1992; Lehmann, Schnee et al., 1992; Pelayo et al., 1996; Snyder et al., 1995; Uusitalo et al., 2000). However, these markers are highly variable across individuals and if an athlete expresses any number of these markers it may be too late for any changes to be made to increase or even regain performance. Since overtraining exists on a continuum (Kentta & Hassmen, 1998), detecting markers that may predict the early stages of the overtraining syndrome, before it is too late, would be beneficial to elite athletes. A common characteristic that occurs along all points of the continuum is a decrease in performance. In this study, performance decreased in 3000 m race times after a nine day recovery period from a nine day overloading period. The length of the increased training load period and the decreased performance after a recovery period suggests that the participants were likely over-reached.

The primary objective of this study was to determine if sleep heart rate is a suitable marker in the detection of the early stages of negative overtraining. Sleep serves as a period of rest and restoration where the body attempts to maintain homeostasis. An acute response from exercise is an elevation in sympathetic activity and catecholamine levels during the recovery period until homeostasis is restored. Heart rate is directly influenced by these physiological responses to exercise and if great enough sleep heart rate may be elevated.

Training loads and sleep heart rates were monitored daily before, during, and after the over-reaching phase to determine if sleep heart rate is altered when an athlete is over-reaching. The time course for each of these variables were analyzed using a single subject research design, which may be a more appropriate methodology when studying physical conditioning in an applied setting (Kinugasa, Cerin, & Hooper, 2004).

Training Load

Athletic performance enhancement relies heavily on training load. Coaches and athletes often vary the amount of training load throughout their training plan along different periods of the season. However, an issue with this approach is being able to quantify the training load to achieve the desired effect. Training impulses (TRIMPS), which depends on heart rates and duration of training, are one way to quantify training load (Banister & Calvert, 1980). Training impulse units are arbitrary and may be determined by a variety of methods (Banister & Calvert 1980; Earnest et al., 2004; Foster, 1998; Foster et al., 2001). A common and easy method is determining heart rate zones based off of maximal heart rate and recording the amount of time spent in those

zones. Each zone is given a value, which is multiplied by the time spent in its' respective zone to give a single quantifiable unit to a training session (Foster et al., 2001).

In the present study, TRIMP values were calculated during a three phases of various training loads in a group of endurance trained triathletes. The phases examined were all part of the periodization plan that was developed by the coach and thus followed their normal training schedule. According to the periodization plan, the baseline, intervention and recovery phases occurred during a build period, a peak training period, and a recovery period, respectively. The TRIMP values demonstrated the large increase in training stimulus during the intervention phase compared to both baseline and recovery phases.

Participants completed 10.98 hours of training during the nine day baseline phase, which is slightly longer than the 7 hour per week moderate training regimen used during the baseline phase in an over-reaching study with endurance cyclists (Halson et al., 2002). With this group of participants, given their training history and fitness levels, recovery from this training load is assumed to be easily attained. The average daily TRIMP value during the baseline phase was 140.19 TRIMPs and was achieved primarily through training between 50% and 75% of maximal heart rate. Cycling at an intensity equivalent to 90% of an individuals' anaerobic threshold for 40 minutes would yield a similar TRIMP value (Foster et al., 2001; Halson et al., 2002).

During the intervention phase, total training time was increased to 36.48 hours, indicating that a large volume of training had taken place. The TRIMP value also increased 266% from the baseline phase to a daily value of 513.67. Cycling at 90% anaerobic threshold for approximately 2.5 hours would produce a TRIMP value similar to

this daily average (Foster et al., 2001; Halson et al., 2002). This amount of increase in training compared to the baseline phase was likely great enough to produce an over-reaching stimulus. Based from decreased performances in a maximal cycle ergometer test and a time trial along with an increase in global mood disturbance, seven days of intensified training have been determined to be long enough to over-reach trained cyclists (Halson et al., 2002). These subjects completed 14.55 hours of total training time for the seven days, which consisted of high intensity interval training. The greatest percentage of change occurred between 82% and 94% of maximal heart rate. In the present study, the largest increases in training during the intervention phase occurred in heart rate zones 3 and 4 zones, or between 75-92% of maximal heart rate. Therefore, comparing the two protocols indicates that in this study the athletes were performing more exercise at a slightly lower intensity. In addition, although the subjects in both studies were endurance trained, the athletes in the present study had higher VO_{2max} levels.

Although the greatest percent change occurred in zones 3 and 4, the highest volume of training was in zone 1 (50-65% HR_{max}), which allowed for a very large amount of training to occur. During the recovery period, training was drastically reduced and its' overall TRIMP value was 21% lower than the TRIMP value in the baseline phase. However, the daily average TRIMP score was only slightly lower than the baseline phase. Therefore, despite occurring in a build period and a recovery period, training in both periods were not great enough that recovery could not be fully attained.

Sleep Heart Rate

At the cessation of exercise a post exercise oxygen consumption period occurs where the body uses up energy in order to return its' functional systems back to their resting or baseline levels (Maehlum et al., 1986). The cardiovascular system plays an important role during this process and studies have shown that components of it's system may still be active hours after exercise has been completed (Carter, 2005; Dixon et al., 1992; Maehlum et al., 1986).

In this study, the average sleep heart rate for the intervention phase increased approximately 2 bpm compared to the baseline phase in 3 of the 4 subjects. The increase in these subjects was similar to endurance trained cyclists who had there sleep heart rates monitored after one week of intensified training (Jeukendrup et al., 1992). However, it was quite modest compared to two studies consisting of two weeks of overloading (Jeukendrup et al., 1992; Stray-Gundersen et al., 1986). These two studies revealed sleep heart rate increases of 5 and 6 bpm measured on a single night after the increased training in comparison to a single control night. However, these studies used longer protocols and the participants in the study by Stray-Gundersen (1986) were recreationally active.

During the recovery phase each subject decreased their sleep heart rate compared to the intervention phase. Additionally, the average sleep heart rate during the recovery phase was lower than the baseline phase in 3 of the 4 subjects. Cyclists and recreational runners did not decrease their sleep heart rates to the level of the participants in the present study and were still higher than their baseline values (Jeukendrup et al., 1992; Stray-Gundersen et al., 1986).

The difference between these results and that in this study may be due to the amount of increase in sleep heart rate during the intervention phase by the participants in the former studies. They showed a greater increase after the intervention, suggesting that the sympathetic system was engaged at a higher level, which persisted throughout the baseline phase. Secondly, the training protocol was different between the studies. In the present study, the training could be classified as high volume training, while the studies by Jeukendrup (1992) and Gundersen-Stray (1986) focused on adding more intense training sessions. Lastly, the sleep heart rate analysis in the other studies was done by comparing single nights before and after an intervention, the present study is using the average value over the entire phase. By taking a closer look at the day-to-day changes in the sleep heart rate it is possible to provide insight as to how this variable reacts during the process of over-reaching.

Following the participants on a day-to-day basis enabled the analysis of the effects of a single overloading bout as well as the cumulative effects of multiple overloading sessions (i.e. time course) on sleep heart rate to be made.

