THE EFFECTS OF CAFFEINE INTAKE ON THE ELECTROPHYSIOLOGICAL PROPERTIES OF THE HEART AMONG COLLEGIATE ATHLETES

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KeyWords
caffeine, collegiate athletes, energy drink, electrocardiogram, sudden cardiac death

ABSTRACT

This study investigates if the changes brought about by caffeine on the electrophysiologic activity of the heart and if it will contribute to the risk factor of sudden cardiac death.

Introduction and rationale

An athlete is a trained individual or group of people who competes in a sport and an athlete's goal is to optimize his capacity to perform his sport. To do so they undergo training to the point of exhaustion and often resort to improve their training capacity by ingesting supplements and energy drinks that has a wide range of ergogenic effects. These will affect the endurance and power of the athlete, as what most journals would assess [1,2,3].Most of these products have an active ingredient which is caffeine. Caffeine can be found in food and energy drink making it widely available to most people.

Caffeine has several physiologic effects on the body, not only in the central nervous system but it's most evident effect is on the cardiovascular system where it inhibits adenosine receptors. This inhibition may cause vasoconstriction that leads to an increase in both blood pressure and heart rate. Caffeine can be found mostly in food.External factors can contribute as to how it is processed in the body. Another factor to be considered is genetics since most studies have mentioned it's correlation with caffeine ingestion. However, this field of study is still under development and still needs to be researched more specially in human subjects.The most common side effects of caffeine are palpitations, anxiety, tremors and nervousness. If taken in large amounts it can lead to cardiac arrest.

Athlete's do train to the point of exhaustion and it may sometimes have negative effects. Death via caffeine overdose has been observed in athletes in some studies [4,5,6] however, most studies failed to mention if the death occurred during exertion. The safe dosage applicable for caffeine consumption in trained and untrained individuals still needs further studies. In a study by Goldstein et al it has been concluded that only ~3-6mg/kg of caffeine can enhance a trained athletes sports performance and consuming more of this low-to-moderate dose will no longer be effective in enhancing sports performance in trained athletes[7]. To support this, several studies have been done regarding the dosing of caffeine pre workout to determine which exact range of caffeine dosing will exhibit its ergogenic effect [8,9,10].

Early prevention can be done to stop irreversible damage from happening in athletes. Screening using a 12 lead electrocardiogram (ECG), a non invasive machine, can have a drastic help to decrease Sudden Cardiac Death (SCD) in athletes[10] and it has been proven to increase the percentage of disqualification of athletes from competitions due to cardiomyopathies such as arrhythmogenic right-ventricular cardiomyopathy. Abnormal findings have been observed in athletes prior to competition and it helped decrease risk factors contributing to SCD. An ECG machine is a noninvasive tool used to diagnose cardiovascular diseases and can also give health care providers insight as to how severe cardiovascular disease is.

In an ECG an electromagnetic force or current that has both magnitude and direction is the main principle as to how the machine translates these findings from the body with the use of electrodes to a piece of special paper for the results to be recorded[10]. However, it's use is not included in the preparticipation screening done in athletes for it is expensive, not everyone has access to it, not a lot of healthcare professionals specialize in the interpretation of the results making mass interpretation less reliable. What is routinely used in the preparticipation program, as suggested by the AHA is a thorough physical exam, family history wherein questionnaires can be given to the parents, and personal history[21].

Based on Soriano E.D. which was conducted in 2 high schools in Metro Manila, Philippines with 61 athletes aged 13-19 years old 10% of the athletes enrolled in her study had ECG abnormalities which required referral to a specialist and they were generally asymptomatic[21]. This study showed that the most common abnormality was T- wave inversion in the right precordial leads which was present in 69% of the athletes and the other abnormal findings were complete RBB (Right Bundle Branch Block), prolonged QT and biventricular hypertrophy and left atrial enlargement.

LITERATURE REVIEW

Regular exercise can be beneficial to cardiovascular health in so many ways. Athletes who train regularly and are well adapted to exercising have demonstrated lower resting heart rates and cardiac hypertrophy[11] making them less vulnerable to cardiovascular diseases. However, signs and symptoms of cardiovascular disease may manifest as shortness of breath and syncope which may also be perceived as a normal response to exercise. Athletes are perceived as healthy individuals thus making SCD in their population a controversial subject.

