

What Swimming Coaches Should Know About Atrial Fibrillation and Cardiac Health

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Abstract

The purpose of this review is to investigate the literature related to atrial fibrillation (AF) in competitive master swimmers (females and males) and possible relationships to training history, individual and family cardiac history, and associated medications and the use of methyl xanthenes as ergogenic aids. Masters swimmers have been reporting AF at increasing rates. This study wanted to determine if this group had a higher incidence of AF when compared to non-exercising age matched master swimmers as well as age matched individuals involved in other sport or exercise activity. Some sources of AF could be related to training history, family cardiac health history, and the use of methyl xanthenes and Beta-2 agonists as ergogenic aids. Since many swimmers have asthma, we wanted to determine whether combining Beta-2 agonist asthma medications with methylxanthines would be useful in predicting atrial fibrillation in master swimmers. Another question examined was whether the use of methyl xanthenes could impact AF when used in combination with Beta-2 agonists. Creatine monohydrate (CM) has been shown to have a positive effect on recovery from coronary artery artificially induced occlusion and reduced fibrillation during and after the occlusion period. The researchers also propose to examine whether ingestion of vitamins like B6, B12, Coenzyme Q10 and Hawthorn extract resulted in lower incidences of AF or cardiac problems reported by master swimmers.

Key words: Atrial Fibrillation, Parasympathetic Tone, Master Swimmers

Introduction AF

Masters swimmers have been reporting atrial fibrillation (AF) at increasing rates. Researchers have examined several different possible influences and causes for these elevated rates of AF like training histories, individual and family cardiac histories, prescribed medications, and the use of methyl xanthenes and Beta-2 agonists as ergogenic aids. This review of literature examines many different questions related to possible causes of AF in master swimmers. Many swimmers have asthma and take Beta-2 agonist medication to treat their asthma. They may use methylxanthines as an ergogenic aid. Researchers have shown that the use of Beta-2 agonists has positive chronotropic and ionotropic effects on the heart. One question addressed by this review was whether Beta-2 agonist asthma medication when used with methyl xanthenes could be contributing to the development of AF in master swimmers. Creatine monohydrate (CM) has been shown to have a positive effect on recovery from coronary artery artificially induced occlusion and reduced fibrillation during and after the occlusion period. The authors also want to examine

whether the use of vitamins B6 and B12, Coenzyme Q10, Taurine, Ubiquinone and Hawthorn extract results in a significant increase or decrease of cardiac problems reported by master swimmers.

Reviewed Articles

Acute Cardiovascular Adaptations to Exercise

At the onset of exercise, several different acute cardiovascular exercise adaptations occur. Williamson, Fadel & Mitchell (17) reported that the Central Command mechanism in the brain processes information related to the process of swimming and the brain responds to the stress of the activity by increasing the heart rate in swimmers. Muscle contractions produced by swimming causes resistance vessels to dilate, and increases muscle conductance, blood flow, and tissue pressure resulting from intravascular pulse pressure (14). With further exercise, the blood vessels will continue to dilate to meet the oxygen requirements of the exercising muscles until maximal cardiac output and oxygen consumption are reached (4). As exercise levels increase, cardiac output and conductance begin to match one another and a steady cardiac level of 50-80 percent of maximal oxygen consumption is created and maintained by the swimmer. An increase in workload over 80% to 90% of maximal oxygen consumption requires the use of non-aerobic energy sources to meet workload demands.

Chronic Cardiovascular Adaptations to Exercise

Master swimmers can maintain training loads of 50 to 80 percent of maximal oxygen consumption for 1 to 4 hours per training session. The chronic cardiovascular training involved in master swimming training results in several different physical adaptations like improvements in cardiac function, and increases in ventricular volume, wall thickness, and end diastolic volumes, and decreases in end systolic volumes that result in increased stroke volume. Resistance vessels become more responsive to vasodilator activity and the parasympathetic nervous system slows the heart rate allowing greater time for ventricular filling. The autonomic nervous system begins to maintain a balance between cardiac output and conductance of the resistance vessels that provides for significant increases in muscle blood flow while maintaining normal blood pressure. A well-documented long-term adaptation to exercise that could be related to AF is an increase in the parasympathetic braking of the heart by reducing the output of the Sino Atrial Node (SAN) that is the heart's pacemaker. Presumably, this allows greater filling time for the left ventricle which then controls the end diastolic volume and results in an associated increase in stroke volume (4). This constant increase in parasympathetic tone could cause the regular cardiac tissue to change its characteristics and to become another pacemaker for the heart causing an irregular heartbeat.

