



Review

Swimming and the heart

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ABSTRACT

Exercise training is accepted to be beneficial in lowering morbidity and mortality in patients with cardiac disease. Swimming is a popular recreational activity, gaining recognition as an effective option in maintaining and improving cardiovascular fitness. Swimming is a unique form of exercise, differing from land-based exercises such as running in many aspects including medium, position, breathing pattern, and the muscle groups used. Water immersion places compressive forces on the body with resulting physiologic effects. We reviewed the physiologic effects and cardiovascular responses to swimming, the cardiac adaptations to swim training, swimming as a cardiac disease risk factor modifier, and the effects of swimming in those with cardiac disease conditions such as coronary artery disease, congestive heart failure and the long-QT syndrome.

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1. Introduction

Swimming has long been a popular recreational activity, but is recently gaining widespread acclaim as an effective option in maintaining and improving cardiovascular fitness. In the United States, swimming is second behind walking as the most popular sports activity [1]. Internationally, there are a reported 18,784 facilities with 20,108 full size year round swimming pools in 168 countries. Its appeal for elderly patients and those with cardiovascular disease primarily lies in its low impact nature. In patients with arthritis, swimming and other water-based activities were shown to improve joint function, without worsening symptoms, thereby making it particularly attractive to those who struggle with these issues [2].

Exercise training is presently accepted to be beneficial in lowering morbidity and mortality in patients with cardiac disease [3]. However, in past studies, swimming has been attributed with sudden cardiac death. One study found that 11% of recreational deaths in Rhode Island were related to swimming [4]. However, cardiovascular deaths that result from swimming must be differentiated from drowning deaths, which are more likely to occur in unsupervised locations than those due to a primary cardiac cause. While some studies have shown that swimming can be an arrhythmogenic trigger for long QT-syndrome [5], data shows that swimming comprises only 2% of all deaths in young athletes, the majority of whom had an underlying cardiac condition [6]. In addition, recent evidence shows that swimmers may have lower all-cause mortality risk as compared to sedentary, walking and

running counterparts [7]. Of course, results are dependent on the duration and intensity of the exercise. Accordingly, the risks and benefits of swimming as a cardiovascular exercise merits review, particularly in those with cardiac disease. Additional objectives of this article are to outline the physiologic effects of swimming, and to describe the cardiovascular responses of swimming and the cardiac adaptations to swim training.

2. Physiologic effects

Swimming differs from most other exercises such as running in many aspects including medium, position, breathing pattern, and the muscle groups used. Body position and medium make swimming an especially unique form of exercise. Water immersion places compressive forces on the body, which serve to increase pressure in the capacitance vessels, thereby causing a shift of volume into the thoracic cavity. This shift of volume into the thoracic cavity thereby augments venous return [8,9]. Greater venous return provides higher preload to the right and left ventricles. Left and right heart filling pressures including central venous, right ventricular end-diastolic, left atrial, and left ventricular end-diastolic pressures increase. Left and right ventricular stroke volume increase according to the Starling mechanism by as much as 75–120 cc, with a resulting increase in cardiac output by 30–60%. Blood pressure elevation then activates baroreceptors, which reduce heart rate [10]. The interplay between the nervous and cardiovascular systems in water is also exemplified by water induced skin wrinkling (WISW). Several studies have shown a relation between the degree of WISW and central autonomic function, which may have prognostic value in cardiovascular health [11–13].

Muscle tissue is thought to be hyperperfused in water since oxygen consumption is identical to land, yet cardiac output is increased

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[14]. In addition, the redistribution of volume that occurs with swimming leads to an elevation of left and right atrial pressure. This stimulates the release of atrial natriuretic peptide, which acts to increase urine output and sodium excretion and leads to antidiuretic hormone (ADH) inhibition, thereby reducing intravascular volume [15]. Different forms of exercise have also been shown to affect the autonomic nervous system differently. The lower levels of epinephrine, norepinephrine and renin levels seen in swimmers is indicative of a blunted sympathoadrenal response with high-intensity exercise [16,17]. A comparison of resting plasma catecholamine levels and adrenergic receptor density and responsiveness in swimmers, runners, weight lifters, wrestlers, and untrained men demonstrated a lower resting sympathetic activity in swimmers and runners [18]. Swimmers and runners had lower baseline plasma norepinephrine levels, lower norepinephrine/epinephrine ratios as well as lower beta receptor density and increased alpha II receptor sensitivity (primarily in vascular and skeletal tissue) as compared to the other groups when measured at rest. Swimmers were unique in that they had labile blood pressure readings, due to reduced baroreceptor sensitivity. While swimmers have demonstrated lower sympathetic activity and lower catecholamine levels at rest, they have a different response with activity. Guezennec et al showed that the volume shift induced by the supine position and water pressure during swimming attenuated the plasma renin activity response, although plasma catecholamine levels were higher than in runners when measured with maximal activity. Both runners and swimmers still demonstrated a lower catecholamine surge when compared to untrained athletes [19]. These factors, coupled with nitric oxide release induced by external compression from hydrostatic pressure, leads to a decrease in systemic vascular resistance [20,21]. Evidence shows, however, that previous physical fitness and skill level determines the extent of sympathoadrenal response, with a reduced response seen in those who were more physically fit [21]. Water immersion has also been shown to increase the amplitude and duration of reflected aortic pressure waves using applanation tonometry, indicating an increased left ventricular workload and oxygen demand. These findings are likely due to increased venous return and LV preload [22].

