PREVALENCE OF CARDIAC ABNORMALITIES IN MALE AND FEMALE COLLEGE ATHLETES WHEN EXPOSED TO PHYSIOLOGICAL AND THERMAL STRESSORS

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Manuscript

Prevalence of Cardiac Abnormalities in College Athletes When Exposed to Physiological and Thermal Stressors

Abstract

Cardiovascular pre-participation screening of collegiate student athletes is inadequate. Physiological stress in the form of submaximal exercise and heat exposure can both alter cardiovascular function, possibly elucidating an abnormality via electrocardiogram (ECG).

Purpose.

To investigate prevalence of cardiac abnormalities in college athletes when exposed to physiological and thermal stress.

Methods.

Eleven participants (males n=5; females n=6; 20.5 ± 1.4 yrs; 167.8 ± 4.8 cm; 60.0 ± 4.5 kg; 56.1 ± 12.2 ml/kg/min) currently participating at the NCAA Division I level volunteered for this study. Participants completed two submaximal treadmill tests (70% of VO_2 max) in varying environmental conditions: thermoneutral (24.8 ± 1.6˚C) and hyperthermic (38˚C) for 30 minutes. ECGs were recorded at five minute intervals. PR interval duration, ST segment elevation/depression, and R and S wave voltage amplitude were measured; ECGs were further analyzed for abnormalities. A repeated measures ANOVA was used to test the effects of condition by time.

Results.

No significant condition by time interactions were found for any variable (p>0.05). Significance across time manifested as a decreased PR interval (p<0.05), R wave voltage, (p<0.05), and S wave voltage (p<0.05). No main effects (p>0.05) were found for ECG abnormalities however; high occurrences of incomplete left bundle branch block (ILBBB) were found.

Conclusion.

Submaximal exercise in the hyperthermic condition did not significantly alter cardiovascular function in the parameters measured; however, the total number of ECG readings with abnormalities was higher in the hyperthermic condition compared to thermoneutral), most notable in incidences of ILBBB.

Abstract Word Count: 245

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Introduction

It is widely accepted that coronary heart disease is the leading cause of death in the US today (Cardiovascular Disease Statistics, n.d.). What is perhaps less well known is that the leading cause of death in young athletes is a phenomenon referred to as sudden cardiac death (SCD) (Maron *et al.*, 2009; cited in Drezner, Pluim, & Engebretsen, 2009). Incidence rates for SCD are relatively unknown due to the difficulty of comparing studies with profoundly different methodology and across vastly different geographic locations. A study conducted by Corrado *et al.*, (2006) across a significantly large population in the Veneto region of Italy, reported incidence figures for SCD in young athletes (aged 12-35 years) as high as 1:28,000. These

findings are similar to that of a recent study conducted at 11 US and Canadian cities that reported incidences of sudden cardiac arrest (SCA) from cardiovascular disease for children and adults (ages 14-24 years) at 1:27,000 (Atkins *et al.*, 2009). With regard to the type of cardiovascular disease responsible for SCD in young athletes, Maron *et al.* (2007) suggest that hypertrophic cardiomyopathy (HCM) is the leading cause. In addition, Papadakis, Whyte & Sharma (2008) adapted the work of Maron *et al.* (2007), stating that 14% of SCD in young athletes can be attributed to arrhythmias.

There is a substantial amount of evidence to suggest that competitive athletes may display abnormal electrocardiograms (ECG) as a result of their training regimes, which may alter both structural and/or electrical components of the myocardium (Corrado, Biffi, Basso, Pelliccia, & Thiene, 2009; Corrado *et al.*, 2010; Pelliccia *et al.*, 2000; Maron & Pelliccia, 2006; Drezner *et al.*, 2009; Langdeau *et al.*, 2001; Basavarajaiah *et al.*, 2008; Holly, Shaffrath, & Amsterdam, 1998; Wilson *et al.*, 2011). Changes that have been reported include sinus bradycardia, left and right ventricular remodeling (including increased wall thickness and chamber size), increased left atrial cavity size (and volume), increased RR, PR, and QT intervals, first-degree (atrioventricular) AV block and Mobitz type I (Wenkebach) second-degree AV block (Corrado *et al.*, 2009; Maron & Pelliccia, 2006; Langdeau *et al.*, 2001). Up to 80% of trained athletes can show some of these ECG changes, often referred to as “athlete’s heart syndrome”. These changes result from non life threatening physiological adaptations of the autonomic nervous system to athletic conditioning, including changes such as increased vagal tone and/or withdrawal of sympathetic activity (Holly *et al.*, 1998).

Rywik, O’Conner, Gittings, Wright, Khan, & Fleg (2002) have suggested that the ECG response to treadmill exercise plays a significant role in the ability to diagnose and assess

individuals with suspected coronary heart disease. The authors outlined research that have shown that a horizontal or down-sloping ST-segment depression ≥ 1 mm in response to exercise is a powerful predictor for future coronary events, including myocardial infarction and SCD, in an apparently healthy population (Rywik *et al.*, 2002). In addition, exposure to heat has also been shown to elicit abnormal cardiac activity both in sedentary exposure (Rautaharju, Wolf, Piironen, Äikäs, & Karvonen, 1966) and exposure combined with some magnitude of physical exertion (Akhtar, Al-Nozha, Al-Harthi, & Nouh, 1993). ECG changes reported during sedentary exposure included shortening of the S-T segment and a flattening of the T wave in lead X (Rautaharju *et al.*, 1966). Furthermore, common changes observed during physical exertion in the heat included increased Q-T interval, sinus tachycardia, and diffuse ST-T changes that were highly suggestive of myocardial ischemia (Akhtar *et al.*, 1993).

These data suggest that initial incidence rates may have significantly underestimated the magnitude of the problem of SCD in athletes and that an exercise stress test may be conducive to bringing about observable cardiac abnormalities in an otherwise healthy, previously undiagnosed individual. Based on these data, there is a need to provide a more clear perspective of this issue, helping to increase awareness and improve preventative measures for athletes. The work of Pfister, Puffer & Maron (2000) provided data that suggest current pre-participation cardiovascular screening processes for US collegiate student-athletes is an area of concern. It was reported that only 26% of institutions analyzed used screening forms that were considered adequate according to the 12 recommendations of the American Heart Association consensus panel for pre-participation cardiovascular screening of athletes (Maron *et al.*, 1996). Therefore, the purpose of this study was to elucidate possible previously undiagnosed cardiac abnormalities in an otherwise healthy, NCAA Division I athlete population through exposure to a given

workload in contrasting environmental conditions (thermoneutral and hyperthermic). We hypothesized that if cardiac abnormalities exist, then exercising in the heat would elicit them to a greater extent than in a thermoneutral environment.

Methods

Participants.

Eleven participants (male n=5; female n=6) volunteered to participate in the study). All participants were current NCAA Division I athletes and included members of the golf, cross country, or track and field teams. Participants were required to report to the laboratory on three separate occasions for either descriptive data collection or treadmill testing. Participants could not participate in any testing if they met any of the following exclusion criteria: previously diagnosed or current cardiac abnormalities, family history of cardiac abnormalities, idiopathic syncope, diabetes, cancer, medications that affect heart rate or blood pressure, pituitary disease, amenorrhea (amenorrheic for three or more months not controlled by medication), history of heat related illnesses, thyroid disease, pregnant or planning on becoming pregnant, smokers (current or quit within the last six months), hypertension (blood pressure >140/90mmHg), and exercise induced asthma. A health history questionnaire was given to participants to screen for these criteria. Indiana State University's Institutional Review Board approved the experimental protocol.

Instrumentation.

Anthropometric Data Collection.

Measurements were taken at seven sites, and body composition was determined using the Siri equation, according to standards set forth by the

American College of Sports Medicine (Thompson, Gordon, & Pescatello, 2009). All skinfolds were taken by the same trained technician. Body weight was measured using Transcell Technology Inc. scales (Model TI – 500 E Class III, Buffalo Grove, IL) and height was measured using a standard standiometer.

Electrocardiogram.

ECG readings were taken prior to any treadmill testing (Burdick Atria 6100, Deerfield, WI). Readings were taken while participants were seated, standing, and supine prior to taking part in any of the three tests (maximal or submaximal). Standard 12-lead electrode placement was used (see Figure 1).

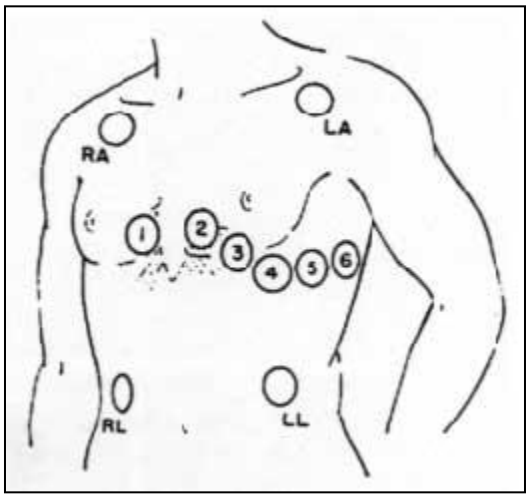

Figure 1. 12 lead ECG electrode placement.

Respiratory Data Collection.

A Medgraphics metabolic cart (Model Ultima CPX, St. Paul, MN) was used to collect breath by breath data. Heart rate was collected during the max test using a Polar heart rate monitor (Model RS200SD, Lake Success, NY) and was monitored during the submaximal tests through the Atria 6100.

Maximal Aerobic Test.

The maximal aerobic test was performed on a treadmill (Model Trackmaster TMX425CP, St. Paul, MN) and followed a previously published protocol (Paavolainen, Häkkinen, Hämäläinen, Nummela, & Rusko, 1999). Participants were deemed to have reached VO_2 max according to commonly used criteria (plateau in VO_2 consumption despite an increase in workload, heart rate ≥ age predicted maximum, RER ≥ 1.15, RPE of 19 or 20). Following completion of the test, the gradient was immediately returned back to 0%, treadmill speed reduced, and participants were required to remain jogging/walking on the treadmill until their heart rate returned to within 20-30 bpm of recorded resting heart rate. Fluids were available ad-libitum during the recovery phase.

Submaximal Treadmill Tests.

After a minimum of 48 hours of rest after the maximal exercise test, participants performed two submaximal treadmill tests at 70% of their pre-determined VO_2 max in different environmental conditions on two different days separated by at least 48 hours. The test days included submaximal tests in a thermoneutral and hyperthermic environment for up to 30 minutes. The order of tests was randomized. Participants remained connected to the ECG machine while exercising at 70% of their pre-determined VO_2 max. Momentary pauses of 30-40 seconds took place at five-minute intervals in order to gain ECG readings with as little interference as possible with the participant straddling the treadmill. Once the reading was printed, participants resumed running. Fluids were available ad-libitum throughout the test.

Statistical Analysis.

The baseline characteristics of the groups were analyzed using one-way analysis of variance (ANOVA). A 2 x 4 repeated measures ANOVA was used to test the effects of condition (thermoneutral, hyperthermic) by time (baseline, 5 minutes, 10 minutes, 15 minutes) on the changes in R and S wave voltage amplitude, PR interval duration, and ST elevation/depression. The analysis of ECG abnormalities was performed by coding the ECG based on adapted criteria from Pelliccia *et al.* (2000). If an interaction was deemed significant, a Tukey's HSD test was used for post-hoc comparisons. Significance was set *a priori* at $p<0.05$. Values are presented as mean ± SD. All statistical analyses were performed using SPSS Version 18 (SPSS, Inc., Chicago, IL, USA).

Results

Participant and Environmental Characteristics.

Participants were all NCAA Division I athletes involved in golf (n=2), cross country (n=8), and track and field (n=1). Participant descriptive data are presented in Table 1. One participant (cross country) of the original study population of 12 only completed two of the three tests (maximal and thermoneutral) and was therefore dropped from the analysis. The average running time in the thermoneutral and hyperthermic submaximal tests was 23.6 ± 8.4 minutes and 20.9 ± 7.7 minutes ($p>0.05$) respectively. Average laboratory temperature during the maximal and thermoneutral submaximal tests was 24.8 ± 1.6˚C (23.3 – 27.8˚C). Environmental chamber temperature was set at 38˚C for the hyperthermic submaximal tests.

Thermoneutral versus Hyperthermic across Exercise Time.

