The sudden cardiac death in young athletes health essay

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Coaches and athletic trainers should be taking further measures to prevent sudden cardiac death, no matter the cost or inconvenience of the method. There are a few reasons the study of sudden cardiac death (SCD) is an important area to investigate and research. SCD is a completely unexpected and traumatic event that is mainly caused by cardiovascular disease but also can be from blunt trauma and heat stroke (Maron, B. J., 2009). In young athletes SCD is particularly distressing because the person is in their prime and this event often challenges beliefs of the relationship between physical activity and good health (Anderson, L., 2012). We need to apply all methods of prevention that we have available to start saving children's lives before these causalities reach higher levels. Considering we're dealing with life or death situations, SCD seems a very applicable subject to spend more time in researching the causes, statistics, and preventative methods. This paper will further explore why the prevention of SCD is necessary and what forms of prevention are available that are frequently not being implemented. The rising causes of SCD are gaining media interest and giving society worry for their children. Advocates of ECG screening argue that the cardiac risk of athletic competition is different, because the athlete is unaware of the danger and benefits if screening tests reduce his or her likelihood of the remote contingency of SCD. The prevalence of SCD in media could be discouraging some parents from putting their children into competitive sports or scaring children from wanting to participate, also calling for an equally important response from coaches and athletic trainers across the world. There are more causes then one would come to expect for this one condition. Hypertrophic myopathy, coronary artery anomalies,

arrhythmogenic right ventricular dysplasia, atherosclerotic coronary artery disease, use and abuse of drugs, aortic rupture and Marfan's Syndrome are all causes related to cases of SCD. Also SCD has been seen in cases of idiopathic ventricular fibrillation, myocarditis, myocardial bridges, mitral valve prolapse, aortic valve stenosis, idiopathic ventricular fibrillation, commotio cordis, exertion-induced rhabdomyolysist (Futterman, L. G., 1998), (Maron, B. J., 2009). This paper will be focusing on the top three causes of SCD in young athletes. The most common cause of SCD agreed upon in a many different studies, (Futterman, L. G., 1998), (Maron, B. J., 2009), (Maron, B. J., 1996), (Cochella, S., 2006), as hypertrophic myopathy (HCM). Because of the adaptions of the athlete's heart, particularly wall thickness in the left ventricle, it becomes very hard even for an experienced doctor to differentiate between HCM and athletic adaptions of the heart from exercise. The hypertrophied muscle causes a reduction in chamber size of the left ventricle and therefore a reduction in volume. Consequently to being SCD's main cause, death is commonly the one and only symptom shown in cases of HCM (Futterman, L. G., 1998). Hypertrophic myopathy occurred in 36% of 251 cases in Maron, B. J.'s 2009 study and 46% in cases in Futterman, L. G.'s 1998 study. Athletes diagnosed with HCM are typically recommended to refrain from most competitive sports, irrespectively on condition of outflow from the left ventricle. Some limited low intensity activities may be permissible (Futterman, L. G., 1998). The second leading cause for SCD would be coronary artery anomalies (CAA) which was observed in both studies (Futterman, L. G., 1998), (Maron, B. J., 2009). An anomaly is just a deviation from the normal expected structure or function. The most common

anomaly is in the left main coronary artery from the right anterior sinus of the Valsalva. There isn't a clear understanding of the effect of CAA on SCD, but it is speculated that the acute angle of the coronary artery (right or left) in this abnormality creates a narrowing of the coronary ostium. This narrowing causes an added dilation from exercise and greatly reduces arterial blood flow. 17% of SCD cases were caused by coronary artery abnormalities and 12-14% of cases in Futterman, L. G.'s 1998 study. When coronary artery anomalies are identified, the intensity of the injury should direct the young athlete's withdrawal from competitive sports. 6 months after surgical correction, the athlete may commonly fully recover and return to participation in competitive sports (Futterman, L. G., 1998). SCD's third most common cause is disputed between studies but myocarditis is surely a top contender. Myocarditis is an acute inflammatory response affecting myocardial tissue and can alter cardiac function (Futterman, L. G., 1998). The inflammatory response can cause arrhythmias such as premature ventricular contraction, premature atrial contraction, and commonly atrial fibrillation. Myocarditis is regarded as a prodromal viral illness followed by fatigue, exertional dyspnea, syncope, palpitations, arrhythmias, or acute congestive heart failure with concomitant LV dilation (Cochella, S., 2006). Myocarditis caused 6% of cases in Maron, B. J.'s 2009 study. Because there are so many various causes to SCD 6% is actually a relatively high percentage of deaths. Typically, when myocarditis is detected in athletes, it is recommended that they withdraw from sports for at least 6 months. Prior to resuming activity the athlete is required to undergo cardiac assessment testing. Cardiac dimension and ventricular functions should be normal and