World Health Organization set guidelines to exercise wherein adults aged 18–64 can perform at least 150 minutes of moderate-intensity aerobic physical activity throughout the week, or perform at least 75 minutes of vigorous-intensity aerobic physical activity throughout the week or an equal combination of moderate- and vigorous-intensity exercise. Athletes on the other hand, especially competitive ones do not match up to these guidelines and train at a higher intensity with much longer duration and frequencies.

Moreover, this study defines exercise as a subset of scheduled, organized and repetitive physical activity with the ultimate or intermediate goal of improving or maintaining physical fitness.[26] This should be differentiated from the normal physical activity that is not planned and organized, exercise and training in these study is the set of planned activities that has the goal of maintaining and improving physical fitness.

For this research we define an athlete as someone that takes part in an organized team or individual sport involving competition from others as a central component, puts a high premium on success and accomplishment, and needs a sort of regular (and typically intense) preparation, [45] this definition is much easier to apply to athletes in high school, college and professional sport organizations.

Athletes who train regularly and are well adapted to exercising have demonstrated lower resting heart rates and cardiac hypertrophy making them less vulnerable to cardiovascular diseases.[11] This cardiovascular adaptation to the rigorous training that is due to the compensation in exertion has intrigued scientists. Furthermore, cardiac enlargement is a type of overuse pathology for athletes and that excessive involvement in sport may lead to the collapse of the cardiovascular system. [28] Moreover, this remodeling, commonly referred to as ' the athlete's heart, maycorrelate with several abnormal individuals, in which the first clinical presentation may be sudden cardiac death. [20]

It is now well known that repeated involvement in intense physical exercise results in important changes in the structure and function of the myocardial. [27] During exercise, the heart is subjected to intermittent hemodynamic stresses of pressure overload, volume overload, or both.To normalize such stress and to meet the systemic demand for an increased blood supply, the heart undergoes morphological adaptation to recurrent exercise by increasing its mass, primarily through an increase in ventricular chamber wall thickness.[11] It contrasts with pathological remodeling due to chronic stress exhaustion, which can result in a loss of contractile control and heart failure. [28] During pathological remodeling of the heart, electrical instability can result from a lack of upregulation of main cardiac ion channel subunits associated with potential repolarization action relative to an increase in myocyte size. [11]In contrast, increased myocyte size in physiological hypertrophy is associated with the upregulation of depolarizing and repolarizing currents, which may be protective against abnormal electrical signaling in the adapted heart. [28]

This is more expounded wherein the pathological remodeling of the heart is associated with a reduction in the production of oxidative energy via fatty acid oxidation and increased reliance on glucose use, biogenesis of mitochondria and fatty acid oxidation capacity after exercise. [29] Changes in myocardial glycolytic activity during acute exercise and the subsequent recovery period may also play a significant role in controlling metabolic gene expression and cardiac remodeling. [11] Possibly upstream of these metabolic changes, studies have also identified a dominant role in protein synthesis and hypertrophy associated with IGF-1 and insulin receptor signaling via the PI3K / Akt1 pathway. [30] Untargeted approaches have established other major determinants of transcriptional programs which drive the hypertrophic response induced by the exercise. [11] In addition to metabolic and molecular remodeling, exercise can also facilitate adaptive cardiac adaptation, which can eventually improve cardiac output and reduce the risk of arrhythmia [28]

Additionally, a recent study shows that people who have completed at least 25 marathons over a 25-year duration have higher than expected rates of coronary artery calcification (CAC) and calcified coronary plaque thickness relative to sedentary individuals.[31] It is also shown that individuals with very high levels of physical activity (~3 times recommended levels) have higher chances of developing CAC, particularly in white males. [32] The shape of the dose-response curve remains obscure with other effects of exercise, and it is not clear at what levels of intensity and duration the effects of exercise begin to taper and where they begin to become detrimental.It is also unknown how personal demographic features such as age, sex, ethnicity and baseline CVD risk affect this threshold of transition from benefit to harm. [11]