In this study, a single overloading bout produced inconsistent sleep heart rate responses among the participants. During the baseline phase, comparing Day 4 (18 TRIMPs) to Day 5 (352 TRIMPs), sleep heart rate was elevated in each of the 4 participants. The elevation in heart rate between these two days ranged from 6% to 20%, or 3.67 to 9.06 bpm, among the participants. However, although still increased, an attenuated sleep heart rate response was observed on the first day of the intervention phase despite almost doubling the training stimulus (641 TRIMPs). Each participant had a lower heart rate on this day compared to Day 5 of the baseline. Moreover, two

participants had decreased sleep heart rates (1.98 and 2.22 bpm), and two participants had increased sleep heart rates (1.82 and 0.11 bpm) compared to Day 9 of the baseline phase which also consisted of no training.

Previous studies have shown that sleep heart rate can be increased in some participants (Buguet et al., 1980; Mischler et al., 2003; O'Connor et al., 1993; Roussel & Buguet, 1982; Walker et al., 1978) and not changed in others (O'Connor et al., 1993; Walker et al., 1978). When comparing these studies, the participants who exhibited increased sleep heart rates also experienced larger training loads. These participants had exercised for either 8 hours at 35% VO_{2max} (Roussel & Buguet, 1982) or a combination of cycling, 2.5 hours at 57% VO2max and running, 2.5 hours at 65% VO_{2max} (Mischler et al., 2003). After the first night, sleep heart rate increased approximately 10.2% and 13.5%, respectively, compared to a control night. The participants who did not change their sleep heart rate exercised for either 35.5 minutes covering 10.2 km (Walker et al., 1978) or 30 minutes of cycling at 75% VO_{2max} (O'Connor et al., 1993).

According to these results, it would be expected that during the baseline phase, sleep heart rate might not be as responsive to the increased training load seen following day 5 as they would be after the first day of the intervention phase. Indeed, on both days that consisted of greater training stimulus, sleep heart rate was elevated and may be explained by an elevated sympathetic tone. However, the day with the greatest training stimulus did not produce the highest sleep heart rates, which may be due to an exercise induced hypervolemia observed following the day of exercise leading to a greater stroke volume (Fellmann, 1992). In addition, fluid ingestion may contribute to the increased plasma volume following the exercise (Bartholomew, O'Brien, & Gill, 2005), although

this was not measured in this study. Dehydration has been shown to increase heart rate during the recovery period from exercise (Carter, 2005), therefore, it is quite possible that hydration levels were at acceptable levels. Lastly, sleep heart rate shows a normal day-to-day variation (Waldeck & Lambert, 2003) of approximately 8 bpm, therefore, the participants may have fully recovered and are just displaying the normal variation.

At the opposite end of the dose-response relationship, the lowest sleep heart rate levels did occur on the days that consisted of the least amount of training. During the baseline phase the lowest sleep heart rate for the entire phase occurred on day 4, which consisted of no exercise. This response was evident during the intervention phase, where the day with the lowest TRIMP value also produced the lowest sleep heart rates over the duration of its phase. Therefore, it appears as if the acute effects of an exercise bout can increase sleep heart rate, although the magnitude of the increase is largely variable. This variation is reflected in the minimum and maximum sleep heart rate values within each phase. The baseline and intervention phase showed ranges of approximately 8 and 9 bpm, while the recovery phase decreased heart rate considerably to approximately 3.5 bpm. This illustrates that heart rate values from day-to-day were more stable in the recovery phase.

Graphing the day-to-day sleep heart rate facilitated the visual analysis of the effects of multiple overloading sessions on this variable. This method of analysis demonstrated that sleep heart rate was not further elevated in any participant compared to their baseline phase. The initial sleep heart rate response for P1, P2 and P3 was a slight increase during the first two days of the intervention phase compared to their last few data points of the baseline phase (i.e. acute effect). However, over the course of the

intervention phase, the sleep heart rate contained multiple overlapping data points with those in the baseline phase. Even though the fluctuations of the sleep heart rate appeared to be somewhat influenced by the TRIMP value in the baseline and intervention phases, the magnitude of the sleep heart rate values were unchanged. In other words, without knowledge of the training load, one would expect that the training stimulus was similar between the two phases.

Interestingly, during the recovery phase, sleep heart rate was decreased immediately compared to the intervention and baseline phases for each participant. This response is more impressive when we see how stable the sleep heart rate was during this phase. We have established that the training stimulus encountered during the baseline and recovery phases were such that recovery could be accomplished in this group of participants. Therefore, we can assume that the day-to-day variation of the sleep heart rates demonstrated during the baseline phase were normal. However, this variation was almost abolished during the recovery phase and the sleep heart rate values were decreased, suggesting sleep heart rate may be altered following an over-reaching period.

How can we explain observing an alteration during the recovery phase despite no change during the intervention phase? During the over-reaching phase we can assume that the sympathetic nervous system is performing at a higher level than normal, which has been shown by an increase in nocturnal nor-epinephrine (Mischler et al., 2003). However, this may not result in an elevated sleep heart rate but more of a complex neural integration, as seen in athletes during peak training (Buchheit et al., 2004; Furlan et al., 1993). The persistent increased sympathetic activation may decrease the sensitivity of target organs resulting in the parasympathetic form of overtraining. Therefore, the

increased training volume endured may have exhausted the sympathetic system, resulting in a greater parasympathetic predominance during rest resulting in a consistently decreased sleep heart rate. The fact that the sleep heart rate was consistently decreased suggests that the participants were still engaged in the parasympathetic form of overtraining during the third 3000 m race. Therefore, supercompensation had not been achieved and the athletes did not improve their performance.

This study has illustrated that the whole night average sleep heart rate is not altered during the intervention phase. Since sleep periods are relatively long, small changes that occur in heart rate throughout the night may be missed in the whole night average of this measure. Additionally, subjects with high levels of fitness may only need the early portion of the sleep period to complete the recovery from their daily activities. Due to this possibility, the sleep period was broken into three hour periods occurring at the beginning and end of sleep to determine whether heart rate activity during different parts of sleep was influenced by the increased training load. In other words, do the heart rate dynamics change during sleep following intensified training?

Sleep follows a cyclical pattern of NREM sleep stages interrupted by REM episodes. When sleep is broken into these cycles, average sleep heart rate decreases throughout the night. Therefore, the early portion of sleep contains a higher average sleeping heart rate than the latter portion. If recovery during the waking hours is incomplete then an elevated sleeping heart rate during the early portion of the sleep period may occur. The sleep heart rate in these subjects showed the typical response of higher values during the first 3 hours compared to the last 3 hours. The dynamics of the

heart rate during each portion of sleep displayed the same variability throughout each phase and therefore did not demonstrate any consistent change.