There was no significant condition by time effect found for any of the parameters (PR interval, $F_{1,6}$=0.870, p>0.05; R wave voltage, $F_{1,6}$=0.861, p>0.05; S wave voltage, $F_{1,6}$=0.607, p>0.05; ST segment position, $F_{1,6}$=1.157, p>0.05) (Figures 2, 3, 4, and 5 respectively). In addition, no significant difference was found with regard to coding the ECGs for abnormalities based on criteria outlined by Pelliccia *et al*. (2000) ($F_{1,6}$=0.571, p>0.05) for the interaction of condition by time.

Thermoneutral versus Hyperthermic.

No significant main effects of environment were found between PR interval ($F_{1,6}$=0.557, p>0.05), or S wave voltage amplitude ($F_{1,6}$=0.049, p>0.05) (Figures 2 and 4 respectively). In addition, changes in R wave voltage and ST segment position were not statistically significant but there was an increased R wave amplitude in the hyperthermic condition and increased ST segment alterations from baseline in the thermoneutral tests ($F_{1,6}$=4.250, p=0.073; and $F_{1,6}$=3.883, p=0.084 respectively) (Figures 3 and 5 respectively). No significant difference in occurrence of ECG abnormalities was observed ($F_{1,6}$=0.023, p>0.05) between environmental conditions according to Pelliccia *et al*. (2000) criteria. However, there were significantly (p<0.05) more ECG readings presenting waveform patterns that demonstrated incomplete left bundle branch block (ILBBB) in the hyperthermic (n=8) compared to the thermonuetral test (n=4).

Exercise Time.

Significant main effects were observed in several of the parameters across time such that PR interval decreased from rest at five, 10 and 15 minutes ($F_{1,6}$=31.343, p<0.05) (Figure 2), R wave voltage amplitude decreased at five, 10 and 15 minutes from

rest ($F_{1,6}$=10.613, p<0.05) (Figure 3) and; S wave voltage amplitude increased at five, 10 and 15 minutes from rest ($F_{1,6}$=9.706, p<0.05) (Figure 4). No significant difference in occurrence of ECG abnormalities was observed ($F_{1,6}$=0.227, p>0.05) across time.

Discussion

The results of the present study suggest that cardiac abnormalities not attributed to common athletic training adaptations, may be elucidated more frequently through submaximal running in a hyperthermic environment compared to a thermoneutral environment. The results demonstrate that 25.8% of ECG readings in the hyperthermic environment displayed waveforms consistent with ILBBB compared to 12.9% in thermoneutral (Table 2). These incidences contrast greatly to results presented in the work of Akhtar *et al.* (1993), in which none of the patients suffering from heat stroke, showed ECG waveform patterns consistent with left bundle branch block (LBBB).

While not statistically significant, perhaps the most physiologically significant results came from the abnormality analyses (Pelliccia *et al.* (2000). While normal cardiovascular responses to exercise such as a decrease in PR interval duration classified some ECGs as ‘mildly abnormal’ , several resting and exercise ECGs showed conditions that have been widely documented as innocent adaptations or “athlete’s heart syndrome”. These changes included sinus bradycardia, left ventricular hypertrophy (LVH), and incomplete right bundle branch block (IRBBB). Electrical and/or structural alterations to the heart of athletes, including those mentioned, have been reported in a number of studies (Corrado *et al.*, 2009; Corrado *et al.*, 2010; Pelliccia *et al.*, 2000; Maron & Pelliccia, 2006; Drezner *et al.*, 2009; Langdeau *et al.*, 2001; Basavarajaiah *et al.*, 2008; Holly, Shaffrath, & Amsterdam, 1998), most notably in athletes involved in endurance sports such as long-distance running, cycling, and rowing/canoeing

(Langdeau *et al.*, 2001; Pelliccia *et al.*, 2000). Results from the present study showed that 10 of the 11 participants had one or more of these common adaptations, either at rest, during exercise, or both at rest and during exercise (Table 2). In total, 82% of participants in the present study showed LVH at rest and 91% during exercise. IRBBB was seen in 45% of participants at rest, and 45% during exercise, regardless of condition (Table 2). With regard to displaying more than one abnormality, a total of four participants produced readings either at rest or during exercise that showed a combination of LVH and either IRBBB or ILBBB. In addition, an additional four participants displaying readings that demonstrated a combination of all three abnormalities during rest and/or exercise. While this incidence rate may appear to be abnormally high, it is not uncommon. Similar incidence rates have been reported previously, with up to 80% of athletes across various studies presenting some type of cardiac abnormality (Holly *et al.*, 1998). That statistic, combined with the study population being composed of primarily long-distance runners, makes the incidence rate of 91% in the present study understandable. What is perhaps most concerning, is that several participants produced ECG readings that met common criteria for ILBBB, an abnormality not usually associated with normal adaptations to athletes' hearts (Corrado *et al.*, 2010; Wilson *et al.*, 2011). Due to greater prevalence of ILBBB in the hyperthermic environment in the present study, it may be of benefit that athletes be evaluated in varying environmental conditions as part of cardiovascular pre-screening, as uncommon abnormalities may show in one condition more frequently than the other.

With regard to the prevalence of these abnormalities, the present study demonstrated similar incidence rates of LVH and IRBBB ('normal' adaptations) as reported in the work of Wilson *et al.* (2011). Wilson *et al.* (2011) observed these and other common training adaptations in the ECGs of 96.7% of the Caucasian athletes examined. More specifically, IRBBB was

observed in 60% of Caucasian athletes examined; a considerably higher occurrence rate than LVH in the same population (Wilson *et al.*, 2011). Similarly, Corrado *et al.* (2010) outlined data that suggests high IRBBB incidence rates in athletes, reporting occurrences of 35 to 50%, compared to just 10% in young, healthy individuals. Data from Langdeau *et al.* (2001) suggest that the right ventricular conduction delay is caused by an enlarged right ventricle cavity size and/or increased cardiac muscle mass and the resultant increased conduction time. Despite the high prevalence of IRBBB reported in athletes (Wilson *et al.*, 2011; Pelliccia *et al.*, 2000; Langdeau *et al.*, 2001), Corrado *et al.* (2010) suggested that further examination is not required in the presence of negative family/personal history and physical examination. In addition, Fagard *et al.* (1983) suggested that the right bundle branch block (RBBB) morphology is reversible with deconditioning. Based on data from the present study, prevalence of IRBBB among participants at rest and during exercise was similar to what has been observed in previous studies (Wilson *et al.*, 2011; Pelliccia *et al.*, 2000; Langdeau *et al.*, 2001) and can be attributed to a common and non-threatening adaptation of athlete's hearts to exercise training.

In the present study, changes observed on the exercise ECGs related to exercise time, while not necessarily statistically significant but may still be clinically relevant, included shortening of PR interval duration, alterations in R wave voltage amplitude, and alterations in S wave amplitude, were expected, and have been reported in previous studies (Nakamoto, Matsukawa, Murata, & Komine, 2005; Wolthuis, Froelicher, Hopkirk, Fischer, & Keiser, 1979; Deckers, Vinke, Vos, & Simoons, 1990; Baron, Ilsley, Sheiban, Poole-Wilson, & Rickards, 1980). While a decreased PR interval was expected during exercise, two participants displayed considerably short PR intervals (≤120 ms) at rest placing those ECGs in the "mildly abnormal" category (Pelliccia *et al.*, 2000). None of the participants presented a prolonged PR interval

(≤200 ms) at rest or during exercise; an abnormality reported in previous studies by Wilson *et al.* (2011) and Pelliccia *et al.* (2007). R wave amplitude alterations followed similar patterns as observed in the work of Wolthius *et al.* (1979) and Baron *et al.* (1980), whereby amplitude initially decreased from resting levels once exercise commenced, but as exercise continued, amplitude once again increased after heart rate rose above 140 bpm (Figure 3). In the present study, S wave progression followed similar changes as observed by Wolthius *et al.* (1979), whereby as exercise duration increased, S wave voltage amplitude became more negative. Taken together these data demonstrate voltage amplitude progression similar to what has been observed in previous studies (Wolthius *et al.*, 1979; Baron *et al.*, 1980) and should be expected as normal ECG waveform patterns during athletic activity.

In contrast to the widely reported prevalence of common ECG changes in athletes, only 5.8% of the Caucasian athletes in the Wilson *et al.* (2011) study showed uncommon ECG changes, such as LBBB, ST segment depression, and right ventricular hypertrophy. This is compared to 55% (*n*=6) of the present study which presented ECGs displaying waveform deformities indicative of ILBBB (either at rest and/or during exercise). More specifically, only one athlete out of the total Caucasian athlete population displayed ILBBB (Wilson *et al.*, 2011). In the present study, the high prevalence of ILBBB could be explained by the same disturbances in conduction seen in athletes related to physiological hypertrophy of the heart (Chapman, 1977). While cases of ILBBB are nearly always associated with organic heart disease, most often ischemic or hypertensive in origin, Chapman (1977) has outlined previous research that have reported findings of ILBBB in patients with apparently normal hearts. In addition, Chapman (1977) presented a case study in which a 30 year old male (in very good physical condition) presented ECG readings with intermittent ILBBB, where the block was present at rest (heart rate

of 78 to 85 bpm), but also appeared during exercise at a heart rate greater than180 bpm (although it was not present at a rate of 175 bpm). The findings of Chapman (1977) may help to explain the high prevalence of ILBBB found in the present study. However, it should not completely exclude the possibility of a previous myocarditis, and therefore further evaluation upon discovery of ILBBB in athletes should be utilized in order to better understand the underlying cause.

Also of interest in the present study was the high rate of abnormalities found in female athletes in the study. This is in contrast to previous studies reporting that female athletes generally present ECG abnormalities considerably less than male athletes (Pellicccia *et al.*, 2000; Langdeau *et al.*, 2001). This could be in large part due to a small study population, and a more significant study population may yield female prevalence of cardiac abnormalities at a rate more similar to what has previously been demonstrated. In addition, inclusion of athletes involved in a wider range of sports that utilize power/explosiveness more than cardiovascular endurance may alter ECG abnormality prevalence.

As previously discussed, current cardiovascular pre-participation screening for college athletes in the US have been reported to be largely inadequate at many institutions across the country (Pfister *et al.*, 2000), however, this issue is not limited to north America. Many health professionals across Europe support the need for a 12-lead ECG to screen for potential cardiac abnormalities in young athletes (Corrado *et al.*, 2005; Corrado *et al.*, 2009). While the present study used a specific population, it is hard to ignore the striking prevalence in ILBBB given the comparatively low incidence rates in previous studies (Corrado *et al.*, 2010; Wilson *et al.*, 2011). While many athletes may show ECG abnormalities that are normal adaptations to training, abnormalities that are less common (such as ILBBB) should be of more concern. Furthermore, if a simple test of cardiac form and function combined with heat stress can elucidate these

abnormalities, incidences of SCD in college student-athletes could potentially be reduced, or perhaps prevented altogether.

It is important to note that the participants in the present study were not necessarily acclimatized to the hyperthermic environment. Heat acclimatization is the collective physiologic adaptive changes to exercising in the heat can be made by the body that allows for improvement in tolerance to heat exposure such as improved cutaneous blood flow, lowered threshold for the start of perspiration, lowered salt concentration of perspiration, and less reliance on carbohydrate catabolism during exercise. Other changes observed as a result of heat acclimatization include a decrease in heart rate and core body temperature (indicative of lessened physiologic strain) as well as an increase in blood plasma (Casa, 1999).

Conclusion

This study has suggested that submaximal running in contrasting environmental conditions does not elicit a significant difference in ECG parameters of PR interval duration, R wave voltage amplitude, S wave voltage amplitude, or ST segment elevation/depression.

While a significant interaction between conditions was not observed, this study reported a high prevalence of ILBBB at various stages of exercise in both environments. This high incidence rate lends support to the body of discussion that already exists around implementing more thorough pre-participation screening protocols for young athletes as a means to reduce SCD in this population. It should be noted that in light of the increased prevalence of ILBBB in hyperthermic conditions, athletes that train in areas with warmer climates may benefit from submaximal ECG screening in a hyperthermic environment.