Caffeine Is a stimulant of the central nervous system that is consumed routinely by a large number of people, usually in the form of coffee or tea. Caffeine raises alertness and decreases tiredness,this can be particularly important in situations of low arousal; Caffeine also improves performance on tasks of caution and simple tasks involving continuous response.On the other hand, effects on more complex tasks are hard to assess and likely involve associations between caffeine and other factors that improve alertness. [34] However, this xanthine alkaloid is profoundly toxic if consumed in high doses and can result in death. [5]

About arrhythmia in cases of caffeine intoxication, ventricular fibrillation is most frequently found to be the cause of death, whereas the most frequently cited arrhythmia pathways involve increased levels of catecholamine, phosphodiesterase inhibition, decreased intracellular calcium and antagonism of antiarrhythmic adenosine receptors. [6] Moreover, coronary vasospasm has been suggested via the adenosine antagonism of caffeine and the release of catecholamine, which can increase the vascular smooth muscle contraction triggering vasoconstriction is the traceable cause for myocardial infarction. [36]

Studies are also pointing into these anhydrous form of caffeine in concentrated caffeine powders wherein 1 tsp of powder may equal to 25 cups of coffee as the cause for death due to cardiac arrhythmias and caffeine overdose seizures. [5] Furthermore, these forms of caffeine are said to be effective in eliciting ergogenic effects in highly trained athletes. [7]

Caffeine (1,3,7-trimethylxanthine) is metabolized by the liver and results in three metabolites through enzymatic action: paraxanthine, theophylline, and theobromine.In the bloodstream, elevated levels may appear within 15-45 min of consumption, and peak concentrations are evident one hour after ingestion. [32] Research studies including the intake of caffeine and physical performance indicate a combined effect on both the main and peripheral systems. Therefore, caffeine may serve as an adenosine antagonist on the central nervous system, but may also influence the metabolism of the substrates and neuromuscular function.[7] Specifically for strength performance, possible mechanisms include increased muscle activation, recruitment of motor units, and enhanced excitation contraction coupling. [3]

Presently, some coaches have developed the use of caffeine as adjunct to athletes training to improve their performance more. Caffeine is the most commonly available psychostimulant in the world. It is estimated that more than 80 percent of caffeine is absorbed.[6] Both recreational and elite athletes have been reported to use caffeine in their training. It is obvious that caffeine supplementation offers an ergogenic reaction to continuous aerobic activities in moderate-to-highly trained endurance athletes.[7] Although, evidence of the potential of caffeine to boost aerobic performance is well known but evidence of an ergogenic contribution to muscle endurance and strength-based activities is minimal. In addition, the limited evidence for the ergogenic benefit of caffeine in muscle endurance and strength is equivocal. [3]

That being said, there is varying evidence on what dosing of caffeine to give athletes to be able to elicit ergogenic response during their training or sport performance. Caffeine is considered to have typically dose-dependent effects with beneficial or favorable effects at lower doses (i.e. ≤ 400 mg) and adverse effects usually above this amount of ingestion, although there is significant inter-individual variation. [34] Increased arousal, alertness, attention and well-being were observed in human subjects at doses of 250 mg,

while a dose of 500 mg was shown to increase tension, nervousness, anxiety, anticipation, irritability, nausea, paresthesia, tremor, suddenness, palpitations, restlessness and likely dizziness. [6]

The effects of varying quantities of caffeine on metabolism and aerobic exercise and significantly increased production of low (3 mg / kg) and moderate (6 mg / kg) caffeine but not 9 mg / kg have been recorded. [7] Based on some subjects' subjective reports, caffeine may have overstimulated the central nervous system at this high dose to the point where the usually positive ergogenic responses were overridden.[35] Moreover, Compared to a cup of caffeinated coffee, the use of caffeine in anhydrous form appears to be of greater benefit for the purpose of improving endurance performance. Furthermore, a low to moderate dose of caffeine between 3 and 6 mg / kg appears to be sufficient to improve performance with maximum sustained endurance effort. [7]

As athletes train their way to achieve their goals in the specific sport they enter, most of them consume caffeine in any form such as supplement tablets or energy drinks to help delay fatigue[12][1 2]. However, it also has its negative effects such as anxiety and palpitations as a mild side effect and arrhythmias as one of its severe side effects[13]but an agency regulates its use in athletes during competition. According to the World Anti-Doping Agency, caffeine is not included in the list of prohibited substances however it is monitored and listed under stimulants in the 2020 Monitoring Program during competition[14]. Caffeine is part of the stimulants to be monitored in competition or during the competition, hence, the practice of coaches and athletes to drink it out of the competition is out of scope for the anti-doping agency also means this aspect of the study of these is very substantial to this aspect of sports management and rehabilitative training.