One participant (P5) was eliminated from the primary investigation when he could not complete the increased training regimen due to a respiratory illness. This subject showed a marked increase in sleeping heart rate, which presented itself just prior to the intervention phase. The increase in sleep heart rate occurred one day prior to the intervention phase and increased progressively throughout the first half of the intensified training, where he completed the same amount of training as the other subjects. Training volume was then reduced in this individual, which elicited an immediate and progressive decrease in sleep heart rate back to baseline levels by the end of the intervention phase. Therefore, this presents a clear example of how sleep heart rate monitoring may be effective and to adjust an athletes' training in order to promote recovery.

Psychometric assessment (Recovery-performance survey)

Subjective feelings about athletes mood states have been shown to be impaired during an overloading period (Urhausen, Gabriel, & Kindermann, 1998; Kellmann & Gunther, 2000; Kellmann, Altenburg, Lormes, & Steinacker, 2001). Early research utilized the Profile of Mood States (POMS) to assess the relationship between mood and training load. More recently, the Recovery-Stress Questionnaire for Athletes (RESTQ-Sport) was developed as a self-report used to measure the frequency of current stress along with the frequency of recovery-associated activities (Kellmann & Kallus, 2001). Both assessments have shown to exhibit a dose-response relationship as the training load increases (Kellmann & Gunther, 2000; Morgan, Brown, Raglin, O'Connor, & Ellickson,

1987). The difference between the two is the RESTQ-Sport addresses the subjective stress and recovery associated activities simultaneously (Kellmann & Gunther 2000). This difference allows the RESTQ-Sport to provide important information for the coach and/or athlete in terms of monitoring overtraining. An abbreviated version has been developed called the Recovery-cue, which also asks subjective questions pertaining to recovery and performance. The shortened version allows for the athlete to fill out the questionnaire regularly and quickly. In addition the coach is able to make adjustments to the training load quickly without having to go over a multitude of questions for each athlete.

During the intervention phase the responses to the questions pertaining to recovery were becoming less affirmative. This result was mostly due to their responses to the first three questions. The responses indicated that they felt as if a lot of effort was required to complete the training, that they were not completely recovered and that they were not successful in their rest and recovery activities. Interestingly, 3 of the 4 participants felt that they were more satisfied and relaxed before they went to sleep during the intervention phase compared to the baseline phase. In 3 of the 4 participants, there was progressive return to the baseline levels during the recovery phase.

The responses to the questions about their feelings about performance were similar among 3 of the 4 participants, while the other participant displayed the opposite. Three of the participants had an increasing score during the intervention phase before settling into a progressive decline during the recovery phase. For this section of the survey, there were only two questions; however, both were increased in the three participants that expressed an elevated perception of their performance. Interesting to

note, the subject who expressed a decreasing performance score also had the highest sleep heart rate and the greatest increase in sleep heart rate during the intervention phase.

Social Validity

The social validity questionnaire completed by the participants indicated that monitoring their sleep heart rates is important and could provide valuable information that they could use to enhance their training. The participants found that collecting such data was neither time nor energy consuming. In addition, all participants stated that they would use sleep heart rates to monitor their training if it was beneficial to them. The participants filled out the questionnaire before they were able to see the data, which is important as to not influence their answers. These results are important as they support one of the criteria of a marker of overtraining; that it is easily attained. If a marker is difficult to attain then it may provide another source of stress that is summated to the training stress. Therefore, despite the fact that sleep heart rate was not altered during the intervention phase compared to the baseline phase, the practical implications of this variable suggest that there may be a place for it in an applied setting.

Single Subject Design in Applied Conditioning Research

Group research designs are overwhelmingly more common in research pertaining to training in comparison to single-participant designs (Kinugasa et al., 2004). Group designs report a mean value of a sample from an experimental group, which is supposed to represent that group. This value is compared to a control group before and after an intervention to determine any effects. In applied conditioning research involving elite

athletes, the response of specific performance predictors shows great inter-individual variability, the focus should be on the individual athlete rather than a mean value representing a group (Kinugasa et al., 2004). Coaches and athletes also like receiving information about a particular variable along different points in time of an intervention to determine the value of a training strategy. This can be examined with the use of a single-subject research design.

Examining the time course of sleep heart rate during each phase of this study demonstrates the importance of using a single-subject design in applied conditioning research. This is especially true when the study involves elite athletes in an 'overtraining' environment. First, physiological responses to high loads of training are highly individual, meaning that not all athletes express the same response when they are over-reached or overtrained. Secondly, it is difficult to ask and get athletes to willingly overtrain. Finally, finding a sample population of subjects large enough to study using group statistical parameters and are actually over-reached or overtrained is also very difficult.

Previous studies have looked at the sleep heart rate before and after a period of increased training load. The day-to-day variation is sleep heart rate makes it difficult to come up with a reliable conclusion about the effects of over-reaching. When untrained women completed the same amount of exercise on separate days, sleep heart rate showed a range of 8 bpm (Waldeck & Lambert, 2003). Therefore, the possibility of producing the same sleep heart rate for two days consisting of equal exercise is highly improbable and any difference may be just a result of the day-to-day intrinsic variation. This is demonstrated by the participants in this study. The ability to conclude that exercise was

the cause of any changes in sleep heart rate would require a tightly controlled laboratory study. However, this is not the way athletes train and the results may not translate well into practice. When searching for markers of over-reaching, it is important to be able to be able to use the measure during a relatively free-living environment (Waldeck & Lambert, 2003).

Limitations and Future Research

The length of the study only gave a snap-shot of the entire training periodization plan. At the time of the baseline phase, these subjects were already in the midst of their third week in a build macrocycle. We have reported that the actual training stimulus during this phase was not great enough to restrict recovery. However, we do not know what type of training the participants were engaged in just prior to the study. Granted the intervention phase was substantially greater in terms of training stimulus, the sleep heart rates attained during the baseline phase could have already been altered by the previous build weeks. The recovery phase provided a more stable period and may have offered a better starting point for the study. Following the sleep heart rate from one recovery phase to another would possibly provide better framework from which data could be analyzed.

Another limitation with this study is the multitude of variables that may affect sleep heart rate. Indeed, one of the aims of the study was to observe this measure in a real life training environment, but other influences may have affected the results that can not be controlled for in an applied setting. Other influences include environment, sleep duration, sleep quality, psychological or emotional stressors and hydration to name a few.

A third limitation was the inability to add the swim training to the total daily TRIMP score. Due to technological difficulties, heart rate data was difficult to attain consistently and accurately. However, swim training was performed on most days of each phase and was performed at similar durations throughout. Therefore, any TRIMP score added to the baseline phase would likely be added during the intervention phase. Slight differences would arise if the subjects performed any workouts at greater intensities.

Future research should include a longer recovery phase to see if the normal day-to-day variation is restored. This would provide more confidence that the participants did develop the parasympathetic form of overtraining. It would also provide valuable information regarding when it would be appropriate to resume training after an over-reaching period. If the variation is restored, the timing may be highly individual and therefore should be conducted with a single-subject research design.

