THE SWIMMER'S HEART

THE CARDIOVASCULAR INFLUENCE ACROSS THE SPECTRUM

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Swimming continues to be one of the most popular sports worldwide. Its attractiveness likely lies in its ease of accessibility, affordability and its modality as a non-weight bearing form of exercise. The role of exercise as a tool in cardiovascular disease prevention and treatment is irrefutable. The associated benefits of exercise include improvements in insulin sensitivity, lipid profile, blood pressure and endothelial function, leading to a decreased risk in all-cause mortality[1]. Nevertheless, there is a lack of dedicated research related to the effects of swimming in this spectrum of cardiovascular disease prevention and management. Due to the aquatic environment being distinctly different, the cardiovascular effects of land based exercise cannot be easily extrapolated to swimming. Measuring the effects of exercise underwater poses obvious challenges and therefore many assumptions have to be made.

In spite of the paucity of research, it appears that swimming can be well tolerated among patients with stable cardiac conditions. Importantly, prior engagement in swimming has been shown to reduce outcome severity and is now considered an effective tool in cardiovascular risk factor modification. The use of swimming as a tool to reduce the risk of cardiovascular disease is an emerging area, yet research has already identified a positive impact on hypertension[2], vascular function[3], lipid profiles[4] and insulin sensitivity[5]. Certainly its role in cardiac rehabilitation programmes shows promise and suggests swimming may be a useful way for stable patients to remain physically active.

The physiological response to water immersion has, however, led practitioners to be hesitant in recommending swimming for certain high risk patients, particularly those with chronic heart failure (CHF) and severe myocardial infarction (MI). The aquatic environment places an increased haemodynamic burden on the individual, principally due to the effects of hydrostatic pressure. The compressive force of hydrostatic pressure typically causes a displacement of body fluid into the thoracic cavity, increasing right atrial pressure[6]. Through this process, extracellular fluid moves into the vascular system and, with an extra 700 ml of blood diverted centrally, it serves to increase venous return[7]. The

horizontal position of the swimmer further aids venous return, which is again only increased by the kicking of the legs[8].

The concomitant increase in preload – the initial stretching of cardiac myocytes prior to contraction – is seen in both left and right ventricles, increasing stroke volume and cardiac output. Generally, increases in stroke volume can be attributed to not only preload, but also afterload and contractility. The marked increase in central venous pressure without any change in vascular compliance, coupled with an increase in left ventricular end diastolic volume of up to 250 ml sugests that the increased stroke volume is, however, likely a result of enhanced preload. For patients with chronic heart failure for example, these changes may place undue stress on an already burdened heart.

Swim training following a severe MI has been shown to have a harmful impact in rats, due to intensified left ventricular remodelling and increased wall stress[9]. These factors led to a significant decrease in overall survival, an effect which was exacerbated when swim training started early after MI. Rats with only small infarcts lacked haemodynamic compromise or alterations to left ventricular structure and volume, implying that only severe infarcts cause adverse remodelling.

Research in human patients with MI and CHF has found similar results. A study conducted by Meyer & Bucking[10] found that even during a resting position in water, patients 3 to 4 months post-MI had increased mean pulmonary capillary pressures (19 mmHg), compared to on land (9 mmHg), indicative of a pathological expression. When the patients engaged in slow pace swimming (20 to 25 metres/min) these pressures further increased to a mean of 32 mmHg; and were higher compared to a cycling group (32mmHg vs 29mmHg). The authors suggest this shows hydrostatic pressure is the driving force behind the increase in preload. These results support concerns that swimming could cause the heart to be overloaded in these specific disease states.

CARDIAC ADAPTATION IN THE ELITE SWIMMER

Structural and functional adaptation

While the characteristics shared between a highly conditioned elite swimmer and an individual with cardiovascular disease may be limited, the heart of both individuals will undergo remodelling to withstand the increased demands placed upon it. The mechanisms behind this remodelling are vastly different, but in essence occur in response to either myocardial injury or elevations in the physiological loading of the heart during exercise.