Caffeine has been proven to be an effective ergogenic boost for prolonged high aerobic exercise, and has also been shown to be very effective in improving performance in time trials. It also enhances glycogen resynthesis during
the recovery phase of exercise. Moreover, is beneficial for prolonged duration high-intensity exercise (including team sports such as soccer, field hockey, rowing, etc.) but performance enhancement is specific to conditioned athletes. [7] That being said, more research should be done in expounding on the doses, type of caffeine, and timing of the ingestion of caffeine to produce the desired effects for increase in exercise capacity and regimen.

The World Anti-Doping Agency (WADA) is a foundation advocating anti-doping in sports and was initiated by the International Olympic Committee. This foundation contributed to the sports community in many ways by consistently regulating the policies, rules, and regulations of anti-doping worldwide. One of the most highlighted contributions by this foundation in this research is its World Anti-Doping Code, list of prohibited drugs, and list of regulated drugs. The lists are published annually and is one of the six international standards [22].

The definition of athlete varies making it difficult to distinguish those who are younger and older because an athlete may be someone who competes in a sport or who plays recreational sport[15]. Estimating the number of SCD in athletes may be challenging because of several factors such as age, where athletes younger than 35 years old is at risk for SCD if they are found to have hypertrophic cardiomyopathy or congenital anomalies in their coronary artery[15]. In athletes older than 35 years old, they are at risk for SCD because of atherosclerotic plaque formation in the coronary arteries[16] Studies have observed that prior to SCD, athletes had symptoms of shortness of breath, syncope and chest pain[15].

Sudden Cardiac Death (SCD) in athletes ranges from 1 in 40,000 to 1 in 80,000 per year[16] and it can be defined as a sudden change in cardiac rhythm in a short time causing unexpected death. Athletes may be perceived as healthy individuals however they can still have cardiovascular diseases which may have been acquired but are asymptomatic and it's first symptom could be Sudden Cardiac Death (SCD) or can have an increased risk of SCD. Symptoms such as shortness of breath, chest pain and syncope have been observed and a professional could be consulted for this may be misinterpreted as exertional symptoms.

Several cardiovascular abnormalities such as atrial fibrillation and ventricular fibrosis can increase an athlete's risk for Sudden Cardiac Death. It has also been observed that there are several factors in determining increased risk for SCD such as black (race), male (gender), and basketball players in collegiate athletes as documented by the National Collegiate Athletic Association[17] .

Sudden cardiac death is considered a multifactorial occurrence and awareness of the various causes and pathways known to increase the likelihood of SCD is important. Majority (80%) of SCD events occur in elderly patients with coronary arterial abnormalities, while minority cases of SCD stem from cardiomyopathy, valvular / congenital heart disease, and electrophysiological anomalies. [38] Several ECG abnormalities are associated with early and late SCD events occurring in middle-age subjects with no known history of

heart disease. Improvements in risk stratification are therefore urgently needed and, because SCD is primarily a result of electrical disruption of the normal cardiac rhythm, 12-lead electrocardiogram (ECG) remains an appealing non-invasive device outside clinical factors [37]

SCD can be prevented as long as the underlying pathology or anomaly can be detected earlier. In a 2017 journal it was proven that the ECG is the most effective and reliable assessment and diagnostic tool for cardiovascular diseases in athletes, the study concluded that the ECG had a sensitivity of 94% and a specificity of 93%[18]18. An ECG is painless and noninvasive and can be administered by healthcare providers such as nurses, certified technicians and doctors. When using this machine, the one administering the procedure should be careful on putting the adhesive leads on the skin of the one being examined for it may cause allergic reactions to the skin[19).