the pre-existing arrhythmias should not be present in the pre-activity ECG tests (Futterman, L. G., 1998). A common and unfortunate problem with SCD is that the cardiovascular diseases that cause SCD are rarely detected before death occurs. Most of these diseases go undetected during rest and aren’t present without the stress of exercise. Patents with SCD-linked conditions should be informed that vigorous exercise creates a 2. 5 times increased risk of SCD with 90% of incidents taking place during or just after exercise (Anderson, L., (2012). There are people with these SCD causing diseases and abnormalities living a normal healthy life. There are also people with these SCD causing diseases and abnormalities that aren’t aware and could be at a high risk for death. The fact that most causes of SCD could be easily prevented by pre-participatory ECG screenings makes it a topic the health field should be making more of a priority. A 1996 study by Maron, B. J., a prevalent researcher in SCD, looked at 134 United States deaths in trained young athletes (median of 17 years old) through were 1985 to 1995. The systematic study analyzed SCD cases categorized by race, cardiovascular cause, and sport. The cases were mostly males (90%). 68% of the cases were in football and basketball related SCDs. A total of 90% (121 athletes) collapsed during or immediately after a formal athletic event (43 cases) or a training session (78 cases). Consequently between 3 and 9 PM, 80 deaths (63%) occurred, being a prime time period for athletic events and training sessions. The results were that out of the 115 athletes who partook in a standard medical preparticipation examination, cardiovascular disease was detected as present in 4 subjects (3%). However, only 1 athlete was correctly diagnosed with the cardiovascular abnormality accountable for

SCD. Although the percentage seems minuscule, if 1 in every 130 athletes could be saved it'd be a great advancement for the prevention of cardiovascular disease (Maron, B. J., 1996). There are two methods to preventing SCD practiced by America and Europe; both involve catching symptoms of the several cardiac diseases mentioned earlier before they become problematic. There are a few different forms of electrocardiogram (ECG) screenings differing mostly in procedure and sometimes the amount of leads used in the ECG. The American Heart Association's 2007 protocol recommends a 12-element complete history and physical examination (with blood pressures) before playing any competitive sports. For athletes that test positive for abnormalities may also require non-invasive testing like 12-lead ECG, echocardiography, exercise testing, and cardiovascular consultation (Halabchi, F., 2011). Europe's procedures by The European Society of Cardiology (ESC) and the International Olympic Committee (IOC) were quite similar to the American approach. The ESC calls for screening questionnaires with similar inquiries to that of the 12-element AHA questionnaire. However, there are more questions than the American version and the content is slightly altered. A general physical examination, 12-lead ECG, and submaximal exercise test are required for any competitive athlete in which a doctor determines whether the athlete is healthy enough to play competitively (Halabchi, F., 2011). The substantial decrease in mortality with the application of more aggressive screenings may lead to the inference that this screening approach should be applied universally in all countries (Halabchi, F., 2011). ECG screening for athletes can provide great information into discovering the connections between exercise and

cardiovascular abnormalities/disease/SCD. Adding routine ECGs increases the effectiveness of the detection of hypertrophic myopathy and arrythmogenic right ventricular cardiomyopathies (HCM and ARVC) (Anderson, L., 2012). However, an ECG is not reliable in identifying congenital coronary artery anomalies and premature coronary artery atherosclerosis (Anderson, L., 2012). Also, follow up studies showed ECG has a 99. 98% chance of detecting HCM and an 80% chance of detecting ARVC, which together account for approximately 20% of SCD cases (Anderson, L., 2012). This statistic provides assurance into why ECG screening should be implemented in competitive athletes. These preventive methods are convenient and efficient in detecting the leading causes. However, not all of the conditions are equally detectable with one standard screening protocol, and this is worsened by the fact that the prevalence of each of the SCD-linked conditions may vary in ethnic groups. ECG screenings are not being implemented in everywhere they could be and aren’t held as mandatory frequently enough that would be necessary to change the effects of SCD. This is speculated as to why we aren’t seeing screenings implemented as commonly as they would be if all SCD-linked disorders could be so easily detected. All the statistics prove the need for the further mandatory and global practice of ECG screening for young competitive athletes. O’ Connor’s 2010 study at using data from the National Federation of State High School Associations concluded that ECG screening for every young adult in high school preparticipation examinations would require an extremely high budget and in most cases prove excessive. Mostly because of the low prediction rate and high expense follow-ups produced from mass false-