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Table 1. *Participant Group Demographics (mean ± SD).*

Age (years)	20.5 ± 1.4
Height (cm)	167.8 ± 4.8
Weight (kg)	60.0 ± 4.5
VO_2 Max (ml/kg/min)	56.1 ± 12.2
Body Fat (%)	14.4 ± 7.9
Body Density	1.066 ± 0.018

Table 2. *Percentage of ECG Readings Taken at Time Intervals Presenting LVH, IRBBB, and/or ILBBB Between Test Conditions (actual number of ECGs in parentheses).*

Condition	Time (mins)	LVH	IRBBB	ILBBB
Thermoneutral	5	91% (10/11)	18% (2/11)	18% (2/11)
	10	91% (10/11)	18% (2/11)	9% (1/11)
	15	78% (7/9)	33% (3/9)	11% (1/9)
Hyperthermic	5	64% (7/11)	18% (2/11)	27% (3/11)
	10	50% (5/10)	30% (3/10)	20% (2/10)
	15	80% (8/10)	10% (1/10)	30% (3/10)

LVH=Left ventricular hypertrophy; IRBBB=Incomplete right bundle branch block;

ILBBB=incomplete left bundle branch block.

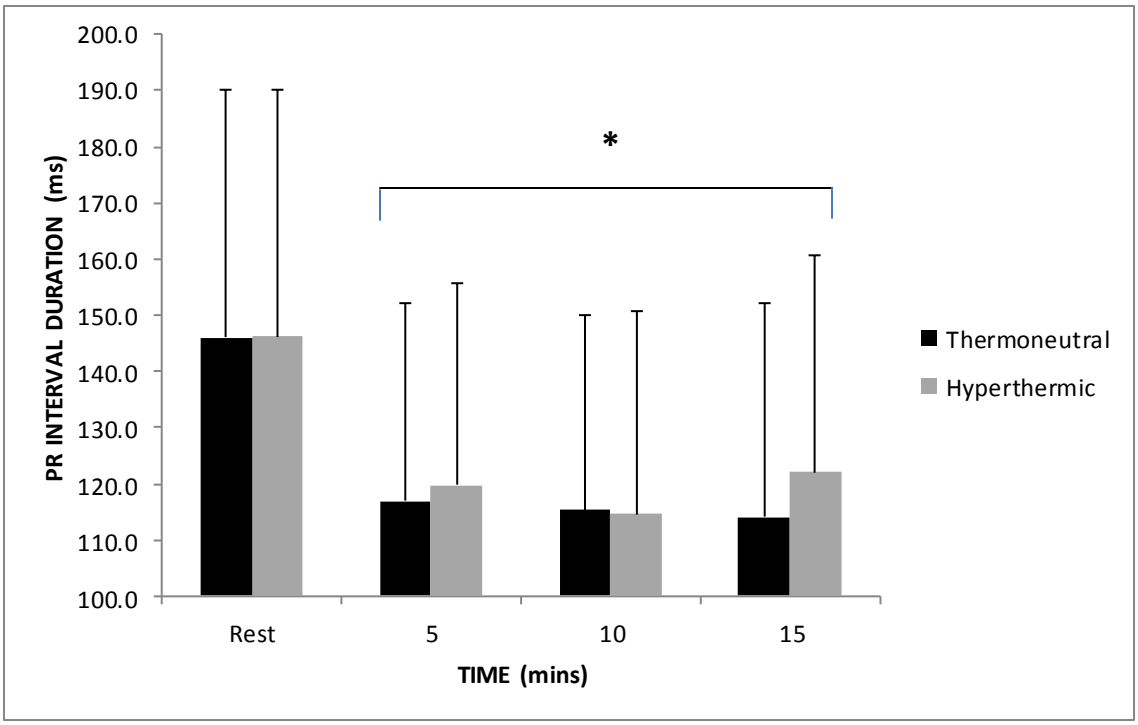

Figure 2. Mean PR interval duration alterations by condition and exercise time.

Data are mean ± SEM

*$p<0.05$, significant difference from rest in both conditions.

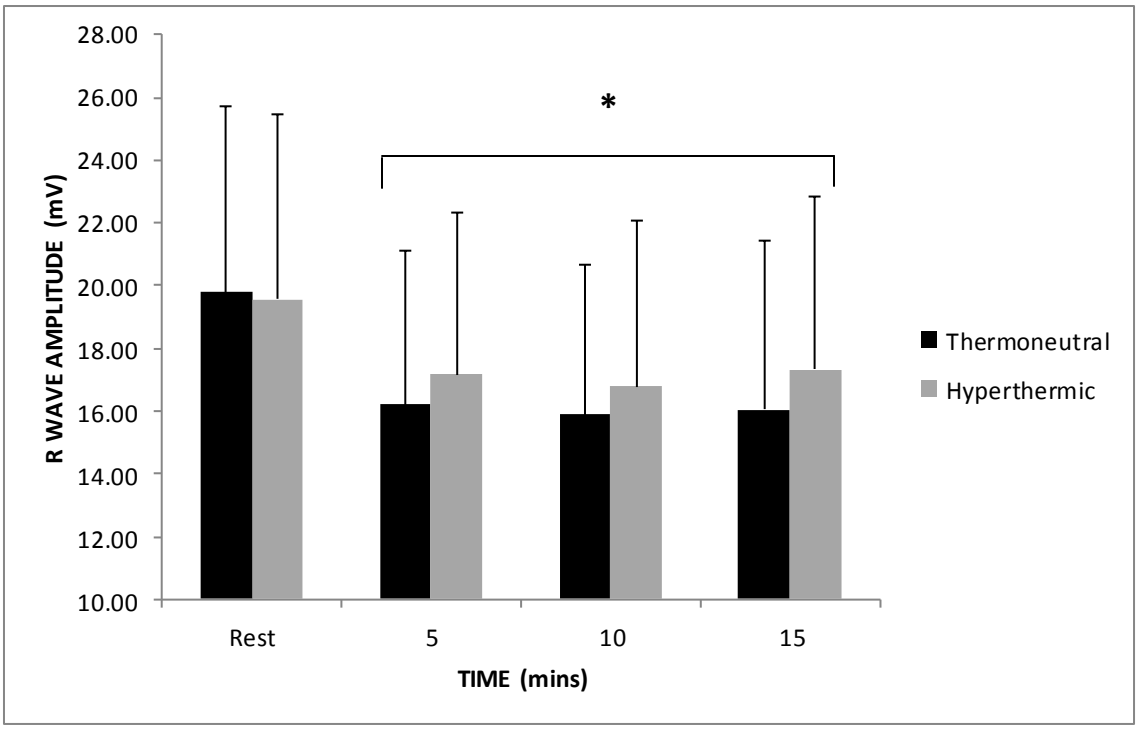

Figure 3. Mean R wave amplitude alterations by condition and exercise time.

Data are mean ± SEM

*$p<0.05$, significant difference from rest in both conditions.

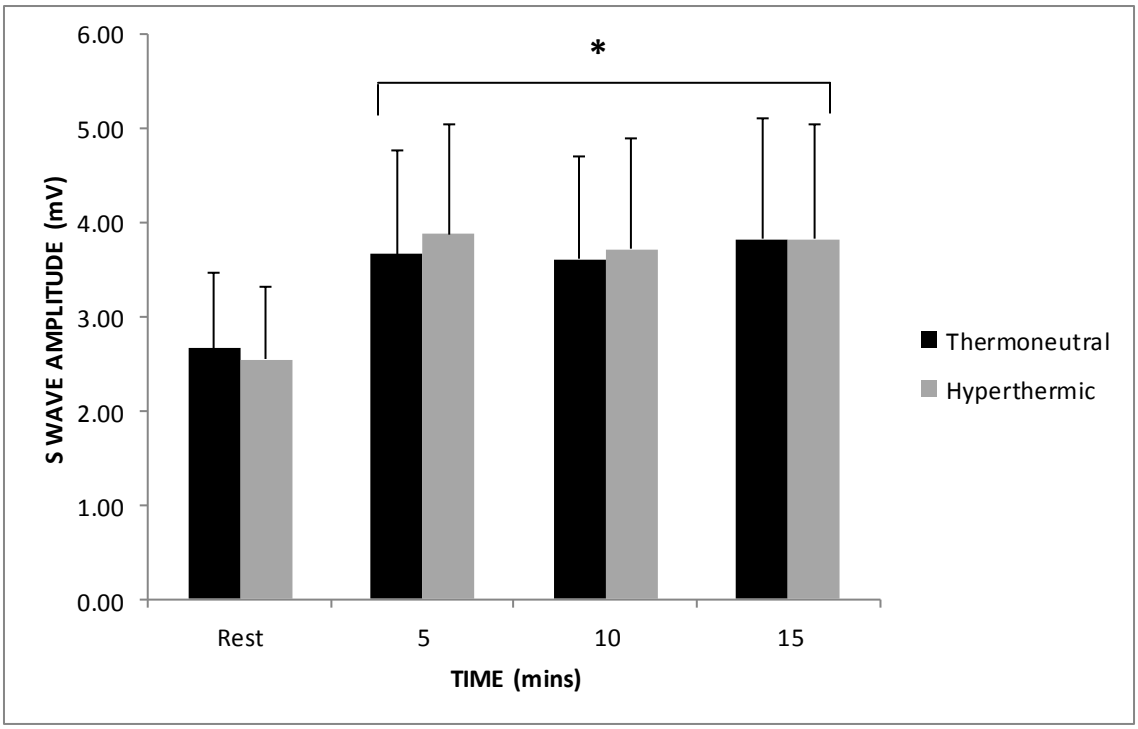

Figure 4. Mean S wave amplitude alterations by condition and exercise time.

Data are mean ± SEM

*p<0.05, significant difference from rest in both conditions.

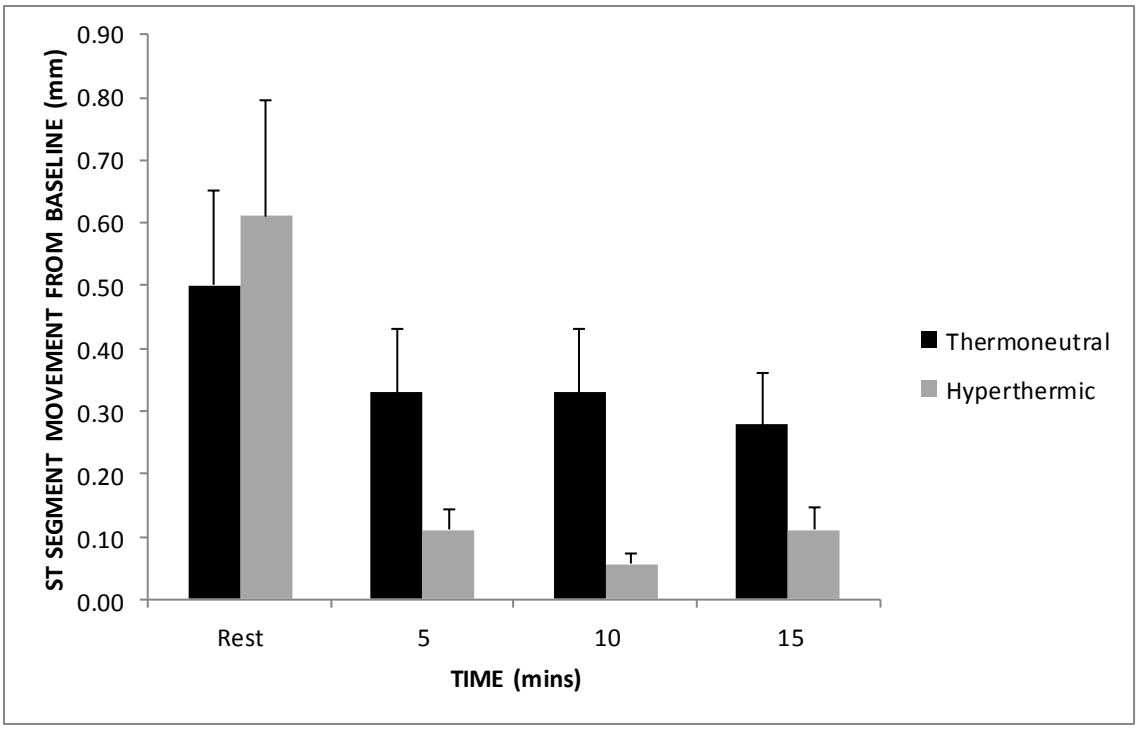

Figure 5. Mean ST segment alterations from baseline by condition and exercise time.