That being said, ECG should be part of the routine examinations before, during, and after playing or training seasons. There seems to be an obvious correlation between the risk of SCD and the number of observed abnormal ECG findings. The prediction of SCD should include multiple clinical risk factors (age, gender, diabetes, smoking, blood pressure, cardiac function) in addition to observed ECG abnormalities. [37] A 12-lead electrocardiogram can elevate the presumption of heart disease and lead to early diagnosis of entities such as hypertrophic cardiomyopathy. Interpreting the electrocardiogram is often challenging, since some changes in athletes are considered physiological. [8] With abnormalities such as sinus arrhythmias (47%) and sinus bradycardia (16%), T-wave inversion in the right precordial leads (69 per cent) is the most frequently seen uncommon ECG finding. In about 10 percent of the participants who were usually asymptomatic, ECG irregularities involving referral to a specialist were observed.[21]

The predictive power of various ECG tests ranged over the 10- and 30-year follow-up cycles.In summary, results suggest irregular QRST-angle, LVH and 0.2 mV with the greatest predictive value for SCD cases, and the cumulative impact of multiple ECG risk variants greatly increases the probability assessment of SCD events in individuals with no history of heart disease. [37] ECG risk variants were evaluated in the context of left ventricular ejection fraction (LVEF) for better prediction of future SCD events. [39] Length of QRS as a risk factor for SCD in 10-year study but in longer follow-up time no significance was observed in risk prediction. Nonetheless, the separation between QRSd / IVCD as independent markers for SCD or merely a manifestation of more proceeding cardiovascular disease remains debatable. Furthermore, middle-aged patients with no history of heart disease, extended QTc did not show significantly increased risk of SCD in any follow-up span of 30 years or 10 years [37]

That being said, the proved predilection of SCD in high intensity trained athletes may worsen in their intake of caffeine upon such high intensity training. The metabolic changes and affectations brought about by caffeine with the cardiovascular and electrophysiological changes brought by the training may increase the risks of cardiac events on the athletes. In some young people, unexplained cardiac arrest occurred after consuming energy drinks, especially at the same time as alcohol intake. Some stimulants are considered to be included in formulations in various energy drinks. [40] The statement that energy drinks impact the coronary conduction system and cause catastrophic events by fatal arrhythmias is more convincing. The goal of achieving higher athletic performance rates and academic success contributes to a steady increase in consumption of these caffeinated among the young population. [41]

The main concern is that these drinks could easily lead to severe cardiovascular events in young and old people with silent cardiovascular disease underlying them. Due to their high quantities of caffeine and other substrates, dangerous arrhythmias can easily develop in the hearts of the people who consume them.[42] The concern is that the packaging masks other potential outlets of caffeine. Frequent additives such as guarana, ginseng and taurine have amounts of caffeine in various energy drinks equal to or greater than those contained in coffee.[43] Although increases in blood pressure can be due mainly to caffeine, some hemodynamic risk may be caused by other ingredients in energy drinks. Taurine has been shown to lower blood pressure in prehypertensive patients, which shows the need to examine the combined consumption of caffeine and taurine. [46] Caffeinated energy drinks substantially extend the QTc period and boost post-acute sensitivity to brachial and central blood pressure. Further research into whether an actual component or a single mixture results in the reported electrophysiological and hemodynamic improvements is warranted. [47]

Additionally, synergistic interactions between energy combination components used in EDs may also have an effect on blood pressure. Caffeine's effects on hemodynamics can last up to five hours after ingestion. Such effects may be amplified by physical activity immediately following consumption. [48] Occasionally small changes in blood pressure are unlikely to cause significant side effects for healthy individuals and blood pressure frequently returns to normal after intake of ED has ceased. [46] This means, that athletes with an already abnormal ECG or ECG with numerous changes even if they could be explained as physiological compensations should decrease energy or caffeinated drink intake.

In any case, it seems clear that energy drinks, some beverages, and some supplements that include stimulants might lead to critical and rarely irreversible cardiovascular events in the young population. [41] It also indicates that regular physical activity when drinking coffee or beer, does not protect individuals with cardiovascular risk factors for coronary disease from sudden cardiac death. It is striking that the risk can be increased especially among those who are more frequently engaged in heavy exertion. [47] After rapid absorption of EDs in young adults atrial fibrillation has been observed. It is not clear whether these people were genetically predisposed to arrhythmia or whether additives in the EDs caused the events[48]

In normal individuals who exercise there will be an increase in blood pressure, cardiac output and heart rate thereby increasing the workload of the heart.

