Cardiovascular Drift – Don Burris, PhD, CSCS

Cardiovascular drift occurs during prolonged steady state exercise and involves a progressive rise in heart rate and diminished stroke volume, cardiac output, and mean arterial pressure (Kounalakis et al., 2008). The increase in heart rate and decrease in stroke volume is caused by the catalyst of rising body temperature which invokes dehydration and a reduction in plasma volume. As plasma volume is depleted, venous return to the heart and stroke volume are also reduced (Powers & Howley, 2018). The increase in heart rate occurs to meet the demand and maintenance of level cardiac output as stroke volume decreases during prolonged steady state exercise. Cardiovascular drift has been found to be more prevalent and exaggerated in cycling than in running due to a greater magnitude of muscle involved and the occurrence of cardiovascular drift may negatively impact and impair performance and can be further affected by hyperthermia and blood pooling (Kounalakis et al., 2008).

During exercise, cardiac output increases to meet the demands of working muscles. Cardiac output is the volume of blood pumped by the heart in liters per minute and is a function of the amount of blood pumped out of the heart with each beat (stroke volume) and how fast the heart is pumping (heart rate) (Haff & Triplett, 2016). Exercise produces a reduction in vagal outflow to the heart in response to muscular metabolic demands and the process of autoregulation is triggered causing vasodilation, a reduction in the resistance within arterioles that supply blood to working muscles, and vasoconstriction, an increase in the resistance within arterioles that supply blood to inactive areas of the body (Powers & Howley, 2018). Relative to the magnitude of muscle mass recruited, sympathetic activation is stimulated by muscular contraction and controlled by the central command theory that describes rapid neurological intervention to achieve cardiovascular control during the initial stages of exercise. The central

command theory is defined by a motor signal that originates within the brain that sets the general pattern of cardiovascular response from the onset of exercise activity. Additionally, peripheral feedback from muscle mechanoreceptors, chemoreceptors, and arterial baroreceptors, provide afferent information back to the brain to modulate and fine-tune cardiovascular processes (Powers & Howley, 2018). The concepts of central command, autoregulation, and peripheral feedback are important to the regulation of cardiovascular function.

Cardiovascular athletes should be aware of physiological responses and process that might cause or affect cardiovascular drift in order to develop optimal training programs and mitigate negative impacts or potential impairments resulting from cardiovascular drift. The cardiovascular system is responsible for the effective delivery of oxygen and nutrients to working muscles and to remove metabolites and waste products (Haff & Triplett, 2016). Therefore, understanding that cardiovascular drift is a function of decreased stroke volume and increased heart rate in response to metabolic demand, rising body temperature, dehydration, and neuromuscular activation and control, a cardiovascular athlete can seek to create favorable training adaptations to mitigate the negative impacts of cardiovascular drift. During a protracted aerobically focused competition or event, an athlete's ability to instigate training adaptations that mitigate cardiac drift is important and valuable. Aerobic endurance training serves to enhance cardiac output, increase stroke volume, and reduce heart rate at rest and during submaximal exercise as well as serves to increase muscle fiber capillary density to improve oxygen delivery and the removal of carbon dioxide (Haff & Triplett, 2016). Additionally, aerobic endurance training results in the training adaptation of a slower discharge rate of the sinoatrial node due to an increase in parasympathetic tone resulting in improved maximal oxygen uptake. Relatedly, aerobic endurance training produces a chronic adaptation of increased stroke volume which

allows more blood to be pumped per contraction and, in turn, less frequent contractions (bradycardia) to satisfy the same cardiac output requirements (Haff & Triplett, 2016). Because cardiovascular drift is a function of reduced stroke volume and increased heart rate, optimizing maximal cardiac output (stroke volume x heart rate) serves to mitigate cardiovascular drift by enhancing stroke volume capacity and reducing, or slowing the increase of, heart rate. Moreover, the cardiovascular adaptations associated with increased muscle fiber capillary density enable more effective delivery of oxygen and nutrients as well as the removal of heat and metabolic by-products (Haff & Triplett, 2016). As previously noted, heat production associated with rising body temperature invokes dehydration and reduces plasma volume that serves to subsequently reduce venous return to the heart and stroke volume. Cardiovascular athletes seeking to mitigate the impacts of cardiovascular drift are benefited by achieving chronic aerobic exercise adaptions that enhance maximal cardiac output, stroke volume, reduce heart rate, and increase muscle fiber capillary density. In concert, these chronic adaptations serve to better maintain cardiac output requirements with less cardiovascular effort.

Understanding the factors that instigate cardiovascular drift and the physiological responses of cardiovascular drift, as enumerated herein, enable an athlete to train intelligently and specifically to strengthen cardiovascular systems and related physiological processes to enhance athletic performance. Ultimately, cardiovascular drift occurs to accommodate cardiac output requirements that are not being satisfied. An athlete's ability to effectively train to optimize cardiovascular function and adaptation is, in effect, improving their ability to better serve cardiac output requirements and mitigate cardiovascular drift.

References

Haff, G., & Triplett, N. T. (2016). *Essentials of strength training and conditioning.* Fourth edition. Champaign, IL: Human Kinetics.

Kounalakis, S. N., Nassis, G. P., Koskolou, M. D., & Geladas, N. D. (2008). The role of active muscle mass on exercise-induced cardiovascular drift. *Journal of sports science & medicine*, *7*(3), 395–401.

Powers, S. K., & Howley, E. T. (2018). *Exercise physiology: Theory and application to fitness and performance*.