positive screenings. Varying ECG analysis, essential staff for ECG distribution and analysis, and time the patent is not participating until the follow-up test are all expected factors in these estimated high costs. But it is stated that by only testing athletes at high risk for cardiac abnormalities associated with causing SCD, the success of ECG screening could be greatly improved (O'Connor, D. P., 2010). Only testing athletes with a high risk for SCD causing abnormalities would definitely be more efficient than no testing at all. It's a great idea hypothetical speaking but has its fatal flaws. The evident loophole is that there are often people, especially young adults, who don't get regular checkups from physicians. If that child is allowed to participate without the further examination of an ECG screening they could have a condition and may not be aware of. This slipping through the cracks of this system, although presumably infrequent, is possible and could cause complications. The authors and researchers at The Michigan Department of Community Health and Michigan State University use data from MDCH Division for Vital Records and Health Statistics to try and identify SCD cases most likely to have a heritable cause with possible implications for surviving family members. Studies like these are being done in order to further our knowledge of SCD and collecting this data could save lives just by their findings. The hereditability of SCD is sometimes unclear and little research has been done in the subject. However, Mukerji, S.'s 2010 study recovered cause of death certificates from next of kin family members and an utilized an expert review panel to determine if SCD had any heritable gene that could been determined. The panel suggested that 17 cases or 73. 9% of the mortalities they studied were correctly detected as to having a possible

heritable cause. If SCD could be predicted by heredity or any other form, the lives of so many could be saved. It would be simple if it could be determined by genetics how at risk a person is for a cardiac abnormality known to cause SCD. A study by Shephard, R. J. in 2011 noted a decrease of SCD in Italian athletes since the introduction of mandatory ECG screenings. For athletes there is now a clearer classification of ECG abnormality, promising evaluations between medical exams with screening compared to medical exams without, and new calculations proposing benefits and lower costs of testing. With the success of these standards in Italy, these improving methods should be brought to the U. S. and set as new mandatory standards. It seems that there are professionals in the field trying to solve the problems of SCD in young adults but we are still just short of a comprehensive solution and just short of a widespread agreement. There is an agreement of the attention for a more effective screening standard including an increase in specificity of the test and focus on the test procedures upon a vulnerable subset of the athletic population to decrease misdiagnoses (Shephard, R. J., 2011). The leading medical cause of death and particularly during exercise in NCAA student-athletes is SCD. The NCAA needs to show more concern for their student’s risks because it’s especially cardiovascular demanding for the heart at the difficulty of the NCAA level. It is known that the current data collection methods typically underrate the risk of SCD. If SCD incidences can be accurately accessed it is necessary to shape health procedure decisions and develop prevention strategies that are most effective (Harmon, K. G., 2011). This is why the more information obtained about SCD we can obtain, the better of we are. Every study has a

different approach and different objectives that allow for new ideas and methods that could discover a solution to better the prevention SCD. Whether a study like Mukerj, S.’s 2010 containing data collection and analysis or a study like Anderson, L. 2012’s involving physically examinations, new information for SCD prevention is needed. With an ECG discovering a SCD-linked disease, a doctor’s recommendation eliminating exercise or sports could save a life. The rate of SCD in the USA is projected at 1 death per 45, 000 athletes per year (Harmon, K. G., 2011). The rates also seem to be climbing every year according to a few studies (Maron, B. J., 2003), (Maron, B. J., 2009), (Mukerji, S., 2010). If any lives can be saved, even if it’s only one child every million, it could prove worth the expense. There can’t be a price on a child’s life especially to their families. The concern for this deadly disease is rising but not at a large enough rate. Now is the time to push further and harder for progress in the sake of young lives. One could only imagine if a true prevention of SCD will ever be plausible but for researchers that is the thrill of the hunt. Nonetheless, sudden cardiac death in young athletes is present now and will be in the near future. Considering the youth is our future, we should be attentive to their health and well-being.