# THE IMPACT OF SWIMMING DURATION ON EXERCISE-INDUCED CARDIAC FATIGUE

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# Abstract

Substantial research has been conducted on exercise induced cardiac fatigue (EICF), with multiple studies showing an acute negative impact on cardiac structure and/or function post prolonged exercise (Oxborough et al, 2010). Although the current literature has highlighted EICF in terrestrial based exercise over varying durations and intensities (Donaldson et al, 2019), little has been investigated around aquatic-based sport. Swimming is a popular sport which exposes the cardiac system to unique physiological challenges, such as changes in pressure and gravity, which may exacerbate the effects of EICF (Riding, 2016). This study aimed to observe the impact a 30min and 60min moderate intensity swim had on cardiac structure and function and how the changes were comparable to the current literature. The results found evidence of reduced function in both ventricles post exercise with LVGLS and RVGLS reducing by 2.4% and 2.3% post 30min swim respectively, comparable to that observed after much longer durations of land-based exercises (Oxborough et al, 2011). The reduction in function appears to be caused by an overall reduction in ventricular filling as no evidence of myocardial fatigue was present in either ventricle.

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**Abbreviations**

**LV- Left ventricle**

**RV- Right ventricle**

**LA- Left atria**

**EDV- End diastolic volume**

**ESV- End systolic volume**

**DA- Diastolic area**

**SA- Systolic area**

**DBP- Diastolic blood pressure**

**SBP- Systolic blood pressure**

**LVEF- Left ventricular ejection fraction**

**LVGLS- Left ventricular global longitudinal strain**

**RVFAC- Right ventricular fractional area change**

**RVFWS- Right ventricular free wall strain**

**RVGLS- Right ventricular global longitudinal strain**

**TAPSE- Tricuspid annular plane systolic excursion**

**FS- Fractional shortening**

**TM- Trans mitral**

**EICF- Exercise induced cardiac fatigue**

**CVD- Cardiovascular disease**

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## Chapter 1- Introduction

Exercise has been proven to be beneficial in maintaining a healthy cardiac system by improving general cardiac function whilst significantly reducing the risk of developing cardiovascular disease (CVD) (Pinckard et al, 2019). The American Heart Association recommends 150 minutes of moderate intensity exercise or 75 minutes of vigorous intensity exercise per week for adults to maintain a healthy cardiovascular system (Lee et al, 2017).

Although exercise is overwhelmingly positive for cardiac health, there is evidence that prolonged intense exercise leads to a transient reduction in right (RV) or left ventricular (LV) systolic and/or diastolic function, termed exercise-induced cardiac fatigue (EICF). It is important to note that EICF is generally considered a normal physiological phenomenon and has frequently been demonstrated in athletes after competing in endurance events such as marathons and ironman races. However, there is some evidence to suggest that repeated bouts of endurance exercise with minimal recovery may lead to maladaptation following EICF (Franklin et al, 2020). Our understanding of EICF has developed significantly over the past four decades due to improvements in echocardiographic techniques of cardiac assessment alongside a multitude of studies that have assessed the impact of duration, intensity and type of exercise. Studies have covered endurance exercises at moderate and high intensities looking at multiple sports such as running (Oxborough, 2011), cycling (Shave, 2004) and rowing (Neilan, 2006).

The focus of early EICF research was based on the LV, however, the literature has developed further to include a comprehensive assessment of RV and hence providing additional understanding of EICF and highlighting potential underlying mechanisms. Due to the RV having thinner myocardial walls and being paired to the pulmonary system (La Gerche et al, 2017), the RV appears to be more susceptible to EICF through structural and anatomical properties, potentially affecting the LV through serial or parallel impact.

Although there has been extensive research into the effects and mechanisms of EICF, almost the entirety of the literature is based upon terrestrial exercise with very limited attention being placed upon aquatic-based sport. Swimming is a widely popular sport which has numerous unique physiological demands due to its nature, including significant

periods of voluntary breath holding as well as full body engagement in a supine position alongside gravitational impact. Each of these separately have been shown to impact the cardiac system through alterations in pressures and volumes that could potentially have a greater impact on the RV. There has been research into triathlons and ironman races (Rifia et al, 1999, McGavock, 2003) which do contain a swimming component, however this is in combination with larger bouts of running and cycling exercise. Therefore, the impact of swimming as an individual sport has remained relatively unresearched.