Data are mean ± SEM

Proposal

Chapter 1: Development of the Study

Introduction

It is widely accepted, that coronary heart disease is the leading cause of death in the US today (Cardiovascular Disease Statistics, n.d.). Data suggest that in 2005, 652,091 deaths were attributed to some form of heart disease, accounting for 27.1% of all US deaths for that year (Kung, Hoyert, and Murphy, 2008; cited in "Heart Disease Facts and Statistics", 2009). What is perhaps less well known is that the leading cause of death in young athletes is a phenomenon referred to as sudden cardiac death (SCD) (Maron *et al.*, 2009; cited in Drezner, Pluim, & Engebretsen, 2009). Incidence rates for SCD are relatively unknown due to the difficulty of comparing studies with profoundly different methodology and across vastly different geographic locations. This leads many estimates to be based upon public media reports and other electronic databases (Drezner, *et al.*, 2009). Some studies have estimated that between 1:160,000 to 1:300,000 competitive athlete deaths in the US can be attributed to cardiovascular diseases (Maron *et al.*, 2009; Van Camp *et al.*, 2009; cited in Drezner, *et al.*, 2009). However, a recent study conducted by Corrado *et al.*, (2006) across a significantly large population in the Veneto region of Italy, reported incidence figures for SCD in young athletes (aged 12-35 years) as high as 1:28,000. These findings are similar to that of a recent study conducted at 11 US and Canadian cities that reported incidences of sudden cardiac arrest (SCA) from cardiovascular disease for children and adults (ages 14-24 years) at 1:27,000 (Atkins *et al.*, 2009). With regard to the type of cardiovascular disease responsible for SCD in young athletes, Maron *et al.* (2007)

suggest that hypertrophic cardiomyopathy (HCM) is the leading cause, with analysis of 1435 cases of SCD in young competitive athletes revealing 36% of deaths could be attributable to HCM. Papadakis, Whyte & Sharma (2008) further adapted the work of Maron *et al.* (2007), stating that 14% of SCD in young athletes can be attributed to arrhythmias.

Rywik *et al.* (2002) suggested that the electrocardiographic response to treadmill exercise plays a significant role in the ability to diagnose and assess individuals with suspected coronary heart disease. The authors outlined research that has shown that a horizontal or down-sloping ST-segment depression ≥ 1 mm in response to exercise is a powerful predictor for future coronary events, including myocardial infarction and SCD, in an apparently healthy population.

The findings outlined suggest that initial incidence rates may have significantly underestimated the magnitude of the problem of SCD in athletes and that an exercise stress test may be conducive to bringing about observable cardiac abnormalities in an otherwise healthy, previously undiagnosed population. Therefore, there is a need to provide a more clear perspective of this issue, helping to increase awareness and improve preventative measures for athletes.

Statement of the Problem

Current standards of cardiovascular pre-participation screening for American collegiate student-athletes are inadequate (Pfister, Puffer, & Maron, 2000). Many health care professionals throughout Europe are proposing that a standard 12-lead electrocardiograph (ECG) in addition to current forms of screening would aid in the ability to accurately detect potential cardiac disorders in young athletes (Corrado *et al.*, 2005). The present study may help further expand on the current literature on incidence rates of cardiac abnormalities in an athletic population and aid in

the discussion to implement a more comprehensive cardiovascular pre-participation screening process for collegiate athletes in the US.

Purpose of the Study

The purpose of this study was to examine the incidence rate of cardiac abnormalities in a collegiate athletic population through exposure to physiological and thermal stressors.

Limitations

1. Availability of athletes for testing.
2. Each individual brought a certain amount of variability with them that presented a threat to the validity of the study.

Delimitations

1. The study was limited to students currently enrolled at Indiana State University; therefore results can only be applied to collegiate athletes that meet the inclusion criteria for the study.
2. The study only included athletes from the golf, cross country, and track and field teams of Indiana State University, therefore results cannot be applied to other NCAA Division I athletes participating in sports other than those mentioned.
3. Heat stress test was performed in a controlled environmental chamber.
4. Physiological stress came in the form of controlled treadmill running.

Hypothesis

It has been suggested that athletes may have some form of underlying, undiagnosed cardiac abnormality that may result in increased incidence rates of SCD (Corrado *et al.*, 2006). If athletes have these undiagnosed cardiac abnormalities, then simple tests of cardiac form and function in response to acute physiological and thermal temperatures may elucidate them.

Therefore, it was hypothesized that if collegiate athletes are exposed to appropriate physiological and thermal stressors, any undiagnosed cardiac abnormalities will manifest on an ECG (APPENDIX C). To test this hypothesis, collegiate student-athletes underwent an ECG test before a maximal stress test, and during submaximal stress tests performed in a thermoneutral and a hyperthermic environment.

Chapter 2: Review of Literature

Prevalence and Common Types of Cardiac Abnormalities in Athletes

Pelliccia *et al.* (2007) examined data of a significant subject population that was predominantly young amateur male athletes (80%) involved in sports such as soccer, volleyball, basketball, and athletics. Based off commonly used clinical criteria for evaluating ECG patterns, 11.8% of the population showed abnormal 12-lead ECG patterns, with the most common abnormalities present being a prolonged PR interval, incomplete right bundle branch block (IRBBB) and early repolarization pattern (Pelliccia *et al.*, 2007). Distinct ECG abnormalities also found included deeply inverted T-waves in .2 precordial and/or standard leads and increased R/S wave voltages suggestive of left ventricular hypertrophy (LVH) (Pelliccia *et al.*, 2007).

In terms of the most prevalent cardiac abnormalities related to SCD, the work of Corrado, Basso, Schiavon, & Thiene (1998) in examining the effectiveness of the pre-participation screening program adopted in Italy, has suggested that arrhythmogenic right ventricular cardiomyopathy (ARVC) is the leading cause of death among young athletes (age ≤35 years). Since 1971, Italian law has required that every athlete undergo an annual clinical evaluation in order to be approved to take part in competitive sports. A competitive athlete is defined as "a participant in an organized sports program requiring regular training and competition" (Maron *et al.*, 1996). From 1979 to1996, a consecutive series of 33,735 young athletes (28,539 male and 5196 female athletes with a mean age of 19 ± 5 years) in the Veneto region of Italy underwent pre-participation cardiovascular screening. The cardiac disease screening process was part of a

more comprehensive medical examination that consisted of general clinical history taking physical examinations, orthopedic examinations, spirometry, and urinalysis (Corrado *et al*, 1998). The initial cardiovascular testing protocol began with taking family and personal history, physical examination (including testing blood pressure), basal 12-lead ECG, and limited exercise testing. Additional tests including echocardiography and 24-hour ambulatory Holtner monitoring were requested for subjects who had positive findings, such as syncope or near-syncope, chest pain or discomfort, and irregular heartbeat or palpitations on exertion (Corrado *et al.*, 1998). Aside from ARVC (22.4%), the most common causes of sudden death in athletes were coronary atherosclerosis (18.4%) and anomalous origin of a coronary artery (12.2%), with HCM accounting for only one sudden death (2.0%) among athletes (although it was more common in non-athletes at 7.3%) (Corrado *et al.*, 1998). The relatively low incidence rate of HCM reported by Corrado *et al.* (1998) differs considerably from similar research coming out of the US, in which causes of cardiac arrest in younger competitive athletes have been attributed to HCM, accounting for about one third of fatal cases (Maron *et al.*, 2007). Similarly, Maron *et al.*, (2003) examining deaths in young competitive athletes, reported that of the 286 documented cardiovascular deaths, most were due to HCM (36%) or anomalous coronary artery of wrong sinus origin (13%), such as Wolfe-Parkinson-White syndrome.

Maron, Roberts, McAllister, Rosing, & Epstein (1980) examined the cases of 29 athletes; of which, 22 had died during or shortly following severe exertion on the athletic field. Their findings showed that in 28 of the 29 athletes studied, structural heart disease was identified and was likely the cause of sudden death in 22 of those cases, with HCM accounting for almost half of the deaths (14 out of 29) in the series (Maron *et al.*, 1980). Perhaps one of the more concerning statistics that arose from this study was that cardiac disease was unrecognized during

life in most of the athletes, with only two receiving a correct diagnosis ante mortem (Maron *et al.*, 1980).

The work of Hebbar & Hueston (2002) supported those findings, suggesting that malignant ventricular tachycardia is perhaps the arrhythmia that is of most concern to athletes and is usually associated with idiopathic HCM.

Differences in Athlete ECG Readings Related to Ethnic Background

As previously mentioned, HCM is the most common structural cardiovascular finding that can be attributed to sudden death in US athletes, with the condition being considerably more prevalent in African-American athletes than Caucasian athletes (Magalski *et al.*, 2008; Maron, Doerer, Has, Tierney, & Mueller 2009; Basavarajaiah *et al.*, 2008; Maron *et al.*, 2003). Research by Maron *et al.* (2003) on a number of sudden deaths in young athletes showed that of the deaths attributable to HCM, 55 percent were African-American compared to 41 percent Caucasian. A study conducted by Magalski *et al.* (2008) on elite American football players reported abnormal ECG patterns in 25% of the athletes tested, with abnormal readings significantly more common among African-American players (30%) compared with Caucasian players (13%) or other races (15%). In addition, distinctly abnormal ECG patterns that were suggestive of cardiac disease were also more common in African-American players (6%) compared with Caucasian (2%) (Magalski *et al.*, 2008). It was suggested that this significant difference in prevalence among races can at least be partly attributed to physiological cardiovascular differences that are common between non-Caucasian and Caucasian athletes, namely increased left ventricular (LV) wall thickness and cavity size in non-Caucasian athletes (Basavarajaiah *et al.*, 2008). Non-Caucasian athletes develop a greater magnitude of left ventricular hypertrophy (LVH) compared with Caucasian athletes, which has the potential to increase the likelihood of generating false-

positive diagnoses of HCM in non-Caucasian athletes (Basavarajaiah *et al.*, 2008). Maron *et al.* (2009) examined sudden deaths in young competitive athletes in the US from 1980-2006, analyzing a total of 1866 deaths. While the absolute number of cardiovascular deaths reported in Caucasian athletes (581; 55%) exceeded that in African-American athletes (377; 36%) and other minority races, the number of deaths due to cardiovascular disease was higher in non-Caucasian than Caucasian athletes (64% and 51%, respectively) (Maron *et al.*, 2009). In addition, non-Caucasian athletes (predominantly African-Amercian) had a higher number of reported deaths attributable to HCM and congenital coronary anomalies than Caucasian athletes, with 136/676 (20%) compared with 112/1135 (10%) for HCM, and 66/676 (10%) versus 52/1135 (5%) for coronary abnormalities (Maron *et al.*, 2009). Conversely, Caucasian athletes had a higher fraction of reported deaths attributable to ion channelopathies (22/1135; 2%) compared to 2/676 (0.3%) among non-Caucasian athletes (Maron *et al.*, 2009).

Gender Differences in Abnormal ECG Readings and Sudden Death Frequency Among Athletes

Pelliccia, *et al.* (2000) reported findings that displayed a significant discrepancy in the frequency of distinctly abnormal (17% versus 8%), or mildly abnormal (28% versus 14%), ECG readings in trained male and female athletes. It was demonstrated that male athletes showed greater maximum R or S wave voltages and more frequently abnormal Q waves compared to female athletes. In addition, there was a marked difference in the number of female athletes that displayed normal ECGs (78%) compared with male athletes (55%) (Pelliccia *et al.*, 2000). Langdeau, Blier, Turcotte, O'Hara, & Boulet (2001) examined heart abnormalities in athletes, and reported that male long-distance runners had a considerably higher incidence rate of cardiac abnormalities. There were none of the female long-distance runners examined that showed left

ventricular hypertrophy (LVH) or incomplete right bundle branch block (IRBBB), compared to six of 10 males that presented with LVH, four of seven IRBBB, and two with both abnormalities.

With regard to gender differences in incidence rates of SCD in athletes, the sheer size of the sample population used by Carrado *et al.* (1998) provides the most comprehensive data set. Of the 269 sudden deaths that occurred from 1979 to 1996 in people 35 years of age or less in the Veneto region of Italy, 49 were among competitive athletes (44 male and 5 female) out of a total sample of 28,539 male, and 5,196 female athletes (0.15% and 0.10% respectively). In a separate article, Corrado *et al.* (2005) highlights the significant male athlete predominance in sudden death incidences, and suggests that this predominance can be related to the higher participation rate of male compared with female athletes in competitive sports. In addition, males generally uitilzed a more intensive training load and higher levels of athletic achievement**.** More recently, it has been proposed that male gender in itself is a risk factor for sports-related sudden death. This is most likely due to the greater prevalence and/or phenotypic expression in young males of cardiac diseases at risk of arrhythmic cardiac arrest (Corrado *et al.*, 2005).