The concept of sport-specific adaptation of the athlete's heart has long been identified and the divergent cardiac remodelling to different forms of exercise was initially explained by the 'Morganroth hypothesis'[11]. The model proposes there to be a dichotomous form of remodelling, whereby those participating in resistance exercise would experience concentric remodelling of the heart, whereas those engaging in endurance sports would see an eccentric form of adaptation. In reality, sports are rarely exclusive to just one of these definitions and such a classification may be deemed over simplistic. It has since been discovered that certain sports involving both a static and dynamic component elicit both concentric and eccentric forms of remodelling[12]. According to the Mitchell classification[13], swimming is categorised as a highly dynamic sport with

a moderate static component, nevertheless, it has, to date, been poorly represented in the published literature. Similar to the possibility of swimming overburdening the heart of a CHF patient, the aquatic environment and the physiological mechanisms for increasing the load placed on the heart, brings about the question of whether swimming could elicit a greater athletic adaptation than those seen in land-based sports.

Among sports in which the upper body predominates, higher pressures have been commonly seen when compared to sports using the larger lower body musculature. In spite of these observations, blood pressure does not rise to the extent of that seen when running or strength training. Swimming has in fact been associated with very little change in afterload - the pressure against which the heart must work to eject blood. The somewhat lower afterload, which represents a lower resistance to ventricular emptying, is exemplified by a lower mean arterial pressure and total peripheral resistance[14]. Studies have shown that total peripheral resistance significantly decreases upon water immersion and may be linked to the lower sympathetic input that is seen when immersed in water[15]. Beyond 60% of an athlete's VO2max, heart rates can be approximately 10 to 15 beats per minute slower when swimming in a thermoneutral temperature than running[8]. In all, a relatively high cardiac output must be maintained to accommodate normal blood pressure when the sympathoadrenal response is lowered[16].

Coupled with the higher indexes of preload and cardiac output during swimming, the four-fold greater energy cost of swimming – associated with the greater resistive forces and less gravitational forces exerted upon swimmers – could suggest that the heart of the swimmer remodels in a bifactorial manner, with a significant increase in both wall thickness and internal diameter of the left ventricle.

Research conducted in swimmers has, however, included small samples and sub-elite populations[17-20]. These studies have collectively shown swimmers to have larger hearts than their non-athlete counterparts but the reason for this development remains equivocal. When swimmers have been included among large scale data sets of competitive/elite athletes the results are somewhat ambiguous[21-26]. Several factors affect the morphological remodelling of the swimmer's heart, including ethnicity, age, body surface area, gender and level of training. All of these must be considered when interpreting the data and, so far, have not been addressed in a well-controlled study using a large scale, truly elite cohort of swimmers.

Several studies have shown no profound differences in terms of structural remodelling among swimmers in a comparison with other elite sportspersons[21,22,26]. Conversely, when the effects of age and body surface area are accounted for, swimming is associated with greater cardiac dimensions. Spirito et al[25] and Whyte et al[24] found that swimmers possessed some of the largest hearts both in terms of wall thickening and ventricular cavity dilatation, ranking third in an analysis of 27 sports.

An area which is under-reported in the literature is remodelling of the right side of the heart due to athletic participation. At rest, the workload of the right ventricle (RV) is minimal due to the very low RV afterload and, as such, the myocardial energy cost of RV cardiac output is approximately one-fifth of that of the left ventricle[27]. During exercise, this significantly changes and it has been

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observed that the greater the cardiac output, the greater the pulmonary artery pressure and RV load. Considering the higher cardiac output seen when swimming, it could be proposed that swimmers may show profound right sided remodelling. Indeed, this is supported by research from Italy[28], which found significantly larger RV outflow tract, right ventricular diameter and right atrial dimensions in middle- and long-distance swimmers. The authors suggested the accompanying decrease in total peripheral resistance may induce chronic venous overload, enhanced right ventricular diastolic filling and elevated right atrial and ventricular dimensions.