The energy requirements of swimming are related to translational motion (drag forces) and horizontal motion (lift forces) [23–25]. Drag forces increase exponentially and lift forces decrease as the speed of swimming increases by the following relationship: $V_{max} = (E_{max}/e)/D$, where V_{max} is the maximal attainable velocity and E_{max} is maximal total energy production rate of the swimmer, e is mechanical efficiency and D is the water resistance to overcome. These factors collectively reduce the mechanical efficiency of front-crawl swimming, which ranges between 5% and 9.5% [26]. Accordingly, the energy cost of swimming a given distance is about four times greater than the cost of running the same distance. Energy expenditure is also related to other variables including individual skill, gender, and stroke. At any given velocity, the energy requirement of an unskilled swimmer is nearly twice as high as that of an elite swimmer [27]. Women swim a given distance at 30% lower energy cost than men due to their higher percentage and more peripheral distribution of body fat, which serve to decrease lift forces required to stay afloat [26]. Therefore, women swim faster at any given energy expenditure. The energy cost at any given swimming speed is lowest for the front-crawl, followed by back-stroke, butterfly and breast stroke [27].

Water poses thermal stress on the body, which has physiologic effects and consequences on performance. In well-trained swimmers, swimming speed, peak heart rate, and lactate production are directly related to water temperature, with faster times, greater heart rates, and higher lactate levels observed at warmer water temperatures [28]. In addition, greater sympathetic tone leading to higher norepinephrine levels and blood pressures were seen in healthy subjects immersed in 20 °C water as compared to 32 °C [29,30]. The increase in parasympathetic tone that is experienced with water immersion

is counteracted by increased sympathetic tone on entering cold water [30]. Further, colder water temperatures are associated with greater left ventricular end diastolic volumes, indicating increased preload in lower temperatures, while higher temperatures are associated with vasodilatation and decreased afterload [14]. This increased preload is the likely result of increased peripheral vasoconstriction. Additionally, the horizontal position of the swimmer provides for greater venous return and is augmented by kicking of the legs. The aquatic environment also necessitates controlled breathing frequency, which varies with stroke mechanics. Breathing frequency has been shown to affect oxygen uptake and minute ventilation but not heart rate or lactate production [31]. As compared to air, water poses greater resistive forces and less gravitational forces to the moving athlete.

3. Cardiovascular responses and adaptations during swimming

Many studies have shown an 8–10% lower VO_{2max} during swimming as compared to other exercises [32,33]. This is likely caused by the smaller size of the active muscle mass, which limits oxygen extraction, and possibly related to previous findings that the supine position results in a 15% decrease in VO_2 relative to being erect [34]. O'Toole reported VO_{2max} in triathletes to be 13–18% less in tethered swimming, compared to treadmill running values [35]. However, this has not been a universal finding in other groups of subjects. While one study showed recreational swimmers to average 19% lower VO_{2max} during swimming than treadmill exercise [36], another found trained swimmers to attain higher VO_{2max} during swimming than during running and cycling [37], and a third study found sedentary subjects to attain similar VO_{2max} during swimming and cycling [38]. The results of these studies likely vary because of differences in the following factors known to influence either oxygen consumption or energy consumption, which are interrelated: individual skill, speed, cardiac status, water temperature, and attire. Previous studies have shown that at any given velocity, the oxygen uptake of an unskilled swimmer is higher than for a trained swimmer [27], and that there is a linear relationship between oxygen uptake and swimming speed. The additional oxygen consumption of swimming in cold water results primarily from the energy expended in shivering as the body attempts to regulate core temperature. A swimmer's attire can also affect oxygen consumption. In one study, oxygen uptake and minute ventilation were lower during swimming with a wet suit as compared to without [39]. Swim training has also been shown to improve performance and physiologic adaptation to future exercise. Lieber et al showed that both run and swim training increases subsequent treadmill VO_{2max} significantly and to the same extent [40]. This finding was in contrast to previous studies, which showed smaller improvements in VO_{2max} after swim training. It was postulated that the differences in the results [41,42]. Lieber et al exercised participants in the swim arm of the study at 75% of the VO_{2max} achieved on the treadmill, which is the may have been due to a difference in the specificity of skeletal muscles trained, and a lower cardiovascular training intensity among the swimmers same as they did for participants in the running arm of the study. In previous studies, swimmers were not exercised at the same maximal intensity. Swimming was also found to improve bicycle exercise performance with VO_{2max} increasing by 16% in 12 sedentary middle-aged persons [43]. Although there were no changes in ejection fraction or end-systolic volume, there was an 18% increase in peak left ventricular end-diastolic volume and an 8% rise in peak systolic blood pressure.

At any given oxygen consumption, cardiac output is about the same in swimming as it is in running; however the maximum value is significantly lower for swimming. Cardiac output is probably not limiting for performance since swimmers easily achieve higher values during running [27]. Despite similar cardiac output, stroke volume is greater and heart rate is lower during swimming as compared to other aerobic sports. Higher stroke volume is related to water immersion and supine body position augmenting venous return and enhancing diastolic filling. The peak heart rate achieved is approximately 10–15 beats/min lower

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