Common ECG Changes Observed in Athletes as a Result of Training

There is a substantial amount of evidence to suggest that competitive athletes may display abnormal ECGs as a result of their training regimes, which may alter both structural and/or electrical components of the myocardium (Corrado, Biffi, Basso, Pelliccia, & Thiene, 2009; Corrado *et al.*, 2010; Pelliccia *et al.*, 2000; Maron & Pelliccia, 2006; Drezner *et al.*, 2009; Langdeau *et al.*, 2001; Basavarajaiah *et al.*, 2008; Holly, Shaffrath, & Amsterdam, 1998; Wilson *et al.*, 2011). Changes that have been reported include sinus bradycardia, left and right ventricular remodeling (including increased wall thickness and chamber size), increased left atrial cavity size (and volume), increased RR, PR, and QT intervals, first-degree

(atrioventricular) AV block and Mobitz type I (Wenkebach) second-degree AV block (Corrado *et al.*, 2009; Maron & Pelliccia, 2006; Langdeau *et al.*, 2001). Up to 80% of trained athletes can show some of these ECG changes that result from physiological adaptation of the autonomic nervous system to athletic conditioning, including changes such as increased vagal tone and/or withdrawal of sympathetic activity (Holly *et al.*, 1998).

These structural and electrical changes have been observed across a wide variety of sports, but are more commonplace in endurance events such as long-distance running, cycling, rowing/canoing, and cross-country skiing (Langdeau *et al.*, 2001; Pelliccia *et al.*, 2000). This phenomenon is commonly referred to as "athlete heart syndrome" and is a crucial factor to consider when analyzing ECG tracings of trained athletes. These innocent adaptations are a result of long-term, intense physical conditioning, which could potentially be misconstrued as abnormal and result in a false-positive diagnosis of a cardiac disorder (Pelliccia *et al.*, 2000).

Sport Participation and Potential Issues for Athletes Diagnosed With or Suspected of Having a Cardiac Abnormality

As was the case of Corrado *et al.* (1998, 2006), part of the screening process involved investigating the family history of the individual concerned, looking for possible warning signs, or 'red flags', that may increase the likelihood that the individual is possibly at risk from a cardiac disorder. Hebbar & Hueston (2002) suggest that symptoms of syncope or near-syncope with exercise, or a family history of SCD in a close relative are significant warning signs of the presence of idiopathic HCM. In addition, if an athlete has an aortic murmur that increases while performing a Valsalva maneuver, a sympathetic stimulator, they should be evaluated for HCM before being allowed to participate in sports (Hebbar & Hueston, 2002). Furthermore, if HCM is identified, treatment with a beta blocker or a calcium channel blocker may reduce cardiac

contractility and heart rate (HR) during exertion. Or alternatively, the insertion of an implantable cardioverter-defibrillator may be required to regulate HR during exercise (Hebbar & Hueston, 2002). In terms of participation in sports, Hebbar & Hueston (2002) and Pelliccia *et al.* (2005) agreed that a restrictive attitude should be employed, and participation in strenuous sports should be avoided in athletes with HCM. Mitchell *et al.* (1994) present low-intensity sports such as bowling, golf, and billiards as possible alternatives (cited in Hebbar & Hueston, 2002). An interesting point is also raised by Hebbar & Hueston (2002) in which they outline reports of high-performance athletes frequently complaining of diminished performance or fatigue while taking medications. This in turn may lead them to be non-compliant with treatment, thus putting them at further risk of SCD.

Current Cardiovascular Pre-participation Screening Protocols for Athletes and the Argument For and Against Changes

As previously mentioned, the extensive work of Corrado *et al.* (1998, 2006) investigating the effectiveness of screening athletes prior to taking part in competitive sports has provided significant results. This is highlighted by a substantial decrease in incidence rates of SCD in young athletes in the Veneto region of Italy since the implementation of the screening program almost 30 years ago. During the period of 1979-1980, the incidence rate was 3.6 deaths per 100,000 persons (8 sudden deaths) and throughout 1981-1982 it had increased to 4.0 deaths per 100,000 persons (9 sudden deaths). Following this, however, the annual death rate decreased markedly over time until the 2001-2004 period, at which point there were only 0.43 deaths per 100,000 persons (one sudden death each year period) (Corrado *et al.*, 2006). The greatest decline in causes of SCD was seen in arrhythmogenic right ventricular cardiomyopathy, from 0.9 deaths

per 100,000 persons during the pre-screening period (1979-1981) to 0.15 deaths per 100,000 persons in the late screening period (1993-2004); a decrease of 84 percent (Corrado *et al.*, 2006).

The success of the pre-participation screening program in Italy has prompted the medical field to propose a common European protocol for sport pre-participation screening. Cardiovascular specialists and other physicians from various European countries that have extensive clinical experience in dealing with young competitive athletes suggest a process that is essentially based on a 12-lead ECG (in addition to the more common practice of taking personal and family history and physical examination) (Corrado *et al.*, 2005). In the US, the current screening process excludes the use of an ECG as it is not seen as a cost-effective method due to the relatively low incidence rate of SCD in athletes and the possibility of inaccurate diagnoses due to inexperience of the tester (Maron, Gohman, & Aeppli, 1998; Williams & Chen, 2003). However, it is possible that it may enhance the sensitivity of the screening process and potentially allow for more effective detection of cardiovascular diseases (Corrado *et al.*, 2009). The current process utilized in the US has been shown to be somewhat ineffective, with one retrospective analysis on 134 high school and collegiate athletes that died suddenly, revealing that only 3% of the examined athletes were suspected of having a cardiac abnormality, with only 1% eventually receiving an accurate diagnosis (Maron, *et al.*, 1996). In addition, Pfister, Puffer & Maron (2000) examined the current pre-participation cardiovascular screening process for US collegiate student-athletes. Of the screening forms analyzed from 625 institutions, only 163 schools (26%) had forms that contained at least 9 of the recommended 12 American Heart Association (AHA) screening guidelines. These forms were considered adequate, whereas forms that contained four or fewer of these questions (24%) were considered to be inadequate (Pfister, *et al.*, 2000). Other findings from the study revealed that NCAA Division I schools were more

likely to have adequate screening forms than Division II and III schools, and often those responsible for performing examinations of the athletes were considerably inexperienced in the area of cardiovascular abnormalities. This prompted the authors to suggest that changes need to be made to improve effectiveness of the screening process (Pfister, *et al.*, 2000).

Common Physiological Responses during Exposure to Heat

Responses to Heat at Rest.

Physiological responses to changes in temperature are regulated by the hypothalamus, which acts to maintain a core body temperature of 37˚C ± 1˚C via two mechanisms. One mechanism is thermal receptors in the skin, and the other through changes in the temperature of blood that perfuses the hypothalamus. Thermal homeostasis is maintained by achieving a balance between heat gain and heat loss from the body. The two recognized sources of heat load are environmental (heat either being gained or lost) and metabolic (generated by muscular activity) (McArdle, Katch & Katch, 2007).

The mechanisms of convection, conduction, radiation, and evaporation are responsible for regulating heat exchange between the body and the external environment, with the effectiveness of these mechanisms decreasing as ambient temperature increases (McArdle, Katch & Katch, 2007).

Convection.

The term convection refers to the repetitive process of cool air taking the place of warm air as it rises from the surface of the skin (due to warm air being less dense than cool) (McArdle, Katch & Katch, 2007). When the air temperature is greater than skin temperature, the body will gain heat from the surrounding air

and conversely, when skin temperature is greater than air temperature, there will be a loss of heat from the body (McArdle, Katch & Katch, 2007). This rate of convective exchange is dictated by differences between the skin and air temperature along with the rate of air movement over the skin (McArdle, Katch & Katch, 2007).

Conduction.

Conduction refers to the process of heat moving down its warmer thermal gradient to the cooler object upon contact, with the heat energy being transferred from molecule to molecule (McArdle, Katch & Katch, 2007). This temperature transfer continues until the temperature of the two objects is equalized, and the rate of this heat transfer depends on two key factors: the temperature between the two objects, and the thermal conductivity of the two objects (McArdle, Katch & Katch, 2007).

Radiation.

The surface of the human body is constantly emitting heat in the form of electromagnetic waves through a process known as radiation, with the absolute temperature of the radiating surface determining the rate of emission (McArdle, Katch & Katch, 2007). The temperature difference between the surface of the body and the average temperature of the various surfaces in the surrounding environment determines if net heat is lost or gained, with the rate of heat loss or gain being directly dependent on the temperature difference (McArdle, Katch & Katch, 2007).

Evaporation.

Evaporation describes the process of transforming water (in the form of perspiration) from a liquid to a gas and being dissipated from the surface of the skin (McArdle, Katch & Katch, 2007). If perspiration drips from the skin or is wiped away, no heat will be lost, as it is the liquid to gas transformation process that acts to cool the body (McArdle, Katch & Katch, 2007). This active process requires energy and is controlled by the sympathetic nervous system, allowing the rate at which it occurs (and subsequent heat loss) to be controlled by the body (McArdle, Katch & Katch, 2007). When environmental temperatures reach high levels (exceeding approximately 36˚C), heat is lost exclusively by evaporation, and as the temperature continues to increase, heat is taken up by the body from the environment via radiation, conduction, and convection, causing sweating to become profuse in order to maintain the balance between heat uptake and heat loss by evaporation (McArdle, Katch & Katch, 2007).

Responses to Heat During Exercise.

During exercise, heat production is 15-20 times greater than at rest, and has the potential to raise core body temperature 1˚C every five minutes if there are no thermoregulatory adjustments (Nadel, Wenger, & Roberts, 1977; cited in Coris, Ramirez, & Van Durme, 2004). In order to avoid significant hyperthermia, mechanisms such as conduction, convection, radiation, and evaporation are responsible for cooling the body's core temperature that is raised by the generated heat as well as ambient heat from the external environment (Bouchama & Knochel, 2002; Armstrong *et al.*, 1996; Simon, 1993; cited in Coris *et al.*, 2004). While all cooling mechanisms will contribute to lower

core temperature, evaporation as perspiration is responsible for the majority of heat dissipation, with research suggesting that in hot, dry conditions, evaporation may be responsible for as much as 98% of heat dissipation (Armstrong & Maresh, 1993; cited in Coris *et al.*, 2004).

Dehydration can not only affect a rise in core body temperature, but also a considerable decrease in athletic performance, work capacity, or both with as little as 2-3% dehydration. In addition, factors that limit evaporation such as high humidity or dehydration can have profound effects on physiological function, athletic performance, and risk for heat illness (Casa, Armstrong, Hillman *et al.*, 2000; Armstrong, Costill, & Fink, 1985; cited in Coris *et al.*, 2004).

Physiological adaptations to exercising in the heat can be made by the body by a process referred to as heat acclimatization; a term that describes the collective physiologic adaptive changes that act to improve tolerance to heat exposure such as improved cutaneous blood flow, lowered threshold for the start of perspiration, lowered salt concentration of perspiration, and less reliance on carbohydrate catabolism during exercise (McArdle, Katch & Katch, 2007). Other changes observed as a result of heat acclimatization include a decrease in heart rate and core body temperature (indicative of lessened physiologic strain) as well as an increase in blood plasma (Casa, 1999). In addition, an individual's body composition can significantly affect their tolerance to heat exposure when exercising. A large overweight individual (with a high level of body fat) will expend more energy than a leaner individual when moving their body mass, resulting in increased metabolic heat production (Armstrong, 2003). The elevation of body temperature is higher, for a given heat load and per kilogram of body mass, in obese

individuals compared with those who are not, putting obese individuals at an increased risk of becoming hyperthermic during exercise (Armstrong, 2003). In addition, large individuals with high levels of muscle mass may also be at an increased risk of hyperthermia due to the level of metabolic heat produced by muscles during exercise (Armstrong, 2003). With regard to gender differences in heat acclimatization, early research suggested that men exhibited a greater tolerance to environmental heat stress than women during a standard bout of exercise; however, this research was severely flawed as it required women to exercise at a higher percentage of aerobic capacity than men (McArdle, Katch & Katch, 2007). When researchers controlled for this factor, the differences in thermoregulation were less pronounced when comparing men and women of equal fitness (or exercising both at the same percentage of VO_2 max) (Drinkwater *et al.*, 1976; Horstmen & Christensen, 1982; cited in McArdle, Katch & Katch, 2007). In contrast to this, Grogan & Hopkins (2002) suggests that the body temperature at which thermoregulatory mechanisms are activated is lower in women than in men, with women appearing to store less heat than men for a given workload. Because of this, women seem to be more "protected" from the onset of exertional heat stroke than men, and very rarely are cases of exertional rhabdomyolosis reported (Grogan & Hopkins, 2002). While the exact reason for this difference is unknown, the author suggests several possible explanations, including the effect of estrogen or men having a greater ability to generate heat due to larger muscle mass (Grogan & Hopkins, 2002).