Electrophysiological adaptation

As already discussed, the heart of the athlete varies significantly to that of a non-athlete. When analysing ECG results, alterations in the electrical pattern seen in athletes can be categorised into two distinct groups – normal (training-related) or abnormal (training-unrelated). These classifications have, however, been drawn using unselected cohorts of competitive athletes from a wide variety of sports[29] and thus it remains to be seen whether these criteria are applicable for swimmers, who predominantly train in an endurance capacity. The limited research that so far exists suggests that elite endurance athletes do have a greater prevalence of normal training-related ECG findings, but notably more abnormal ECGs than their non-endurance counterparts. In the first study to investigate this concept, a research team from Australia found that 1 in 3 endurance athletes had an abnormal ECG, according to the ESC 2010 guidelines, compared to only 1 in 6 non-endurance athletes[30]. These results must be interpreted with caution given the significant advancements in ECG interpretation and adoption of the latest 2012 Seattle criteria[31] would undoubtedly decrease this prevalence. Nevertheless, the difference was largely due to findings suggestive of RV pathology among the endurance athletes, in particular RV hypertrophy and right precordial T-wave inversions. The cohort was predominantly Caucasian (97%) and T-wave inversions in this population should always raise suspicion of disease. However, over 14% showed no indication of pathology upon further investigation, suggesting the abnormal findings could be an adaptation of intense endurance exercise, potentially due to chronic RV overload. While such athletes should always be investigated further for signs of pathology (principally arrhythmic right ventricular cardiomyopathy and pulmonary hypertension), it opens up questions regarding whether current ECG interpretation guidelines are specific enough for elite endurance athletes.

The findings from a pilot study conducted at the recent 12th FINA World Swimming Championships (25m) support the previously-documented findings in suggesting swimmers exhibit profound electrocardiographic remodelling. This unpublished data showed 94% of athletes had at least one training-related adaptation, such as isolated left ventricular hypertrophy, early repolarisation and/or sinus bradycardia. The most common abnormal finding was right atrial enlargement. Together with the finding that one in five demonstrated right ventricular hypertrophy, this once again indicates the potential for an increased RV load in elite swimmers.

Research investigating structural, functional and electrophysiological adaptations of the heart allows us to identify the characteristics of physiological remodelling in elite athletes. The responsible team physician or cardiologist can then place these findings in the context of pathology. The consistent problem for physicians has been negotiating the 'grey zone' of cardiac remodelling that is found between physiological adaptation and underlying cardiac pathology related to sudden cardiac death (SCD).

SCD prevalence among collegiate swimmers has been reported at one in 42,000[34]

SUDDEN CARDIAC DEATH IN SWIMMERS

While offering protection against a wide variety of non-communicable conditions, exercise can transiently act as a substrate for SCD among those with an underlying cardiovascular condition. Strenuous exercise places these athletes at a greater risk of associated mortality than their non-athletic counterparts, increasing the risk of SCD 2.3 fold[32] and sudden cardiac arrest 3.7 fold[33]; a fact particularly pertinent when one considers that elite swimmers are consistently training up to 40 hours per week. Incidence of SCD in athletes is typically between one in 50,000 to 100,000. Recent data investigating American collegiate athletes has revealed this value to vary significantly according to sporting discipline. SCD prevalence among collegiate swimmers has been reported at one in 42,000[34].