Other Forms of Exercise and Atrial Fibrillation

Atrial Fibrillation has not been studied in master swimmers exclusively and long distance runners may not be a viable model to study because necrotic effects on heart muscle have been documented after training and after participation in long

duration physical activity such as marathon and ultra-marathon races over a period of years. (3,6,7,8,13,16,) The impact that aerobic training has on AF amongst long distance runners has been well documented. However, a comparison of the impact of training on AF between master swimmers and long distance runners may not be a valid comparison. Several researchers (1,3,16,) have reported that long distance running training and participation in marathon and ultra-marathon races over a period of years has a necrotic impact on heart muscles in long distance runners.

Parasympathetic adaptation to exercise has been a well-documented function of cardiovascular fitness. Swimmers practice exercise durations that are similar to those observed in long distance runners; however, long distance runners many times cover total distances that are between 3.5 to 4 times greater than master swimmers while training. Another significant difference between runners and swimmers is during supine recovery from exercise. There is a significantly greater parasympathetic influence on the heart from training among swimmers. When master swimmers or endurance athletes train their Vagus nerves innervates the SAN which is the primary pacemaker of the heart. It is possible that after years of training this constant parasympathetic inhibition of discharge from the SAN causes the adjacent cardiac tissue to remodel and become conductive tissue that results in AF. This could also be true for runners or cyclists but it appears that the parasympathetic effect is greater during and after supine exercises. Therefore, there should be a study that specifically focuses on the impact that master swimmer training has on AF.

While AF may be a medically controllable event, it has also been associated with atrial enlargement. This enlargement could lead to lower atrial pressures if the venous return remains the same. This could also result in a significant decrease in ventricular diastolic volume and a lower stroke volume that is the major determining factor for increasing cardiac output (14). Many master athletes compete with defibrillators or pace makers. Some have undergone cardioversion which is an electrical stimulation to normalize the rhythm of the heart. Finally, there is a medical procedure known as ablation which destroys the new pacemaker tissue that has developed. Therefore, AF in master swimmers is a condition that deserves a greater amount of scrutiny from researchers.

Possible Causes of Atrial Fibrillation Related to Swimming Exercise

Many master swimmers have been training and active in competitive swimming since they were in elementary or high school. This long-term training by many swimmers puts unusual strains on the heart and ventricular remodeling resulting in significant increases in ventricular size and strength. This has been documented by Mont, Elosua & Brugata (10). Swimmer training has been shown to result in significant anatomical changes like increases in ventricular chamber size and end-diastolic volume and decreases in end systolic volume. Many years of swimming training can result in these cardiac modifications that generally have a positive impact on health (6), but some researchers consider that the exercise and

accompanying ventricular remodeling. Other studies have shown that these altered morphologies of the heart lead to either physiological or pathological abnormalities in the ECG (7). With dramatic ventricular remodeling and function it could be possible that the atrium might attempt to adapt to training by developing more conductive tissue. This adaptation has been associated with Lone Atrial Fibrillation (LAF) (7,9). AF is associated with pathological conditions that have been observed in long-term active sports participants. A study by Maron and Pelliccia (8) found AF present in 63 percent of long-term endurance sports participants. These self-reported long-term sports participants were extracted from a larger population to study but the nature of their activities was not reported. In addition, female participants were used as part of the control group for these studies but not in the sports participation group only (8) and we assert that female master athletes should be studied.

Recently, there has been a particularly high representation of asthmatics competing in swimming. The high rates of asthma amongst swimmers are thought to be a result of swimmers breathing in warm air in close proximity to pool water with a higher humidity than the ambient environment. The expiration capacity of asthmatic swimmers could also be enhanced by the hydrostatic pressure of the water on their chest. Many asthmatic swimmers use Beta-2 agonists that are inhaled during practice and competition to control the effects of their asthma. Many young asthmatic competitive swimmers, when they become adults, will continue to use asthma inhalers and medications as they transition into participating in master swimming competitions. Researchers have recently found Beta-2 receptors in the human hearts proving the old adage learned in exercise physiology that there are no Beta-2 receptors in the heart. (3). This suggests that asthmatic swimmers that have used asthma medications for many years may have a greater chronotropic and ionotropic stimulus to their hearts possibly resulting in an abnormal sinus rhythm contributing to AF.