ECG Changes Observed During Heat Exposure and Exercise in Hyperthermic Conditions

Exposure to a hyperthermic environment has been shown in several cases to alter cardiovascular function and provide abnormal ECG readings (Rautaharju, Wolf, Piironen, Äikäs,

& Karvonen, 1966; Akhtar, Al-Nozha, Al-Harthi, & Nouh, 1993). This has been reported in both sedentary exposure to ambient heat (Rautaharju *et al.*, 1966) and in cases where prolonged exposure to heat combined with some magnitude of physical stress has lead to heat stroke (Akhtar *et al.*, 1993). The condition of heat stroke, defined by three common symptoms: hyperthermia (rectal temperature in excess of 41˚C), hot, dry skin combined with an absence of perspiration, and severe central nervous system disturbances, occurs when the body is unable to cool itself to normal levels (36.5 – 37.5˚C core temperature) when exposed to hot and/or humid conditions (Akhtar *et al.*, 1993). The work of Akhtar *et al.* (1993) examined Hajj pilgrims (mean age 59.6 years) from different parts of the world that had been admitted to a medical center diagnosed with heat stroke after performing the Hajj pilgrimage in Mecca (or other holy places), with ECGs recorded 12 and 24 hours after cooling. Common ECG changes observed included an increased Q-T interval, sinus tachycardia, and diffuse ST-T changes that were highly suggestive of myocardial ischemia. In addition, other parameters such as atrial fibrillation, intraventricular conduction defect, right bundle branch block, and acute myocardial infarction were also present (Akhtar *et al.*, 1993). While parameters such as sinus tachycardia and increased Q-T interval had normalized in many of the patients 24 hours after cooling, patients suffering from atrial fibrillation and paroxysmal supraventricular tachycardia required further treatment (Akhtar *et al.*, 1993).

Rautaharju *et al.* (1966) examined changes in ECG readings and cardiovascular function in a participant group that had prolonged exposure to a very high ambient temperature while sedentary. Following a period of rest in a room of 25˚C while heart rate and body temperature were stabilized, the nude participants were required to move into an environmental chamber and remain in a supine position. The period of exposure ranged from 27 to 28 minutes, with the air

temperature at the level of the participant varying from 76 to 78˚C, and the mean temperature at the site occupied by the participant varying from 83 to 88˚C (Rautaharju *et al.*, 1966). The authors noted common changes that would be observed on an ECG reading in a participant exposed to thermal stress, including shortening of the S-T segment, flattening of the T wave in lead X, and a lack of any obvious changes in the QRS complex (Rautaharju *et al.*, 1966). In the present study, results showed a significant decrease in the magnitude of the ventricular gradient (VG) during thermal stress, as well as large but inconsistent changes in its orientation; almost no change was observed in the magnitudes of the QRS area vectors (Rautaharju *et al*, 1966). In addition, HR increased from a mean of 66 bpm at rest to an average value of 109 bpm after 25 minutes of exposure to thermal stress, with a range of 96 to 141 bpm (Rautaharju *et al.*, 1966).

Chapter 3: Methodology

Participant Selection and Exclusion Criteria

Participants used in the study were student-athletes enrolled at Indiana State University, participating in one or more of the following NCAA Division I representative sports: track and field, cross country, and golf. From these demographics, a total of 11 participants (male n=5; female n=6) took part in the study. If potential participants met one or more of the following criteria, they were excluded from participating further in the study. Reasons for exclusion were as follows:

> 1) previously diagnosed or current cardiac abnormalities, 2) family history of cardiac abnormalities, 3) idiopathic syncope, 4) diabetes, 5) cancer, 6) medications that affect heart rate or blood pressure, 7) pituitary disease, 8) amenorrhea (amenorrheic for three or more months), 9) history of heat related illnesses, 10) thyroid disease, 11) sedentary, 12) pregnant or planning on becoming pregnant, 13) smokers (current or that have quit within the last six months), 14) hypertension, and 14) exercise induced asthma.

Pre-Test Procedure

Participants were required to sign and date an informed consent document before taking part in the study (APPENDIX A). In addition, participants were also required to complete a health history questionnaire which covered general health history, as well as more specific questions on personal and family cardiac health history (APPENDIX A). Participants were encouraged to eat a similar meal prior to starting any of the physical testing and ECG data

collection to ensure as much consistency as possible. Participant were also instructed to abstain from caffeine or alcohol for at least 24 hours prior to reporting to the laboratory for testing and were required to follow a hydration protocol to ensure they arrived at the laboratory adequately hydrated to perform the tests (APPENDIX A).

Furthermore, a daily health questionnaire was completed every day of data collection to ensure that the participant was feeling well prior to beginning the testing protocol (APPENDIX A).

Testing Procedure

Participants were required to report to the Exercise Physiology Laboratory on the campus of Indiana State University for testing on three separate occasions. The first visit consisted of having the participants read and sign the informed consent (during which time participants were encouraged to voice any questions or concerns they may have about the testing), answer the health-history questionnaire (APPENDIX A), and become familiar with the equipment used for data collection during treadmill tests. This familiarization process included a brief introduction to the treadmill where participants were exposed to a variety of speeds and gradients in order to help determine the initial starting speed for the formal treadmill tests. Following baseline data collection (age, height, weight, body fat, blood pressure, resting ECG), participants performed a maximal VO_2 test on the treadmill to allow for determination of the appropriate intensity for the submaximal tests in the subsequent two visits (Maximal Aerobic Test below)

Anthropometric Data Collection

Body fat data was collected using skinfold measurements from seven sites for both males and females (chest, abdominal, thigh, midaxillary, triceps, subscapular, and suprailiac) using the specific site locations outlined by the ACSM (Thompson, Gordon, & Pescatello, 2009). The

Jackson and Pollock equation was used to estimate body density. In order to calculate body fat percentage, the Siri equation of [(4.95/Db) – 4.5] x 100 was used for Caucasian and African American female participants, where Db = body density (g/cm^3). For African American male participants, ACSM guidelines that suggest that a more applicable formula of [(4.37/Db) – 3.93] x 100 should be used for those aged 18-32 was followed (Thompson *et al.*, 2009).

Maximal Aerobic Test

Prior to the beginning of the testing, participants had ECG readings recorded while they were standing, seated, supine, and hyperventilating (to simulate a condition of physical stress). The maximal test was performed on a treadmill and followed a modified Bruce/Olympic protocol (Paavolainen, Häkkinen, Hämäläinen, Nummela, & Rusko, 1999). The initial speed was determined by the participant. Participants were instructed to set the speed at a pace slightly higher than they were comfortable running at for an extended period of time (10-15 minutes). To ensure the speed was set solely based off comfort and feeling without any influence of the actual miles per hour figure, the speed indicator was covered on the treadmill and participants were instructed to adjust the speed until they felt it was at the appropriate level. The speed that was determined by the participants remained constant throughout the duration of the test. The first three minutes of the test were at a gradient of 0% but following this initial stage, the gradient increased by 1% every minute until the participant reached exhaustion. During the test, participants had respiratory and cardiovascular data recorded such as volume of O_2 and CO_2 (VO_2, $VCO_{2)}$, respiratory exchange ratio (RER), ventilatory exchange (V_E) and heart rate (HR). In addition, ratings of perceived exertion (RPE) were also recorded using the Borg (1998) scale (6-20) at the end of each three minute stage, as this aided in determining how close the participant was to reaching exhaustion. Following completion of the test, the gradient was

immediately returned back to 0%, treadmill speed reduced, and participants were required to remain jogging/walking on the treadmill until their heart rate returned to a normal level (within 20-30 bpm of recorded resting heart rate). Fluids were readily available at this time for participants to re-hydrate.

While the goal of the test was for participants to terminate the test where they felt at the point of maximal exertion, steps were in place in the event that the investigators were required to terminate the test before maximal exertion was achieved. In the event that it was necessary, any one of the following criteria outlined by the ACSM (Thompson *et al.*, 2009) would have facilitated one of the investigators to stop the test immediately:

- Onset of angina or angina-like symptoms
- Drop in systolic blood pressure (BP) of >10 mm Hg from baseline BP despite an increase in workload
- Excessive rise in BP: systolic pressure >250 mm Hg or diastolic pressure >115 mm Hg
- Shortness of breath, wheezing, leg cramps, or claudication
- Signs of poor perfusion: light-headedness, confusion, ataxia, pallor, cyanosis, nausea, or cold and clammy skin
- Failure of heart rate to increase with increased exercise intensity
- Noticeable change in heart rhythm
- Participant requests to stop
- Physical or verbal manifestations of severe fatigue
- Failure of the testing equipment

The test was performed in a thermo-regulated laboratory at a temperature of 24.8 ± 1.6˚C (23.3 – 27.8˚C).

Submaximal Test at Ambient Temperature

Baseline data recorded during the maximal treadmill test was used to determine where the submaximal threshold was set and maintained by the participants. The threshold was set at 70% of the participant's maximal oxygen uptake (VO_2 max) and participants were asked to maintain this workload for anywhere up to 30 minutes. Prior to the beginning of testing, participants had resting ECG readings recorded while standing, seated, and supine. As with the maximal test, where the test finished was entirely the decision of the participant, but participants were encouraged to run for as long as possible within the time limit. Respiratory data was collected until the investigators observed the 70% of maximum VO_2 threshold, at which time the neoprene mask was removed and the participant continued running until voluntary termination or the 30 minute time limit was reached. During the test, participants were continuously monitored through a 12-lead ECG. There were momentary pauses (30-40 seconds) in the test at five minute intervals to better examine the ECG data and minimize interference caused by the running movement which would have increased the difficulty of interpreting the ECG readings. At five minute intervals, participants were required to straddle the treadmill belt and remain still while the ECG printed a reading. Once a reading was achieved with minimal or no interference, participants lowered themselves back onto the belt and continued running. Before stepping back onto the treadmill belt, the speed and gradient was lowered to ensure participant safety. Once they had gained balance on the belt, the speed and gradient were increased to their previous levels. The submaximal testing also followed the same initial protocol as used in the maximal test with regard to the starting treadmill speed and gradient of 0%, however, the gradient was only increased to a point where the participant was at a workload of 70% of their VO_2 max. Following completion of the test, participants were required to complete a jogging/walking cool

down on the treadmill in the same manner as the maximal test (until heart rate returned to 20-30 bpm or recorded resting rate). Fluids were available ad libitum to participants while performing the test and cool down. The test was performed in a thermo-regulated laboratory at a temperature of 24.8 ± 1.6˚C (23.3 – 27.8˚C).

Submaximal Test in Hyperthermic Environment

As an additional stressor to the running workload, participants took part in a second submaximal test while placed in an environmental chamber, set at a temperature of 38˚C (100.4˚F) where they were required to perform a submaximal treadmill test following the same protocol as the previous submaximal test and using the same termination point as the ambient temperature submaximal test (70% of VO_2 max).

Water and/or an electrolyte replacement beverage (Powerade/Gatorade) was available to the participants to consume ad libitum during the test. The hydration protocol was required to be followed the night prior to the test, and participants were also instructed to consume regular fluids (excluding caffeine and alcohol) during the hours leading up to the test to aid in maintain hydration levels. During the test, participants were monitored through a 12-lead ECG.

Analysis of ECG Testing

ECG Classifications.

Analysis of ECGs will be defined based on adapted guidelines proposed by Pelliccia *et al.*, 2000):

Distinctly Abnormal ECG.

These ECGs would be strongly suggestive of cardiovascular disease. Criteria for this diagnosis includes: (1) striking increase in R or S wave voltage (≥35 mm) in any lead, (2) Q waves ≥4 mm in depth and present in

≥2 leads, (3) repolarization pattern with inverted T wave >2 mm in ≥2 leads, (4) left bundle branch block, (5) marked left (≤-30°) or right (≥110°) QRS axis deviation, and (6) Wolff-Parkinson-White pattern.

Mildly Abnormal ECG.

These ECGs would be compatible with the presence of cardiovascular disease. Criteria for this diagnosis includes: (1) increased R or S wave voltage (30 to 34 mm) in any lead; (2) Q waves 2 to 3 mm in depth and present in ≥2 leads; (3) repolarization patterns with either flat, minimally inverted, or particularly tall (i.e, ≥15 mm) T waves in ≥2 leads; (4) abnormal R wave progression in the anterior precordial leads; (5) right bundle branch block (RSR[1] pattern ≥0.12 s in V_1 and V_2); (6) right atrial enlargement (peaked P waves ≥2.5 mm in leads II, III, or V_1); (7) left atrial enlargement (prolonged positive P wave in lead II and/or deep, prolonged negative P wave in V_1); and (8) short PR interval (≤0.12 s).