Special Focus - Long QT syndrome

Swimming also represents a specific risk for some individuals harbouring a particular disease related to SCD – long QT syndrome (LQTS). Long QT syndrome affects around 1 in 2000 individuals[35], however may be underreported given the presence of concealed LQTS. Diagnosis is based upon a combination of symptoms, family history, genetic testing and electrocardiogram (ECG) findings. It is a congenital ion channel disorder whereby, through the impaired regulation of the movement of the specific ions, the heart takes longer to recharge – or repolarise – after each heartbeat. A window of electrical vulnerability may result, during which the heart could be caught off guard and trigger a potentially lethal arrhythmia such as torsades de pointes and ventricular fibrillation. The mutations responsible for LQTS have been found in 15 genes to date[36]. Of these genes, it is LQT1 which is of particular concern for swimmers due to exercise being the predominant trigger. In fact, one third of events in LQT1 occur during swimming[37]. Why swimming is a particular trigger over any other land based exercise is a question that has been the focus of researchers for two decades.

Although swimming may induce a lesser degree of adrenergic stimulation, the combination of voluntary apnoea, face immersion, potential cold water exposure and physical exertion cause a conflicting drive between both parsympathetic and sympathetic activity, or 'autonomic conflict'[38,39]. In individuals who are already susceptible, their underlying condition of LQT1 may predispose to the pro-arrhythmic effects of autonomic conflict. Furthermore, when heart rate increases due to the sympathetic drive, the typical response is for specific potassium channels to increase activity and shorten the QT interval; however, in those with LQT1, there is a reduced activity of these channels, preventing QT shortening. Together with facial immersion in water prolonging the QT interval, this increases susceptibility, making the heart vulnerable to harmful ventricular arrhythmia.

Current guidelines from the American College of Cardiology and European Society of Cardiology vary somewhat with regards to competitive restriction[40,41]. Both recommend disqualification in symptomatic swimmers or those with QT prolongation. Although the American Heart Association permits athletes with a genotype positive/phenotype negative presentation to participate in competitive sport, swimmers are an exception to this rule. The European Society of Cardiology advises disqualification for all athletes. Recent research has, however, suggested that these guidelines may, in fact, be too strict. Work from Johnson & Ackerman[42] showed that among LQTS-diagnosed athletes, none who were genotype positive/phenotype negative had any cardiac events over a 6-year follow-up and only one athlete who was genotype positive/phenotype positive had a cardiac-related event, with no deaths recorded. Ultimately, managing an athlete with LQTS is challenging, however it is clear, particularly in light of recent findings, that more long-term research is needed.

PREVENTION OF SCD IN SWIMMERS

Cardiovascular pre-participation screening

With SCD being the first presentation of any pathological condition in up to 80% of cases, together with the increased risk of SCD in athletes, pre-participation screening for this population is now widely acknowledged on medical, legal and ethical grounds as a necessary step in detecting these often silent conditions. Early detection of potentially lethal conditions may allow timely intervention and management to reduce mortality. It is this outlook that has led to the widespread adoption of cardiovascular pre-participation screening by numerous sporting organisations and governing bodies, including swimming's own federation, FINA.

In accordance with the organisations mission, FINA:

"should take care that sport is practised without danger to the health of the athletes and with respect for fair play and sports ethics. To that end, it takes the measures necessary to preserve the health of athletes and to minimise the risks of physical injury and psychological harm"[43].

FINA currently recommend screening for all their elite athletes and stress that the completion of cardiovascular screening should be under the provision of a specially trained physician. Conversely, on the back of several high profile cases, sporting bodies such as FIFA, FISA and the UCI have taken a fully comprehensive approach by mandating the practice in all athletes prior to its flagship events. While commendable in its preventative efforts, this approach is open to several logistical challenges, largely related to concerns regarding infrastructure, financial support and the necessary qualified expertise to interpret the results and manage the athlete, particularly in developing countries. Indeed it is athletes of African/Afro-Caribbean descent who pose the greatest risk of SCD. Adopting a unique position, FINA (in collaboration with Aspetar) have recently piloted an

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in-competition screening programme, allowing every effort to be made to provide the equal opportunity for athletes to undergo cardiovascular screening, where it would otherwise not be possible.