Atrial Fibrillation and Ergogenic Aids

Swimmers at all levels use ergogenic enhancing substances that can have a real or perceived effect on their performance. Methyl xanthenes, found in coffee, tea and chocolate, can have positive cardiac chronotropic and ionotropic effects on the heart. This means they speed up the heart rate and make the force of cardiac contraction greater. It is possible that years and years of taking this cardiac stimulant could result in abnormal ECGs. In addition, taking methyl xanthenes over a long period of time could produce undue cardiac stimulation possibly resulting in arrhythmias (5). We propose that if methyl xanthenes are taken in combination with a Beta-2 agonist there would be greater chronotropic and ionotropic effects on the heart. Recent studies have shown that the heart does have Beta-2 receptors and can be stimulated by a Beta-2 agonist which has previously been thought to work only on the vasculature of the lungs (5).

Some supplements have supposed positive effects on the heart and circulation. Creatine monohydrate (CM) is an ergogenic aid that provides a creatine molecule

that binds to the free inorganic phosphate to phosphorylate an ADP molecule during short-term exercise where energy is supplied via the ATP-PC system. Creatine monohydrate has been shown to have some positive effects on occlusion induced AF (13) and this suggests that swimmers using CM could be less susceptible to AF. If CM has been used over a long period of time it could have a positive effect on AF and could possibly be masking the effects of events that cause AF.

Health maintenance is a very important goal for many competitive master swimmers and they will take a variety of substances that they feel will improve their cardiorespiratory health. Some of these substances include vitamin B6 and B12, Co-enzyme Q10 or Ubiquinone a mitochondrial electron carrier (11). Another substance, Taurine, is taken by many master swimmers and is thought to strengthen the heart ventricles and reduce the risk of congenital heart failure. Hawthorne Berry is an herb that is taken by many and is thought to prevent coronary artery disease and high blood pressure. It would be interesting to determine if master swimmers who are free of AF take these supplements and determine if they have had any significant positive impact on their heart health.

Another factor related to AF is the accumulation of fat on the heart and in areas adjacent to conducting tissues. It was found that obese patients had better outcomes and mortality than lean patients when they were treated for AF. Badheka and coworkers (2) hypothesized that fat deposits around the heart serve as a protective mechanism and promote cardiac health. Since master swimmers are generally lean and highly fit, it seems plausible that lack of fat on the heart of master swimmers might contribute to higher incidences of AF. The authors suggest that estimation and analysis of percent body fat could be an important variable to study when determining the impact that AF. Could extremely lean master swimmers be experiencing AF at higher rates than master swimmers with higher rates of percent body fat?

Training History

Another possible variable that could lead to significantly higher rates of AF amongst master swimmers could be their training regimens. Detailed records should be collected that outline the training histories of master swimmers. How many years have they trained, did they train prior to puberty, did they train for endurance, middle distance or sprinting events. Longitudinal data regarding information on training volumes and incidents of AF in master swimmers could provide insight into the influence that modes and duration of training have on the parasympathetic impact that endurance swimming training has on the heart. We propose that the following factors should be examined along with training history: 1) individual and family history of cardiovascular disease and abnormal ECG, 2) training history of the swimmer including weight training and other supplemental training, 3) achievement levels in swimming for short, intermediate and long distances, 4) demographic data and physical health profiles, 5) use of ergogenic aids including methyl xanthenes, creatine monohydrate and other aids, 6) and any history of Beta-

2 agonist use to determine if this population has greater incidence of AF or other cardiac problems than the corresponding normal population. A research project should determine whether duration and intensity of training, achievement levels, demographics and the use of ergogenic aids; either by themselves or in combination with Beta-2 agonists, have any relationship with or can help predict incidence of AF in master swimmers. It should also be determined whether Vitamin B complexes, Ubiquinone and Hawthorn Root are associated with lower incidences of AF and other types of heart disease or cardiac abnormalities.

Discussion and Conclusion

This review of literature has shown that many athletes with a history of long-term training have made a strong positive cardiac adaptation to exercise as indicated by the enhanced baroreceptor reflex (16). However, there may be a negative side effect to this adaptation to exercise in some people. As the heart rate normally slows over the years, and with the added adaptation of training, the heart rate may slow to a point where there is cardiac tissue remodeling that results in AF not associated with any heart disease. Since this problem does not occur in all endurance athletes, we propose other factors associated with LAF such as family history, use of methyl xanthines, and beta-2 agonists be investigated. Conversely we propose that the use of a variety of substances used by master swimmers reputed to positively impact cardiac health also be investigated.

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