Normal ECG or ECG with Minor Alterations.

These ECGs would either be completely normal or showed minor alterations that could be attributed to athlete's heart syndrome. Criteria for this diagnosis includes: (1) increased PR interval duration (>0.20 s), (2) mild increase in R or S wave voltage (25 to 29 mm), (3) early repolarization (ST elevation ≥2 mm in >2 leads), (4) incomplete right bundle branch block (RSR[1] pattern in V_1 and V_2 of <0.12 s in duration), and (5) sinus bradycardia <60 bpm.

Statistical Analysis

The baseline characteristics of the groups were analyzed using One-way analysis of variance (ANOVA). A 2 x 4 repeated measures ANOVA was used to test the effects of condition (thermoneutral, hyperthermic) by time (baseline, 5 minutes, 10 minutes, 15 minutes) on the changes in R and S wave voltage, PR interval duration, and ST elevation/depression. The analysis of ECG abnormalities was performed by coding the ECG based on adapted criteria from Pelliccia *et al.* (2000). If an interaction was deemed significant, a Tukey's HSD test was used for post-hoc comparisons. Significance was set *a priori* at $p<0.05$. Values are presented as mean ± SEM. All statistical analyses were performed using SPSS Version 18 (SPSS, Inc., Chicago, IL, USA).

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APPENDIX A: Relevant Study Forms

Participant ID
Age
Gender
Height
Weight
Resting BP

Skinfold sites:		
thigh		
suprailiac		
triceps		
abdominal		
infraspinatus		
chest		
axilla		

sum of all seven:

Body fat on skinfolds:

Waist cirmcumference
Hip circumference

Submax Test (hyperthermic)

ID#_____ Date_____

Body Weight: Pre _____Post_____

Time stage ends	Speed	HR	BP	RPE	VO_2 (liters)	VCO_2 (liters)	V_E (liters)	RER
Rest								
3:00								
6:00								
9:00								
12:00								
15:00								
18:00								
21:00								
24:00								
27:00								
30:00								

Max/Submax Test (ambient temp)

ID#_____ Date_____

Body weight: Pre _____Post_____

Time stage ends	Speed	HR	BP	RPE	VO_2 (liters)	VCO_2 (liters)	V_E (liters)	RER
Rest								
3:00								
6:00								
9:00								
12:00								
15:00								
18:00								
21:00								
24:00								
27:00								
30:00								

Indiana State University
CURRENT HEALTH QUESTIONNAIRE

Participant ID: **Date:**

1.	Have you experienced any vomiting or diarrhea in the last 48 hours?	Yes	No
2.	Have you consumed an excessive amount of alcohol in the last 48 hours?	Yes	No
3.	Have you had the cold or the flu within the last week?	Yes	No
4.	Have you had a gastrointestinal illness within the last week?	Yes	No
5.	Have you had any other general illness the investigator should know about in the last week?	Yes	No
6.	Have you followed the hydration protocol given to you?	Yes	No
7.	Have you eaten before arriving at the laboratory? *If "Yes", please give a brief description of what you ate below.*	Yes	No
8.	Do you feel dizzy or "unwell" in other ways that the primary investigators should be aware of?	Yes	No

** If you answered yes to some of the questions above, the investigator may ask to delay you participation. Data collection will be delayed until all symptoms have resolved to ensure your safety during the study.

Indiana State University
HEALTH HISTORY QUESTIONNAIRE

Study Title: Prevalence of Cardiac Abnormalities in Male and Female College Athletes When Exposed to Physiological and Thermal Stressors.

Participant ID: Date:

Please answer the following questions to the best of your knowledge:

1.	Are you currently an NCAA athlete involved in one or more of the following representative sports: football, basketball, track and field, softball, cross country, soccer, golf, volleyball, and baseball.	Yes	No
2.	Are you currently under a doctor's care?	Yes	No
3.	**Women only:** Are you pregnant or are you planning on becoming pregnant?	Yes	No
4.	**Women only:** Are you currently amenorrheac (have not had a period for three or more months) for reasons other than birth control?	Yes	No
5.	Do you have a pacemaker or automatic implanted cardiac defibrillator (AICD)?	Yes	No
6.	Do you have, or suspect that you have, any circulatory problems or vascular (problems with your veins or arteries) disorders, conditions, disorders, or diseases?	Yes	No
7.	Do you have, or suspect that you have, any rheumatoid (joint) or muscular conditions, disorders or diseases?	Yes	No
8.	Has your doctor ever told you that you may have a heart valve problem?	Yes	No
9.	Do you experience numbness, tingling, or decreased sensation in extremities, or have other neurological problems, conditions, disorders, or diseases?	Yes	No
10.	Do you have any problems, conditions, disorders or diseases that affect your ability to keep your balance?	Yes	No
11.	Are you currently taking any prescription medications? *If YES, please list all prescription medications.*	Yes	No
12.	Are you taking any over-the-counter medications or supplements? *If YES, please list below.*	Yes	No
13.	Have you suffered from a lower extremity injury in the past 6 months?	Yes	No
14.	Has your doctor ever told you that you have heart disease?	Yes	No
15.	Have you ever had a heart attack?	Yes	No
16.	Have you ever had a stroke?	Yes	No
17.	Have you ever had chest pain when exercising?	Yes	No
18.	Has your doctor ever told you that you have a heart murmur?	Yes	No
19.	Does anyone in your immediate family have any important cardiac conditions? (e.g. Marfan syndrome, long-QT syndrome)	Yes	No
20.	Has your doctor ever told you that you have high blood pressure?	Yes	No

21.	Have you had a heart aneurysm?	Yes	No
22.	Have you ever had thyroid disease?	Yes	No
23.	Are you currently a smoker or have quit within the last six months?	Yes	No
24.	Do you suffer from exercise induced asthma?	Yes	No
25.	Have you ever fainted for any unknown reasons?	Yes	No
26.	Have you experienced any heat illnesses such as heat exhaustion or heat stroke?	Yes	No
27.	Have you been told that you are a diabetic?	Yes	No
28	Have you been told that you have cancer?	Yes	No
29.	Are there any other medical problems (past or present) not already mentioned that we should know about? If YES, please explain below:	Yes	No

Participant Informed Consent

Prevalence of Cardiac Abnormalities in Male and Female College Athletes When Exposed to Physiological and Thermal Stressors

You have been asked to take part in a research project that looks at the effect of treadmill running in a normal temperature and in a heated room on how your heart functions. Matthew A Tucker, a graduate student at Indiana State University, is the primary investigator on this study while Drs. Derek Kingsley, and Susan Yeargin are co-investigators. Your participation in this study is entirely voluntary and you are able to stop your involvement at any time. Please read the information below with the investigator and ask questions at anytime about anything you do not understand, before deciding whether or not to take part.

Purpose of the Study.

The purpose of this study is to look at how treadmill running in different environments (normal temperature, 73-75˚F and hot temperature, 100.4˚F) affects your heart and how it functions. This will allow us to look for any unusual activity that might be going on with your heart. By doing this, we can hope to gain a better understanding of how common non-life threatening heart disorders in a safe environment are in college-aged athletes; giving us data that may one day help in having more thorough screening processes for college athletes before they are allowed to participate in sports.

Procedures.

The investigators Matt Tucker and Dr. Derek Kingsley have explained the requirements and procedures for the four laboratory visits and the time commitment that this will involve. I understand and accept the requirements of participating in these tests.

Checklist:	Initial	
Health history questionnaire	☐	
Order of testing	☐	
Metabolic cart	☐	
Environmental chamber	☐	
ECG machine and electrodes	☐	
Private lockable bathroom	☐	
Rectal thermometer emergency protocol	☐	
Anthropometric data collection equipment	☐	
Treadmill – participant can safely maneuver themselves off and back onto the treadmill (if 'No', repeat until satisfactory).	☐ Yes	☐ No

_____ Date

Potential Risks and Discomforts.

There are possible risks involved with this study when performing maximal or near maximal tests. The American College of Sports Medicine (ACSM) lists criteria for stopping a test early as meeting one or more of the following conditions: onset of angina or angina-like symptoms, drop in systolic blood pressure (BP) of >10 mm Hg from baseline BP despite an increase in workload, excessive rise in BP, shortness of breath, wheezing, leg cramps, or calf cramping, signs of poor perfusion: light-headedness, confusion, ataxia (inability to walk), pallor (white, ashen skin), cyanosis (blue fingernail beds or lips), nausea, or cold and clammy skin, failure of heart rate to increase with increased exercise intensity, noticeable change in heart rhythm, and failure of the testing

equipment. In addition, there is also the risk of a cardiac abnormality occurring such as an arrhythmia (an abnormal heart rhythm); however, the chances of a serious abnormality occurring will be minimized through pre-screening questionnaires before you take part in any testing and during testing, investigators will be monitoring your ECG which will show us if there is any abnormality occurring which would require the test to stop.

Because the tests will be completed on a treadmill, there is the risk of possibly falling off while the treadmill is in motion. At all times during testing, the treadmill will be surrounded by padding, and one of the investigators will act as a spotter so that in the event that you do fall off, you will be well protected and the risk for injury is minimized. There will be an ice bath ready when you take part in the submaximal test in the heat so that we can cool your core body temperature quickly if it gets too high during the test. It's not likely that this will be needed, but it will be there as a precaution. Secondly, any testing that involves maximal effort or combines exercise with a heated environment has some level of risk; however, there are steps in place to make sure you are safe while taking part in this study, such as keeping a close focus on your data and physical condition so that we are ready to step in and stop the test if need be. In addition, we will ensure that you meet the ACSM's criteria to be considered low risk before any testing takes place.

Potential Benefits to Participants and/or Society.

There are no direct benefits to participants or society, only inferred.

Compensation to Participate.

There will be no compensation for participating in this study.

Confidentiality.

Any information that is obtained during this study that can identify you will remain confidential and will be revealed only with your permission, as required by law. Your health history questionnaire and the informed consent will be kept in separate locked cabinets in secure rooms after the investigators, Matt Tucker and Dr. Derek Kingsley, review it for safety reasons. To make sure your identity is kept confidential, an ID number will be given to the data that we collect from your testing. The master sheet with your ID number and name will be kept in a locked cabinet during the study that only the investigators will have access to. It will be stored in a separate location from the questionnaires and collected data so that your confidentiality can be maintained. All collected data from testing and informed consent documents will be stored for three years after completion and will be destroyed (shredded) at the end of this time. The master key that identifies you from the ID number given to you will be destroyed immediately following completion of testing. Health history questionnaires and current health questionnaires will be stored in a locked cabinet in Dr. Kingsley's office, separate from the collected data and informed consent documents, for seven years and will be destroyed (shredded) at the end of this time. The results of this study will be published in a summary format and there will be no way for anyone to identify that you participated in this study.

Participation and Withdrawal.

You can choose whether or not to be in this study if you are aged between 18 and 30 years old. If you volunteer to be in this study, you may withdraw at any time without penalty or consequences, or loss of benefits to which you are otherwise entitled. In the

unlikely event that physical injury results from your participation in this study, you will be provided with on-site emergency medical treatment at no cost to you.

Costs of Participation.

In the event that medical attention is required past that which can be provided by the investigators in the Exercise Physiology laboratory, the cost of additional medical treatment (e.g., student health center, hospital emergency room) will be your responsibility and financial compensation is not available from Indiana State University or the investigators.

Identification of Investigators.

If you have any questions or concerns about participating in this research project, please contact Matt Tucker or Dr. Derek Kingsley.

Primary Investigator: Matt Tucker

Arena C-54

Graduate Assistant

Indiana State University

(812) 243-5814

Email: mtucker15@indstate.edu

Faculty Sponsor/Co-Investigators:

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Terre Haute, IN 47809
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Rights of Research Subjects.

If you have any questions about your rights as a research subject, you may contact the Indiana State University Institutional Review Board (IRB) by mail at 114 Erickson Hall, Terre Haute, IN 47809, by phone at (812) 237-8217, or e-mail the IRB at irb@indstate.edu. You will be given the opportunity to discuss any questions about your rights as a research subject with a member of the IRB. The IRB is an independent committee composed of members of the University community, as well as lay members of the community not connected with ISU. The IRB has reviewed and approved this study. I understand the procedures described above. My questions have been answered to my satisfaction, and I agree to participate in this study.