However in a trained athlete, cardiovascular changes may occur and it will depend on several factors such as an athlete's age, gender and intensity of exercise. Since in an athlete's heart, the workload is increased and volume is increased, it is commonly observed that they have increased left atrium diameter causing an increased left ventricle volume as well which is another possible contributor for supraventricular arrhythmias in athletes[15] due to frequent physical activity or exertion in exercising.

Different exercises may also contribute to the cardiovascular changes and physiologic changes. In athletes frequently undergoing endurance exercises it has been observed that there is normal or decreased peripheral vascular resistance and increased cardiac output together with increased maximum oxygen consumption[20]. Moreover, in athletes who frequently do isometric exercises, it has been observed that there is normal or only slight increase in cardiac output and increased blood pressure together with increased peripheral vascular resistance[20].

As discussed above and outlined, regular physical activity can boost a number of risk factors for CVDs, such as dyslipidemia or hypertension, but a well-powered study of the cardiovascular effects of exercise has shown that reducing the burden of classical risk factors will account for only around 59% of overall cardiovascular mortality reduction. [11] Which accounts for the remaining 41 percent risk reduction remains unclear, but it may be related to improvements in systemic inflammation as well as beneficial responses to acute inflammatory challenges. Indeed, exercise has profound effects on immune cells — natural killer cells, neutrophils, monocytes, regulatory T cells, and exercise changes the balance of T-cell types. [44]

Different types of exercise may be a contributor to the physiologic cardiovascular changes but environmental stress and emotional stress experienced by the athlete during competition is not widely researched[20]. Increased emotional stress during competitions and environmental stress may contribute to the possibility of triggering arrhythmias and myocardial ischemia in the heart due to the added increase in myocardial contractility, heart rate and blood pressure[20]. An athlete's heart may be able to tolerate this but there are some cases which can result in death. Diagnosing SCD in athletes may be difficult because the physiologic changes in an athlete's heart can be normal or abnormal. Misdiagnosing may cause adverse consequences or unwanted disqualification from the sport.

That being said, caffeine when consumed in the right dose shall produce positive effects that would benefit an athlete and produce the desired effects. [48] WADA, does not prohibit the consumption of caffeine but rather is part of the monitored substances during competitions and just recommends the usual dosage as accepted which is less than to equal of 400 mg per day, however since this is just a monitored drug, no specific percentage in urine or in blood has been established by the WADA as to quantify the intake of caffeine as doping during competitions. Conversely, The National Collegiate Athletic Association, urinary concentrations which surpass 15 µg / ml after competition are considered illegal. [7]

Moreover, the kind and type of caffeine is important in knowing the effects and how they will affect athletic performance. Coffee has around 1.01-1.37% caffeine[49], tea has varying caffeine content dependent on the steep time and type of tea with commercial teas varying from 9mg to 35mg of caffeine per 12oz, chocolates and chocolate products has around 2-57 mg of caffeine, and energy drinks has caffeine that range from 20-75mg per 12 oz., furthemore, the controversial caffeine powder has caffeine equivalent to 25 cups of coffee per one teaspoon of consumption. [5]

The efficacy of the intake of caffeine tends to vary depending on the type of workout. More stable, beneficial changes tend to occur at lower intensities (i.e. 60 percent 1RM) and for a still unknown reason, especially in lower body exercise.[3] The scientific literature associated with caffeine supplementation is comprehensive. It is clear that caffeine is indeed ergogenic to sport success but is unique to the athlete's condition as well as strength, length and mode of exercise. [7]

Repetitive involvement in intense physical exercise is now well known to result in significant improvements in myocardial structure and function.[27] High-intensity trained athletes who have cardiac compensations such as cardiac hypertrophy, abnormal ECGs who are already at risk for sudden cardiac deaths. Knowing that, and that a maximum of 6mg of caffeine produces these ergogenic effects, more importantly changes in cardiac functions, the adjunct use of caffeine in athletic training may increase the likelihood for sudden cardiac death in these athletes that do not present any cardiac symptoms at maximal exertion.