The initiative of FINA in piloting a screening programme not only allows for the determination whether screening in-competition is feasible, but also allows us to establish the impact of swimming on electrocardiographic remodelling of the heart and whether swimmers have a greater prevalence of abnormal findings.

Secondary prevention

With the implementation of ECG-led screening, longitudinal trends of SCD incidence have seen a reduction of up to 89%[44]. Cardiac arrests do, nevertheless, still occur and the preparedness to deal which such an event is essential. In the case of a drowning victim the most significant consequence is hypoxia[45]. As such, when the victim is on land, rescue breaths should be immediately performed. It is important oxygenation, ventilation and perfusion be restored as quickly as possible and warrants the traditional 'A-B-C' approach (airway, breathing, circulation) to initial CPR[46]. While cardiac arrest in drowning victims is most often associated with non-shockable cardiac rhythms[47], in the event that an individual does present with a shockable rhythm, the most important determinant of sudden cardiac arrest survival is the time from arrest to defibrillation. With the chance of survival dropping up to 10% with each passing minute[48], on-site automated external defibrillator (AED) availability is vital. The aquatic environment makes recovery of the victim more difficult. Prompt recognition and management of such event is again imperative, all the while, in such situations the rescuers' safety must also be considered[46]. Although drying the chest prior to AED pad placement is recommended, it is not a contraindication for the use of an AED.

OPEN WATER SWIMMING

In 2008 open water swimming became an Olympic event and its popularity among the general public has sharply increased. With this surge in mass-participation event popularity there is an increased risk of adverse medical events. Indeed as many as 95% of all triathlon deaths occur during the swimming segment of the event and the incidence is positively associated with the number of participants[49]. What causes more triathletes to suffer an arrest during the swim rather than the cycle or run has been another question at the forefront of research in recent times and could greatly assist in medical team readiness.

Timely intervention may be an important factor in the rate of SCD attributed to swimming. A loss of consciousness which would otherwise be easily identified on land may be more difficult to detect in the mass events of open water swimming. The result could be drowning and death. While rescue personnel are always present on watercraft, rescue and timely defibrillation is made more challenging in this environment.

The mechanisms for such events include exertional heat stroke, swimming-induced pulmonary hypertension and 'autonomic conflict'[38,50]. As already discussed, co-activation of the parasympathetic and sympathetic nervous systems may be exacerbated in susceptible individuals in open water due to the confounding

factors of cold water, increased anxiety and extended breath-holding. Current FINA rules state that during open water swimming, temperatures must lie between 16 to 31°C[51]. It has been shown that rapid submersion in cold water (15°C), while holding ones breath, is a particularly strong stimuli for the autonomic conflict by eliciting both the cold shock response and diving response. In predisposed individuals, such as those with LQTS, atherosclerosis, myocardial hypertrophy or ischemic heart disease, this reaction may lead to a confused rhythm and ventricular arrhythmias[38]. Unlike the other mechanisms cited, it is particularly under-appreciated and, while difficult to prove, could provide a reason for deaths with no explanation and often associated with drowning.

A factor in preventing such incidences may be the athlete's readiness to participate. It is known habitual cold water exposure leads to a diminished cold shock response; however with the exception of the very elite, swimmers do not often train in the environment they are going to compete in. Typically, the amateur swimmer trains in a heated indoor pool, with proportioned lane markers where any chance of collision is less likely and where they can take a break at any point and thus it is not representative of the true competitive environment. As well as any anxiety, open water swimmers may have to contend with rough water, crowding, currents, deep-water starts, beach run-in starts and poor visibility[52]. These factors all mean a swimmer's first encounter with open water swimming can be daunting.

SUMMARY

Whether we are talking about swimming, diving or any other form of aquatic activity, the effect of water immersion has a profound impact on the human cardiovascular system. This effect resonates strongly throughout the entire human lifespan and ranges from the positive impact swimming can have on several cardiovascular disease risk factors, to its potentially lethal role in athletes with underlying cardiac pathology.

References

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