_____

Printed Name of Subject

_____ _____

Signature of Subject Date

Pre-test Hydration Protocol

The following instructions will be given to participants regarding their fluid intake prior to arriving at the Exercise Physiology Laboratory for testing:

The following protocol **MUST** be followed the night before reporting to the Exercise Physiology Lab, located in the Arena Building on the Indiana State University Campus.

Failure to report hydrated will result in **delay** when your next test can begin.

Approximately **4 hours** before going to bed drink **600 ml (20 oz)** of water, sports drink, or flavored water. Avoid caffeinated beverages and alcohol.

Immediately before going to bed drink an additional **600 ml (20 oz)** of water, sports drink, or flavored water. Avoid caffeinated beverages and alcohol.

Below you will find a table for you to write your scheduled data collection days to help you keep track of your commitment.

Schedule

Date	Time Report	Est. Time Completed	Hydration

APPENDIX B: Analyses

PR Interval ANOVA Table

Mauchly's Test of Sphericity[b]

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon[a]		
					Greenhouse-Geisser	Huynh-Feldt	Lower-bound
cond	1.000	.000	0	.	1.000	1.000	1.000
time	.402	6.123	5	.299	.655	.867	.333
cond * time	.287	8.397	5	.139	.654	.865	.333

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

b. Design: Intercept + Group

Within Subjects Design: cond + time + cond * time

Tests of Within-Subjects Effects.

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
cond	Sphericity Assumed	117.556	1	117.556	.557	.477	.065	.557	.101
	Huynh-Feldt	117.556	1.000	117.556	.557	.477	.065	.557	.101
time	Sphericity Assumed	11480.500	3	3826.833	31.343	.000	.797	94.030	1.000
	Huynh-Feldt	11480.500	2.601	4413.160	31.343	.000	.797	81.537	1.000
cond * time	Sphericity Assumed	208.111	3	69.370	.870	.470	.098	2.611	.211
	Huynh-Feldt	208.111	2.595	80.210	.870	.459	.098	2.258	.196

a. Computed using alpha = .05

R Wave ANOVA Table

Mauchly's Test of Sphericity[b]

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon[a]		
					Greenhouse-Geisser	Huynh-Feldt	Lower-bound
cond	1.000	.000	0	.	1.000	1.000	1.000
time	.438	5.543	5	.358	.693	.940	.333
cond * time	.421	5.814	5	.329	.698	.951	.333

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

b. Design: Intercept + Group
Within Subjects Design: cond + time + cond * time

Tests of Within-Subjects Effects.

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
cond	Sphericity Assumed	9.389	1	9.389	4.250	.073	.347	4.250	.442
	Huynh-Feldt	9.389	1.000	9.389	4.250	.073	.347	4.250	.442
time	Sphericity Assumed	130.681	3	43.560	10.613	.000	.570	31.839	.996
	Huynh-Feldt	130.681	2.821	46.317	10.613	.000	.570	29.943	.994
cond * time	Sphericity Assumed	5.750	3	1.917	.861	.475	.097	2.582	.209
	Huynh-Feldt	5.750	2.852	2.016	.861	.471	.097	2.455	.203

a. Computed using alpha = .05

S Wave ANOVA Table

Mauchly's Test of Sphericity[b]

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon[a]		
					Greenhouse-Geisser	Huynh-Feldt	Lower-bound
cond	1.000	.000	0	.	1.000	1.000	1.000
time	.340	7.243	5	.207	.650	.856	.333
cond * time	.141	13.158	5	.023	.562	.696	.333

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

b. Design: Intercept + Group
Within Subjects Design: cond + time + cond * time

Tests of Within-Subjects Effects.

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
cond	Sphericity Assumed	.056	1	.056	.049	.830	.006	.049	.054
	Huynh-Feldt	.056	1.000	.056	.049	.830	.006	.049	.054
time	Sphericity Assumed	18.056	3	6.019	9.706	.000	.548	29.118	.993
	Huynh-Feldt	18.056	2.568	7.032	9.706	.001	.548	24.923	.984
cond * time	Sphericity Assumed	.278	3	.093	.607	.617	.071	1.822	.157
	Huynh-Feldt	.278	2.089	.133	.607	.563	.071	1.269	.136

a. Computed using alpha = .05

ST Segment ANOVA Table

Mauchly's Test of Sphericity[b]

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon[a]		
					Greenhouse-Geisser	Huynh-Feldt	Lower-bound
cond	1.000	.000	0	.	1.000	1.000	1.000
time	.119	14.313	5	.015	.491	.574	.333
cond * time	.198	10.898	5	.056	.543	.662	.333

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

b. Design: Intercept + Group
Within Subjects Design: cond + time + cond * time

Tests of Within-Subjects Effects.

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
cond	Sphericity Assumed	.347	1	.347	3.883	.084	.327	3.883	.411
	Huynh-Feldt	.347	1.000	.347	3.883	.084	.327	3.883	.411
time	Sphericity Assumed	1.681	3	.560	2.620	.074	.247	7.859	.568
	Huynh-Feldt	1.681	1.723	.975	2.620	.114	.247	4.514	.408
cond * time	Sphericity Assumed	.403	3	.134	1.157	.347	.126	3.471	.271
	Huynh-Feldt	.403	1.987	.203	1.157	.339	.126	2.299	.217

a. Computed using alpha = .05

APPENDIX C: Sample Electrocardiograms

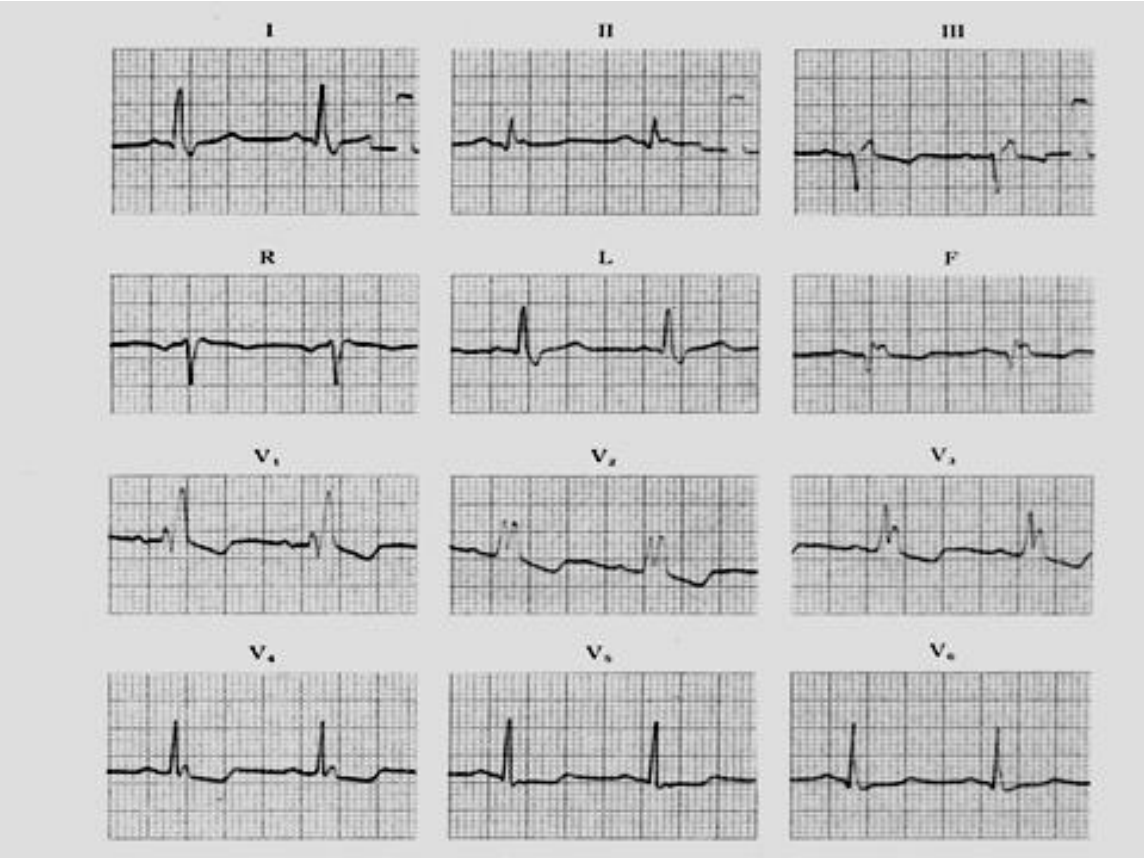

Figure 6. Right bundle branch block.

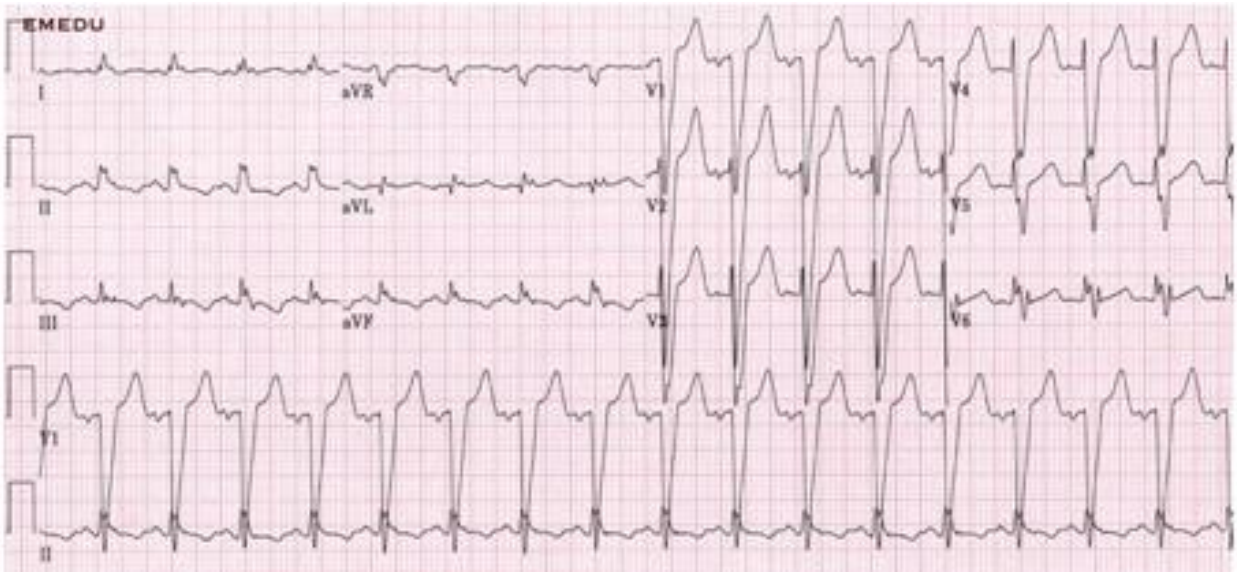

Figure 7. Left ventricular hypertrophy and left bundle branch block.

APPENDIX D: Recommendations

Methodology Improvements

In order to improve upon the present study, several factors could be altered to provide more robust and conclusive results. Firstly, a larger study population would increase the statistical power, therefore increasing the robustness of the data. More specifically, participants from a wider range of sports could be used, including athletes participating in more anaerobic/power sports (i.e. volleyball, basketball, softball, baseball, football, etc.) to test for differences that exist in terms of prevalence of cardiac abnormalities among sports. In addition, athletes from different ethnic backgrounds could be tested, as previous research has demonstrated a considerably higher incidence rate of cardiac abnormalities in African American athletes; particularly males.

Clinical Implications

While the present study demonstrated a high prevalence of an uncommon ECG abnormality in athletes (most notably incomplete left bundle branch block), given the relatively low study population, it should be noted that our results are limited in their ability to advance the current support for improved pre-screening protocols. The ultimate goal in this line of research is to lower the incidence rate of SCD in athletes; however, one should not expect current collegiate pre-screening protocols to move from considerably inadequate to adequate in one swift motion, rather, that the steps currently in place are improved upon, beginning with the most simplistic factor of the pre-screening process: the health history questionnaire. While many athletes will

show almost nothing of concern on their pre-screening questionnaire, if something abnormal were to show, further investigation by way of a resting ECG may be warranted. Therefore, it is the hopes of the authors that this research will help contribute to the eventual inclusion of a resting ECG in pre-screening protocols for collegiate athletes, ultimately leading to a decrease in incidences of SCD in this population. The ongoing success of the Italian pre-screening model is something that should be both commended and embraced as a vision of the future for US collegiate athletic programs.