Since there are other factors that may influence sleep heart rate, an additional variable should also be monitored. Possible additional markers may include catecholamine levels, lactate levels, hydration levels and heart rate variability. These parameters may provide answers as to why the sleep heart rate did or did not change. This would allow for greater confidence that an autonomic imbalance exists in a particular athlete or if other avenues should be explored to explain the performance (i.e. glycogen depletion).

Conclusion

This study does not support the hypothesis that during over-reaching, sleep heart rate is altered in trained triathletes in an applied setting. However, monitoring sleep heart rate after over-reaching has developed may provide valuable information for athletes and coaches. The participants in this study may have developed the parasympathetic form of over-reaching that has been described in athletes that participant in endurance sports that employ high volumes of training (Kuipers, 1998).

REFERENCES

Achten, J., Jeukendrup, A. E. (2003). Heart rate monitoring: Applications and limitations. *Sports Medicine*, 33(7), 517-538.

Adam, K. (1980). Sleep as a restorative process and a theory to explain why. *Progess in Brain Research*, 53, 289-305.

Akselrod, S., Gordon, D., Madwed, J. B., Snidman, N. C., Shannon, D. C., & Cohen, R. J. (1985). Hemodynamic regulation: Investigation by spectral analysis. *American Journal of Physiology*, 249(4 Pt 2), H867-875.

Akselrod, S., Gordon, D, Ubel, F. A., Shannon, D. C., Berger, A. C., & Cohen, R. J. (1981). Power spectrum analysis of heart rate fluctuation: A quantitative probe of beat-to-beat cardiovascular control. *Science*, 213, 220-222.

Aldredge, J. L. & Welch, A. J. (1973). Variations of heart rate during sleep as a function of the sleep cycle. *Electroencephalography and Clinical Neurophysiology, 35(2)*,193-198.

Ansorge, E. J., Shah, S. H., Augustyniak, R. A., Rossi, N. F., Collins, H. L., & O'Leary, D. S. (2002). Muscle metaboreflex control of coronary blood flow. *American Journal of Physiology. Heart Circulatory Physiology, 283(2)*, H526-532.

Arai, Y., Saul, J. P., Albrecht, P., Hartley, L. H., Lilly, L. S., Cohen, R. J.et al. (1989). Modulation of cardiac autonomic activity during and immediately after exercise. *American Journal of Physiology, 256(1)*, H132-141.

Aubert, A. E., Seps, B., & Beckers, F. (2003). Heart rate variability in athletes. *Sports Medicine, 33(12)*, 889-919.

Banister, E. W. & Calvert, T. W. (1980). Planning for future performance: Implications for long term training. *Canadian Journal of Applied Sport Sciences, 5(3)*, 170-176.

Bartholomew, K., O'Brien, B. J., & Gill, N. D. (2005) Plasma volume expansion 24-hours post-exercise: Effect of doubling the volume of replacement fluid. *Journal of Sports Science and Medicine, 4*, 179-184.

Berne, R. M., Levy, M. N., Koeppen, B. M., & Stanton, B. A. (2004). *Physiology.* (5th ed.). St. Louis, Missouri: Mosby.

Boettcher, D. H., Zimpfer, M., & Vatner, S. F. (1982). Phylogenesis of the Bainbridge reflex. *American Journal of Physiology, 242(3)*, R244-246.

Bosquet, L., Papelier, Y., Leger, L., & Legros, P. (2003). Night heart rate variability during overtraining in male endurance athletes. *Journal of Sports Medicine and Physical Fitness, 43(4)*, 506-512.

Brage, S., Brage, N., Franks, P. W., Ekelund, U., & Wareham, N. J. (2005). Reliability and validity of the combined heart rate and movement sensor Actiheart. *European Journal of Clinical Nutrition, 59(4)*, 561-570.

Browman, C. P. (1980). Sleep following sustained exercise. *Psychophysiology, 17(6)*, 577-580.

Buchheit, M., Simon, C., Piquard, F., Ehrhart, J., & Brandenberger, G. (2004). Effect of increased training load on vagal-related indexes of heart rate variability: A novel sleep approach. *American Journal of Physiology. Heart and Circulatory Physiology, 287*, H2813-2818

Buguet, A., Roussel, B., Angus, R., Sabiston, B., & Radomski, M. (1980). Human sleep and adrenal individual reactions to exercise. *Electroencephalography and Clinical Neurophysiology, 49(5-6)*, 515-523.

Bunnell, D. E., Bevier, W. C., & Horvath, S. M. (1983). Nocturnal sleep, cardiovascular function, and adrenal activity following maximum-capacity exercise. *Electroencephalography and Clinical Neurophysiology, 56(2)*, 186-189.

Bunnell, D. E., Bevier, W. C., & Horvath, S. M. (1985). Effects of exhaustive submaximal exercise on cardiovascular function during sleep. *Journal of Applied Physiology, 58(6)*, 1909-1913.

Cajochen, C., Pischke, J., Aeschbach, D., & Borbely, A. A. (1994). Heart rate dynamics during human sleep. *Physiology & Behavior, 55(4)*, 769-774.

Callister, R., Callister, R. J., Fleck, S. J., & Dudley, G. A.(1990). Physiological and performance responses to overtraining in elite judo athletes. *Medicine and Science in Sports and Exercise, 22(6)*, 816-824.

Carter, J. B., Banister, E. W., & Blaber, A. P. (2003). Effect of endurance exercise on autonomic control of heart rate. *Sports Medicine, 33(1)*, 33-46.

Carter, R., Cheuvront, S. N., Wray, D. W., Kolka, M. A., Stephenson, L. A., & Sawka, M. N. (2005). Influence of hydration status on heart rate variability after exercise heat stress. *Journal of Thermal Biology, 30*, 495-502.

Casadei, B., Cochrane, S., Johnston, J., Conway, J., & Sleight, P. (1995). Pitfalls in the interpretation of spectral analysis of the heart rate variability during exercise in humans. *Acta Physiologica Scandinavica, 153(2)*, 125-131.

Catai, A. M., Chacon-Mikahil, M. P., Martinelli, F. S., Forti, V. A., Silva, E., Golfetti, R. et al. (2002). Effects of aerobic exercise training on heart rate variability during wakefulness and sleep and cardiorespiratory responses of young and middle-aged healthy men. *Brazilian Journal of Medical and Biological Research, 35(6)*, 741-752.

Convertino, V. A., Mack, G. W., & Nadel, E. R. (1991). Elevated central venous pressure: A consequence of exercise training-induced hypervolemia? *American Journal of Physiology, 260(2 Pt 2)*, R273-277.

Cornish, K. G., Barazanji, M. W., Yong, T., & Gilmore, J. P. (1989). Volume expansion attenuates baroreflex sensitivity in the conscious nonhuman primate. *American Journal of Physiology, 257(3 Pt 2)*, R595-598.

Costill, D. L., Flynn, M. G., Kirwan, J. P., Houmard, J. A., Mitchell, J. B., Thomas, R., et al. (1988). Effects of repeated days of intensified training on muscle glycogen and swimming performance. *Medicine and Science in Sports and Exercise, 20(3)*, 249-254.