Theoretical Framework

This study is structured on the Activation Theory where a lot of factors are considered in how the caffeine is produced, ingested, processed, and consumed for the caffeine to take effect in the body. These effects would also vary depending on the consumer's age, gender, and race[23,24]. Caffeine can be sourced from teas, coffee beans and cacao beans, energy drinks, sodas, tablets or powder. In this study, caffeine was sourced from an energy which had 80mg of caffeine per 250mL. This amount of caffeine still falls under the considered safe dose of caffeine that can be consumed in a day which is 400mg supported by the study of Evans J[23].

Conceptual Framework

The conceptual framework shows a simplified input-process-output where athletes from a private university participates in the pre assessment to establish baseline with the use of an ECG machine and prior to ingesting caffeinated energy drinks and the same procedure will be done an hour after ingesting caffeinated energy drink so that results from the pre assessment and post assessment will be interpreted and compared to determine electrophysiological changes.

Research Objectives

The project proposal is a new study by the proponent. At the end of this research the proponent aims to:

1. Investigate what are the electrophysiologic changes observed after caffeinated energy drink ingestion

2. Investigatewhethertheelectrophysiologicchangesobservedarejustnormal for athletes or if it increases their risk of sudden cardiac death

Research Hypothesis

There will be observable electrophysiological changes that will contribute to the stress an athlete will feel prior to competing

Significance of the Study

This study was conducted to observe the electrophysiological changes in the cardiac vasculature under stress. The following will benefit from this study:

The subjects of the study. The study will benefit the subjects by imparting awareness.

The community. The study will serve as a basis for imparting information for the community of the risks associated with caffeine ingestion with associated cardiac diseases.

The coaches. The study will benefit them by imparting knowledge of what are other red flag signs that the athletes may manifest.

The future researchers. The research outcome will help as an additional reference and a source of information that will aid the future development of new studies regarding the utilization of coffee in increasing exercise endurance.

Scope and Limitations of the Study

This study will be conducted in the Southwestern University Rehabilitation Center and the participants are the athletes from a private university in Cebu City

The limitations of this study is that the number of subjects needed was not.

Research Design

This study utilized experimental research.

Results and Discussion

The electrophysiologic changes observed after caffeinated energy drink ingestion
All of the 5 subjects presented with changes expected in athletes. 3 had normal ECG and sinus rhythm before and after consuming caffeinated energy drink, 1 had rightward axis, borderline ECG, inferior ST elevation - possible early repolarization, and sinus arrhythmia after consuming caffeinated energy drink but had rightward axis, borderline ECG and possible ectopics atrial rhythm prior to consuming caffeinated energy drink, another 1 had sinus arrhythmia, rightward axis and borderline ECG after consuming caffeinated energy drink but had sinus rhythm, rightward axis, borderline ECG, and short PR interval before consuming caffeinated energy drink. These findings are studied to be normal in athletes whose hearts have already adapted physiologically.

Are the electrophysiologic changes observed normal for athletes or does it increase their risk of sudden cardiac death? All the electrophysiologic changes observed after ingestion of caffeinated energy drink were normal and expected with the subjects. The 250ml energy drink (Red Bull) consumed in this study only had 80mg of caffeine and it did not produce any abnormal arrhythmias or pathologic changes in the cardiovasculature of the subjects as supported by the literatures cited in this study [41,43]. Blood pressure and heart rate was not included in the comparison for this study. The right axis deviation in this study is considered normal since it occured in isolation and are normal in trained athletes since they are expected to have physiologic right ventricular hypertrophy and this does not need further investigation. The possible early repolarization observed in this study manifested as an ST elevation and is also considered normal because this is commonly seen in young athletes and will usually resolve during exercise [40]. However, early repolarization should be distinguished from pathologic findings such as Brugada syndrome and acute myocardial infarction. Sinus arrhythmias are commonly observed in ~15-20% of young athletes because of the neurovegetative changes related to practice (parasympathetic predominance), and its prevalence is especially high in aerobic sports with high level resistance.

Conclusion

The caffeinated energy drink used in the study did not contribute to risks associated with sudden cardiac death in collegiate athletes. The ECG changes observed were not pathological and were in parallel with the expected changes that will be seen in a trained young athlete.

Acknowledgment

The authors would like to thank her family and friends for their continued support in the implementation of this paper whether financially, emotionally and physically.

The authors would like to acknowledge the respondents, the athletes for their voluntary participation and cooperation in this study.

To the licensed Physical Therapists for helping the authors in completing this study.

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