CHAPTER I

INTRODUCTION

The cardiovascular system delivers blood to all organs of the body. It takes part in the maintenance of homeostasis by constantly altering the pump output of the heart, consequently changing the diameter of resistance vessels. The pumping output of the heart is altered by a beat-by-beat adjustment in its pump rate and force (David and Lois, 1997).

While the heart inherently beats on its own, cardiac function can be influenced profoundly by neural inputs from both the sympathetic and parasympathetic divisions of the autonomic nervous system (ANS). These inputs allow us to modify cardiac pumping as appropriate to meet the changing homeostatic needs of the body. All portions of the heart rate are richly innervated by the adrenergic sympathetic fibers. In active stage, these sympathetic nerves release norepinephrine on cardiac cells. Norepinephrine interacts with beta-adrenergic receptors on cardiac muscle cell to increase the heart rate, the action potential conduction velocity, and the force of contraction. Overall, sympathetic activation acts to increase cardiac pumping (Malliani, 1982).

Cholinergic parasympathetic nerve fibers travel to the heart via the vagus nerve and innervate the sinoatrial node, the atrioventricular node, and atrial muscle. In the active stage, these parasympathetic nerves release acetylcholine on cardiac muscle cell. Acetylcholine interacts with muscarinic receptors on cardiac muscle cell to decrease the heart rate and action potential conduction velocity (DiFrancesco and Tromba,1988). Parasympathetic nerves may also act to decrease the force of contraction of atrial (not ventricular) muscle cell. Overall, parasympathetic activation acts to decrease cardiac pumping. An increase in parasympathetic nerve activity is usually accompanied by a decrease in sympathetic nerve activity (Schwartz et al., 1973).

It was extensively demonstrated that the beat to beat variability in R-R interval (or HR) depends on the neural control mechanisms elicited by sympathetic and/or parasympathetic nervous system and their interaction (Dixon et al., 1992). Therefore, in order to assess the state of ANS under the various physiological, psychological, and pathological conditions, the non-invasive power spectral analysis of spontaneous oscillatory fluctuation in the R-R interval (Akselrod et al., 1981) has recently been used to provide frequency specific oscillations whose amplitude reflect neural regulation of short-term cardiovascular adaptation under various conditions. That is, the power spectral of oscillations in the R-R interval has three well defined peaks: a very-low frequency (VLF) peak occurring typically between 0.003 and 0.04 Hz, a low frequency (LF) peak (ranging between 0.04 – 0.15 Hz) and high frequency (HF) peak (ranging between 0.15 and 0.40 Hz) (Akselrod et al.,1985). The VLF peak reflects the influence of circulating neurohormones, thermoregulatory, vasomotor tone and other slow variations in autonomic nerve activity (Akselrod et al., 1985). The LF fluctuations are mediated jointly by the parasympathetic and sympathetic activity, and HF fluctuations are modulated by the parasympathetic activity alone (Pomeranz et al., 1985).

Neurohumoral factors are considered important in governing the cardiovascular responses to exercise and training. Yet the exact mechanisms of their actions are still largely unexplored or are interpreted on the basis of indirect findings. The available evidence can be summarized as follows. The slow heart rate which is observed at rest after long term athletic training (Blomgrist and Saltin, 1983) is considered to reflect a combination of the reduced intrinsic heart rate (Lewis et al, 1980) and the enhanced vagal drive (Bedford and Tipton,1987). The latter has been inferred from the finding of raised levels of acetylcholine in the myocardium of trained rats as well as a greater gain of baroreceptive mechanisms in physically conditioned men and animals (Bedford and Tipton,1987). It is also possible that there is a reduction in adrenergic receptor density at SA node level or that down regulation occurs (Butler et al, 1982). Other

study suggested that the sympathetic activity at rest is reduced in the trained men (Smith et al, 1989).

It remains equivocal whether the enhanced vagal tone can contribute to the resting bradycardia in long-term physical trained athletes. Previous investigators have reported that cardiac vagal tone, which was assessed by either respiratory sinus arrhythmia or pharmacologic maneuvers, was enhanced at rest in long-term physical trained athletes, and the enhanced cardiac vagal tone might in part contribute to resting bradycardia in these athletes (Blomgvist and saltin, 1983;Kenney,1985). In contrast, other investigators have found that there was significant difference in cardiac vagal tone between athletes and nonatheless (Shin et al,1997). Several studies have demonstrated that HF power of RR interval spectra, which has been well known in representing the cardiac vagal efferent activity, showed the significantly higher value in athletes compared with non-athletes (Shin et al, 1997; Goldsmith et al,1997). This finding indicates that the parasympathetic tone enhanced with endurance exercise training may contribute in part to the resting bradycardia in the athletes.

Currently, there have been no studies conducted by comparing between the autonomic nervous system (ANS) controlling the heart rate of the endurance-trained athletes and the resistance-trained athletes, using auto-spectral analysis of RR interval variability.

Objectives

To study the result of the endurance-trained athletes and the resistance-trained athletes on the adjustment of ANS controlling the heart rate.

Research Question

Whether the endurance-trained athletes have more high frequency (HF), analyzed from the electrocardiogram (ECG), while taking the rest and exercising at the rate of 50 per cent of VO_2max than that of the resistance-trained athletes or not?

Hypothesis

The autonomic nervous system, controlling the heart rate, of the endurance-trained athletes while taking the rest and exercising at the rate of 50 percent of VO_2max is more active than that of the resistance-trained athletes.

Operational definitions

Heart rate variability (HRV) is defined as the amount of heart rate or RR interval fluctuations, coupling with normal average HR and may use as a mirror of the cardio respiratory control system.

Expected Benefit

1. To know the result of the endurance training and the resistance training in changing the functioning of ANS controlling the heart rate.

2. The result can be used as the instruction in the exercise training, which enhances the functioning of ANS controlling the heart rate.

3. The result is basic for the further study.