Cousineau, D., Ferguson, R. J., de Champlain, J., Gauthier, P., Cote, P., & Bourassa, M. (1977). Catecholamines in coronary sinus during exercise in man before and after training. *Journal of Applied Physiology, 43(5)*, 801-806.

Dampney, R. A. (1994). Functional organization of central pathways regulating the cardiovascular system. *Physiological Reviews, 74(2)*, 323-364.

Degaute, J. P., van de Borne, P., Linkowski, P., & Van Cauter, E. (1991). Quantitative analysis of the 24-hour blood pressure and heart rate patterns in young men. *Hypertension, 18(2)*, 199-210.

Dela, F., Mikines, K. J., Von Linstow, M., & Galbo, H. (1992). Heart rate and plasma catecholamines during 24 h of everyday life in trained and untrained men. *Journal of Applied Physiology, 73(6)*, 2389-2395.

Dixon, E. M., Kamath, M. V., McCartney, N., & Fallen, E. L. (1992). Neural regulation of heart rate variability in endurance athletes and sedentary controls. *Cardiovascular Research, 26(7)*, 713-719.

Dressendorfer, R. H., Wade, C. E., & Scaff, J. H (1985). Increased morning heart rate in runners: A valid sign of overtraining. *The Physician and SportsMedicine, 13(80)*, 77-86.

Earnest, C. P., Jurca, R., Church, T. S., Chicharro, J. L., Hoyos, J., & Lucia, A. (2004). Relation between physical exertion and heart rate variability characteristics in professional cyclists during the Tour of Spain. *Brazilian Journal of Sports Medicine, 38(5)*, 568-575.

Eckberg, D. L. (1980). Parasympathetic cardiovascular control in human disease: A critical review of methods and results. *American Journal of Physiology, 239(5)*, H581-593.

Eckberg, D. L. (1983). Human sinus arrhythmia as an index of vagal cardiac outflow. *Journal of Applied Physiology, 54(4)*, 961-966.

Ekblom, B., Kilbom, A., & Soltysiak, J. (1973). Physical training, bradycardia, and autonomic nervous system. *Scandinavian Journal of Clinical & Laboratory Investigation, 32(3)*, 251-256.

Fellmann, N. (1992). Hormonal and plasma volume alterations following endurance exercise. A brief review. *Sports Medicine, 13(1)*, 37-49.

Foss, M. L. & Keteyian, S. J. (1998). *Fox's physiological basis for exercise and sport. (6th Ed.).* WCB/McGraw-Hill.

Foster, C. (1998). Monitoring training in athletes with reference to overtraining syndrome. *Medicine and Science in Sports and Exercise, 30(7)*, 1164-1168.

Foster, C., Florhaug, J. A., Franklin, J., Gottschall, L., Hrovatin, L. A., Parker, S., et al. (2001). A new approach to monitoring exercise training. *Journal of Strength and Conditioning Research, 15(1)*, 109-115.

Fry, R. W., Morton, A. R., & Keast, D. (1991). Overtraining in athletes. An update. *Sports Medicine, 12(1)*, 32-65.

Furlan, R., Piazza, S., Dell'Orto, S., Gentile, E., Cerutti, S., Pagani, M, et al. (1993). Early and late effects of exercise and athletic training on neural mechanisms controlling heart rate. *Cardiovascular Research, 27(3)*, 482-488.

George, C. F. & Kryger, M. H.(1985). Sleep and control of heart rate. *Clinics in Chest Medicine, 6(4)*, 595-601.

Gleeson, M. (2002). Biochemical and immunological markers of overtraining. *Journal of Sports Science and Medicine, 1*, 31-41.

Goldberger, J. J. (1999). Sympathovagal balance: How should we measure it? *American Journal of Physiology, 276(4 Pt 2)*, H1273-1280.

Goldsmith, R. L., Bigger, Jr., J. T., Steinman, R. C., & Fleiss, J. L. (1992). Comparison of 24-hour parasympathetic activity in endurance-trained and untrained young men. *Journal of American College of Cardiology, 20(3)*, 552-558.

Griffin, S. J. & Trinder, J. (1978). Physical fitness, exercise, and human sleep. *Psychophysiology, 15(5)*, 447-450.

Hague, J. F., Gilbert, S. S., Burgess, H. J., Ferguson, S. A., & Dawson, D. (2003). A sedentary day: Effects on subsequent sleep and body temperatures in trained athletes. *Physiology & Behavior, 78(2)*, 261-267.

Hakumaki, M. O. (1987). Seventy years of the Bainbridge reflex. *Acta Physiologica Scandinavica, 130(2)*, 177-185.

Halson, S. L., Bridge, M. W., Meeusen, R., Busschaert, B., Gleeson, M., Jones, D. A., et al. (2002). Time course of performance changes and fatigue markers during intensified training in trained cyclists. *Journal of Applied Physiology, 93(3)*, 947-956.

Hedelin, R., Kentta, G., Wiklund, U., Bjerle, P., & Henriksson-Larsen, K. (2000). Short-term overtraining: Effects on performance, circulatory responses, and heart rate variability. *Medicine and Science in Sports and Exercise, 32(8)*, 1480-1484.

Hedelin, R., Wiklund, U. Bjerle, P., & Henriksson-Larsen, K. (2000). Cardiac autonomic imbalance in an overtrained athlete. *Medicine and Science in Sports and Exercise, 32(9)*, 1531-1533.

Helyar, R., Green, H., Zappe, D., & Sutton, J. (1997). Blood metabolite and catecholamine responses to prolonged exercise following either acute plasma volume expansion or short-term training. *European Journal of Applied Physiology and Occupational Physiology, 75(3)*, 268-273.

Herd, J. A. (1991). Cardiovascular response to stress. *Physiological Reviews, 71(1)*, 305-330.

Hooper, S. L., Mackinnon, L. T., Howard, A., Gordon, R. D., & Bachmann, A. W. (1995). Markers for monitoring overtraining and recovery. *Medicine and Science in Sports and Exercise, 27(1)*, 106-112.

Horne, J. A. & Porter, J. M. (1975). Exercise and human sleep. *Nature, 256(5518)*, 573-575.

Hrycaiko, D. & Martin, G. (1996). Applied research studies with single-subject designs: Why so few? *Journal of Applied Sport Psychology, 8*, 183-199.

Iellamo, F., Legramante, J. M., Pigozzi, F., Spataro, A., Norbiato, G., Lucini, D., et al. (2002). Conversion from vagal to sympathetic predominance with strenuous training in high-performance world class athletes. *Circulation, 105(23)*, 2719-2724.

Jansen, A. S., Nguyen, X. V., Karpitskiy, V, Mettenleiter, T. C., & Loewy, A. D. (1995). Central command neurons of the sympathetic nervous system: Basis of the fight-or-flight response. *Science, 270(5236)*, 644-646.

Jeukendrup, A. E., Hesselink, M. K., Snyder, A. C., Kuipers, H., & Keizer, H. A. (1992). Physiological changes in male competitive cyclists after two weeks of intensified training. *International Journal of Sports Medicine, 13(7)*, 534-541.

Karvonen, J. & Vuorimaa, T. (1988). Heart rate and exercise intensity during sports activities. Practical application. *Sports Medicine, 5(5)*, 303-311.

Katona, P. G. & Jih, F. (1975). Respiratory sinus arrhythmia: Noninvasive measure of parasympathetic cardiac control. *Journal of Applied Physiology, 39(5)*, 801-805.

Katona, P. G., McLean, M., Dighton, D. H., & Guz, A. (1982). Sympathetic and parasympathetic cardiac control in athletes and nonathletes at rest. *Journal of Applied Physiology, 52(6)*, 1652-1657.

Katona, P. G., Poitras, J. W., Barnett, G. O., & Terry, B. S. (1970). Cardiac vagal efferent activity and heart period in the carotid sinus reflex. *American Journal of Physiology, 218(4)*, 1030-1037.

Kellmann, M., Altenburg, D., Lormes, W., & Steinacker, J. M. (2001). Assessing stress and recovery during preparation for the world championships in rowing. *The Sport Psychologist, 15*, 151-167.

Kellmann, M. & Gunther, K. D. (2000). Changes in stress and recovery in elite rowers during preparation for the Olympic Games. *Medicine and Science in Sports and Exercise, 32(3)*, 676-683.

Kellmann, M. & Kallus, K. W. (2001). *Recovery-Stress Questionnaire for Athletes - User Manual.* Champaign, IL: Human Kinetics Publishers.

Kentta, G. & Hassmen, P. (1998). Overtraining and recovery: A conceptual model. *Sports Medicine, 26(1)*, 1-16.

Khatri, I. M. & D. Freis, E. (1967). Hemodynamic changes during sleep. *Journal of Applied Physiology, 22(5)*, 867-873.

Kinugasa, T., Cerin, E., Hooper, S. (2004). Single-subject research designs and data analyses for assessing elite athletes' conditioning. *Sports Medicine, 34(15)*, 1035-1050.

Kollai, M. & Koizumi, K. (1989). Cardiac vagal and sympathetic nerve responses to baroreceptor stimulation in the dog. *Pflugers Arch, 413(4)*, 365-371.

Kuipers, H. (1998). Training and overtraining: An introduction. *Medicine and Science in Sports and Exercise, 30(7)*, 1137-1139.

Lehmann, M. (1988). Sympathetic activity in relation to performance diagnostics, training and overtraining. *International Journal of Sports Medicine, 9*, 390.

Lehmann, M., Baumgartl, P., Wiesenack, C., Seidel, A., Baumann, H., Fischer, S. et al. (1992). Training-overtraining: Influence of a defined increase in training volume vs training intensity on performance, catecholamines and some metabolic parameters in experienced middle- and long-distance runners. *European Journal of Applied Physiology and Occupational Physiology, 64(2)*, 169-177.

Lehmann, M., Foster, et al. C., Dickhuth, H. H., & Gastmann, U. (1998). Autonomic imbalance hypothesis and overtraining syndrome. *Medicine and Science in Sports and Exercise, 30(7)*, 1140-1145.

Lehmann, M., Schnee, W., Scheu, R., Stockhausen, W., & Bachl, N. (1992). Decreased nocturnal catecholamine excretion: Parameter for an overtraining syndrome in athletes? *International Journal of Sports Medicine, 13(3)*, 236-242.

Lewis, S. F., Nylander, E., Gad, P., & Areskog, N. H. (1980). Non-autonomic component in bradycardia of endurance trained men at rest and during exercise. *Acta Physiologica Scandinavica, 109(3)*, 297-305.

Loimaala, A., Huikuri, H., Oja, P., Pasanen, M., & Vuori, I. (2000). Controlled 5-mo aerobic training improves heart rate but not heart rate variability or baroreflex sensitivity. *Journal of Applied Physiology, 89(5)*, 1825-1829.

Maehlum, S., Grandmontagne, M., Newsholme, E. A., & Sejersted, O. M. (1986). Magnitude and duration of excess postexercise oxygen consumption in healthy young subjects. *Metabolism, 35(5)*, 425-429.

Miller, J. C. & Horvath, S. M. (1976). Cardiac output during human sleep. *Aviation, Space, and Environmental Medicine, 47(10)*, 1046-1051.

Mischler, I., Vermorel, M., Montaurier, C., Mounier, R., Pialoux, V. Pequignot, J. M. et al. (2003). Prolonged daytime exercise repeated over 4 days increases sleeping heart rate and metabolic rate. *Canadian Journal of Applied Sport Sciences, 28(4)*, 616-629.

Mitchell, J. H. (1985). Cardiovascular control during exercise: Central and reflex neural mechanisms. *American Journal of Cardiology, 55(10)*, 34D-41D.

Monti, A., Medigue, C., Nedelcoux, H., & Escourrou, P. (2002). Autonomic control of the cardiovascular system during sleep in normal subjects. *European Journal of Applied Physiology, 87(2)*, 174-181.

Morgan, W. P., Brown, D. R., Raglin, J. S., O'Connor, P. J., & Ellickson, K. A. (1987). Psychological monitoring of overtraining and staleness. *Brazilian Journal of Sports Medicine, 21(3)*, 107-114.

Mostoufi-Moab, S., Widmaier, E. J., Cornett, J. A., Gray, K., & Sinoway, L. I. (1998). Forearm training reduces the exercise pressor reflex during ischemic rhythmic handgrip. *Journal of Applied Physiology, 84(1)*, 277-283.

Murali, N. S., Svatikova, A., Somers, V. K. (2003). Cardiovascular physiology and sleep. *Frontiers in Bioscience, 8*, S636-652.

Nonidez, J. F. (1937). Identification of the receptor areas in the venae cavae and pulmonary veins which initiate reflex cardiac acceleration (Bainbridge's reflex). *American Journal of Anatomy, 61*, 203-231.

O'Connor, P. J., Crowley, M. A., Gardner, A. W., & Skinner, J. S. (1993). Influence of training on sleeping heart rate following daytime exercise. *European Journal of Applied Physiology and Occupational Physiology, 67(1)*, 39-42.

O'Connor, P. J. & Youngstedt, S. D. (1995). Influence of exercise on human sleep. *Exercise and Sport Sciences Review, 23*, 105-134.

O'Leary, D. S. (1993). Autonomic mechanisms of muscle metaboreflex control of heart rate. *Journal of Applied Physiology, 74(4)*, 1748-1754.

Pelayo, P., Mujika, I., Sidney, M., & Chatard, J. C. (1996). Blood lactate recovery measurements, training, and performance during a 23-week period of competitive swimming. *European Journal of Applied Physiology and Occupational Physiology, 74(1-2)*, 107-113.

Penzel, T., Kantelhardt, J. W., Lo, C. C, Voigt, K., & Vogelmeier, C. (2003). Dynamics of heart rate and sleep stages in normals and patients with sleep apnea. *Neuropsychopharmacology, 28*, S48-53.

Pequignot, J. M., Peyrin, L., Mayet, M. H., & Flandrois, R. (1979). Metabolic adrenergic changes during submaximal exercise and in the recovery period in man. *Journal of Applied Physiology, 47(4)*, 701-705.

Pichot, V., Busso, T., Roche, F., Garet, M., Costes, F., Duverney, D. et al. (2002). Autonomic adaptations to intensive and overload training periods: A laboratory study. *Medicine and Science in Sports and Exercise, 34(10)*, 1660-1666.

Pigozzi, F., Alabiso, A., Parisi, A., Di Salvo, V., Di Luigi, L., Spataro, A. et al. (2001). Effects of aerobic exercise training on 24 hr profile of heart rate variability in female athletes. *Journal of Sports Medicine and Physical Fitness, 41(1)*, 101-107.

Polman, R. Houlahan, K. (2004). A cumulative stress and training continuum model: A multidisciplinary approach to unexplained underperformance syndrome. *Research in Sports Medicine, 12*, 301-316.

Portier, H., Louisy, F., Laude, D., Berthelot, M., Guezennec, C. Y. (2001). Intense endurance training on heart rate and blood pressure variability in runners. *Medicine and Science in Sports and Exercise, 33(7)*, 1120-1125.

Rechtschaffen, A. & Kales, A. (1968). *A manual of standardized terminology, techniques and scoring system for sleep stages of human subjects*. Washington, DC: US Government Printing Office, National Institute of Health Publication.

Robinson, B. F., Epstein, S. E., Beiser, G. D., & Braunwald, E. (1966). Control of heart rate by the autonomic nervous system: Studies in man on the interrelation between baroreceptor mechanisms and exercise. *Circulation Research, 19(2)*, 400-411.

Roussel, B. & Buguet, A. (1982). Changes in human heart rate during sleep following daily physical exercise. *European Journal of Applied Physiology and Occupational Physiology, 49(3)*, 409-416.

Sagnol, M., Claustre, J., Cottet-Emard, J. M., Pequignot, J. M., Fellmann, N., Coudert, J. et al. (1990). Plasma free and sulphated catecholamines after ultra-long exercise and recovery. *European Journal of Applied Physiology and Occupational Physiology, 60(2)*, 91-97.

Sagnol, M., Claustre, J., Pequignot, J. M., Fellmann, N., Coudert, J. & Peyrin, L. (1989). Catecholamines and fuels after an ultralong run: Persistent changes after 24-h recovery. *International Journal of Sports Medicine, 10(3)*, 202-206.

Sanders, J. S., Mark, A. L., & Ferguson, D. W. (1989). Importance of aortic baroreflex in regulation of sympathetic responses during hypotension. Evidence from direct sympathetic nerve recordings in humans. *Circulation, 79(1)*, 83-92.

Sedgwick, A. W., Craig, R. J., Crouch, R., & Dowling, B. (1974). The effects of physical training on the day and night long-term heart-rates of middle-aged men. *European Journal of Applied Physiology and Occupational Physiology, 33(4)*, 307-314.

Shapiro, C. M., Bortz, V, Mitchell, D., Bartel, P., & Jooste, P. (1981). Slow-wave sleep: A recovery period after exercise. *Science, 214(4526)*, 1253-1254.

Shapiro, C. M., Griesel, R. D., Bartel, P. R., & Jooste, P. (1975). Sleep patterns after graded exercise. *Journal of Applied Physiology, 39(2)*, 187-190.

Smith, M. L., Graitzer, H. M., Hudson, D. L., & Raven, P. B. (1988). Effect of changes in cardiac autonomic balance on blood pressure regulation in man. *Journal of the Autonomic Nervous System, 22(2)*, 107-114.

Smith, M. L., Hudson, D. L., Graitzer, H. M., & Raven, P. B. (1988). Blood pressure regulation during cardiac autonomic blockade: Effect of fitness. *Journal of Applied Physiology, 65(4)*, 1789-1795.

Snyder, A. C., Kuipers, H., Cheng, B., Servais, R., & Fransen, E. (1995). Overtraining following intensified training with normal muscle glycogen. *Medicine and Science in Sports and Exercise, 27(7)*, 1063-1070.

Snyder, F., J. Hobson, A., Morrison, D. F., & Goldfrank, F. (1964). Changes in respiration, heart Rate, and systolic blood pressure in human sleep. *Journal of Applied Physiology, 19*, 417-422.

Somers, V. K., Conway, J., Johnston, J., & Sleight, P. (1991). Effects of endurance training on baroreflex sensitivity and blood pressure in borderline hypertension. *Lancet, 337(8754)*, 1363-1368.

Somers, V. K., Dyken, M. E., Mark, A. L., & Abboud, F. M. (1993). Sympathetic-nerve activity during sleep in normal subjects. *New England Journal of Medicine, 328(5)*, 303-307.

Stray-Gundersen, J., Videman, T., Snell, P. G. (1986). Changes in selected objective parameters during overtraining. *Medicine and Science in Sports and Exercise, 18*, S54-55.

Torsvall, L., Akerstedt, T., & Lindbeck, G. (1984). Effects on sleep stages and EEG power density of different degrees of exercise in fit subjects. *Electroencephalography and Clinical Neurophysiology, 57(4)*, 347-353.

Tulppo, M. P., Makikallio, T. H., Seppanen, T., Laukkanen, R. T., & Huikuri, H. V. (1998). Vagal modulation of heart rate during exercise: Effects of age and physical fitness. *American Journal of Physiology, 274(2 Pt 2)*, H424-429.

Ueno, L. M. & Moritani, T. (2003). Effects of long-term exercise training on cardiac autonomic nervous activities and baroreflex sensitivity. *European Journal of Applied Physiology, 89(2)*, 109-114.

Urhausen, A., H. Gabriel, H., & Kindermann, W. (1998). Ergometric and psychological findings during overtraining: A long-term follow-up study in endurance athletes. *International Journal of Sports Medicine, 19(2)*, 114-120.

Urhausen, A. & Kindermann, W. (2002). Diagnosis of overtraining: What tools do we have?. *Sports Medicine, 32(2)*, 95-102.

Uusitalo, A. L., Uusitalo, A. J., & Rusko, H. K. (2000). Heart rate and blood pressure variability during heavy training and overtraining in the female athlete. *International Journal of Sports Medicine, 21(1)*, 45-53.

Vatner, S. F., Boettcher, D. H., Heyndrickx, G. R., & McRitchie, R. J. (1975). Reduced baroreflex sensitivity with volume loading in conscious dogs. *Circulation Research, 37(2)*, 236-242.

Versace, F., Mozzato, M., De Min Tona, G., Cavallero, C, & Stegagno, L. (2003). Heart rate variability during sleep as a function of the sleep cycle. *Biolological Psychology, 63(2)*, 149-162.

Victor, R. G., Seals, D. R., & Mark, A. L. (1987). Differential control of heart rate and sympathetic nerve activity during dynamic exercise. Insight from intraneural recordings in humans. *Journal of Clinical Investigation, 79(2)*, 508-516.

Waldeck, M. R.& Lambert, M.I. (2003). Heart rate during sleep: Implications for monitoring training status. *Journal of Sports Science and Medicine, 2*, 133-138.

Walker, J. M., Floyd, T. C., Fein, G., Cavness, C., Lualhati, R. & Feinburg, I. (1978). Effects of exercise on sleep. *Journal of Applied Physiology, 44(6)*, 945-951.

Wilmore, J. H., Stanforth, P. R., Gagnon, J., Leon, A. S., Rao, D. C., Skinner, J. S., et al. (1996). Endurance exercise training has a minimal effect on resting heart rate:

The HERITAGE Study. *Medicine and Science in Sports and Exercise, 28(7)*, 829-835.

Yamamoto, K., Miyachi, M., Saitoh, T., Yoshioka, A, & Onodera, S. (2001). Effects of endurance training on resting and post-exercise cardiac autonomic control. *Medicine and Science in Sports and Exercise, 33(9)*, 1496-1502.

Zavorsky, G. S. (2000). Evidence and possible mechanisms of altered maximum heart rate with endurance training and tapering. *Sports Medicine, 29(1)*, 13-26.

Zemaityte, D., Varoneckas, G., & Sokolov, E. (1984). Heart rhythm control during sleep. *Psychophysiology, 21(3)*, 279-289.

APPENDICES

Appendix A: Approval Certificate

APPROVAL CERTIFICATE

19 July 2006

TO: **Lindsey Dahl** (Advisor G. Gannon)

Principal Investigator

FROM: **Stan Straw, Chair**

Education/Nursing Research Ethics Board (ENREB)

Re: **Protocol #E2006:066**

"Effects of Over-reaching on Sleep Heart Rate"

Please be advised that your above-referenced protocol has received human ethics approval by the **Education/Nursing Research Ethics Board**, which is organized and operates according to the Tri-Council Policy Statement. This approval is valid for one year only.

Any significant changes of the protocol and/or informed consent form should be reported to the Human Ethics Secretariat in advance of implementation of such changes.

Appendix B: Informed Consent

Informed Consent

Research Project Title: Effects of Over-Reaching on Sleep Heart Rate

Researcher: Greg Gannon/Lindsey Dahl

This consent form, a copy of which will be left with you for your records and reference, is only part of the process of informed consent. It should give you the basic idea of what the research is about and what your participation will involve. If you would like more detail about something mentioned here, or information not included here, you should feel free to ask. Please take the time to read this carefully and to understand any accompanying information.

1. The purpose of the proposed study is to determine the time course of sleep heart rate fluctuations during the transition from acute fatigue to over-reaching in relation to subjective feelings of stress in elite athletes. Specifically, the purpose of this assessment is to determine whether sleep heart rate provides a suitable marker for the detection of the early stages of overtraining.
2. In this study we would like to review and analyze the data you provided for the Canadian Sport Center Manitoba during a training camp held in February, 2006.
3. Your participation in this study is completely voluntary.
4. The time commitment will be approximately 15 minutes and is limited to the completion of a short questionnaire.
5. Any personal information and experimental data obtained will be treated as privileged and confidential.

Data obtained will be recorded in association with a subject number that only the principal researchers and yourself will know.

The information obtained during this research will be used for statistical analyses and scientific purposes only, with your rights to privacy maintained.

Upon study completion you will be informed by mail of the results specific to your participation as well as the general results of the study.

Your signature on this form indicates that you have understood to your satisfaction the information regarding participation in the research project and agree to participate as a subject. In no way does this waive your legal rights nor release the researchers, sponsors, or involved institutions from their legal and professional responsibilities. You are free to withdraw from the study at any time, and /or refrain from answering any questions you prefer to omit, without prejudice or consequence. Your continued participation should be as informed as your initial consent, so you should feel free to ask for clarification or new information throughout your participation.

Lindsey Dahl Telephone: 275-8261

Greg Gannon Telephone: 474-7649

This research has been approved by the *Education/Nursing Research Ethics Board*. If you have any concerns or complaints about this project you may contact any of the above-named persons or the Human Ethics Secretariat at 474-7122, or e-mail margaret_bowman@umanitoba.ca. A copy of this consent form has been given to you to keep for your records and reference.

Consent to Participate

I understand that a study is being conducted at the University of Manitoba as part of the requirements to complete a Master's degree, to analyze the effects of over-reaching on sleeping heart rate in attempt to find a suitable marker of the early stages of overtraining.

I have been asked to permit my data from a previous training camp to provide the data set required for analysis. I understand that there will be a short (15 minute) time commitment required.

I agree to participate in this study and understand that:

All personal information and data will be treated confidentially

I will not be identified personally when the results of the study are presented

I can stop participating any time that I choose without any penalty or questions asked

_____	_____	_____
Participant Name (please print)	Participant Signature	Date
_____	_____	_____
Parent/Guardian Name (please print) (If participant is under 18 years of age)	Parent/Guardian Signature	Date

Please specify your relationship with the participant _____

_____	_____
Researcher Signature	Date

Appendix C: Social Validity Questionnaire

Social Validity Questionnaire

Athlete:_____

Please answer the following questions concerning the study. For some questions please fill in the best answers to the question. For other questions please write in your answers.

1 = Completely disagree

2 = Somewhat disagree

3 = Not really sure

4 = Somewhat agree

5 = Completely agree

	1	2	3	4	5
1. Does monitoring physiological responses provide important information when determining whether you are overtraining?	O	O	O	O	O
Did you feel that the monitoring sleep heart rate on a daily basis was worth the time and energy expended?	O	O	O	O	O

3. Are you satisfied with the results obtained from monitoring sleep heart rate on a daily basis? O O O O O

4. Do you feel that monitoring sleep heart rate would benefit other athletes? O O O O O

5. Which aspects of monitoring sleep heart rate did you like?

_____

_____

_____

_____

Which aspects of monitoring sleep heart rate did you not like?

_____

_____

_____

_____

Appendix D: Recovery-Performance Survey

Recovery-Performance Survey

The information you provide is intended to help us assist you with your preparation and recovery pertaining to your training and competitive performances.

Please rate yourself after reading each of the following statements:

1 = Completely disagree

2 = Somewhat disagree

3 = Not really sure

4 = Somewhat agree

5 = Completely agree

Recovery	**1**	**2**	**3**	**4**	**5**
Very little effort was required to complete my training over the past few days.	O	O	O	O	O
I felt completely recovered prior to my training over the past few days.	O	O	O	O	O
I was very successful at my rest and recovery activities over the past few days.	O	O	O	O	O
I was satisfied and relaxed before sleep over the past few days.	O	O	O	O	O
I felt physically recovered over the past few days.	O	O	O	O	O
Performance					
I had lots of fun over the past few days.	O	O	O	O	O
I felt very confident over the past few days.	O	O	O	O	O

How can your coach or sport science staff help with your training and/or performances in the immediate future?