It is unknown whether a change in the sporting environment would have an impact on EICF, however given the additional physiological pressures aquatic-based exercise imposes on the cardiac system (Riding et al, 2016) it is reasonable to speculate some augmentation on the nature and magnitude of EICF in this setting.

Therefore, this thesis aims to provide an assessment of the structural and functional cardiac response to a prolonged swim. The thesis consists of four main sections. Firstly, the literature review provides a background on the current understanding and methodologies underpinning EICF and the physiological demands of swimming as a sport highlighting how these demands may exacerbate the effects of EICF over a prolonged duration. This is followed by two empirical studies. The first study aims to establish the impact of a 30-minute and 60-minute swimming exercise on LV structure and function. The second study focuses on RV structure and function under the same conditions. This study also highlights any serial or parallel impacts the RV may have had on the LV. Finally, the thesis ends with a general discussion on the overall outcomes of the studies and how these findings build upon the current literature whilst considering the limitations and future directions.

## Chapter 2- Literature review

The cardiovascular system is responsible for the delivery of oxygen to every cell in the body as well as removing various waste products. The consequences of not maintaining a healthy cardiovascular system can be severe as highlighted by CVDs being the leading cause of noncommunicable death globally consistently since 2000, with them representing 32% of all global deaths in 2019 (World Health Organization: WHO, 2021). There is overwhelming evidence that regular moderate exercise benefits cardiovascular health (Myers, 2003, Joyner and Green, 2009, Lavie et al, 2015) and is recommended to reduce the risk of developing CVD. The American Heart Association and WHO guidelines recommend 150 minutes of moderate intensity exercise or 75 minutes of vigorous intensity exercise per week for adults to maintain a healthy cardiovascular system (Lee et al, 2017).

In an acute setting, exercise stimulates an increase in blood pressure and carbon dioxide concentrations within the contracting skeletal muscles, increasing metabolic demand. In response, these alterations are detected by baroreceptors and chemoreceptors which stimulate afferents, signalling the vagal centre in the medulla to reduce and increase parasympathetic and sympathetic outflow respectively to the sinoatrial node and inhibit vasoconstriction (Armstrong et al, 2023). This is known as the exercise pressor reflex, leading to an increase in blood pressure, ventilation and heart rate (Kaufman and Hayes, 2002). Heart rate (HR) is increased initially through a reduction in parasympathetic tone which allows for a decrease in blood pressure, causing a following reduction in parasympathetic tone to stabilise and later increase SV (White and Raven, 2014). As workload from exercise increases and HR exceeds 100bpm, sympathetic tone is further stimulated and parasympathetic is depressed to maintain a rise in HR (McConnell et al, 2014). The response from the exercise pressor reflex is intensity dependent, with higher intensities stimulating larger increases in heart rate and stroke volume (Teixeira and Vianna, 2022).

Stroke volume (SV) is increased due to the vasodilation, allowing for more blood to be in circulation. Preload, intrinsic contractility, and afterload all impact SV. Preload represents ventricular wall stress at the end of diastole, intrinsic contractility is the ability to produce force through myocyte contractions and afterload represents wall stress during systole

(Bruss and Raja, 2019). During exercise there is a greater venous return as more blood is being circulated to and from the muscles, resulting in a higher preload volume. Subsequently, the ventricles are forced to widen during diastole beyond the regular dimensions, lengthening the myocardial fibres. According to the Frank-Starling relationship this overstretching of the myocardial fibres is optimal for force production as it lengthens the sarcomeres and builds muscle tension, increasing intrinsic contractility and ultimately SV (Delicce and Makaryus, 2017). Afterload is the resistant pressure of the blood circulating through the vasculature as well as the chambers, meaning higher afterload results in lower SV. During exercise afterload decreases as the vasculature and chambers dilate to accommodate for the increase in blood volume, increasing SV.

**Exercise induced cardiac fatigue**

Athletes who participate in regular structured exercise display cardiac adaptations such as increased cavity dimensions and enhanced myocardial wall thickness which can improve blood circulation, allowing them to complete higher intensity/duration exercises. An athlete's heart is effective at responding to physical activity, however when medium to high intensity exercise is performed over a prolonged duration it can lead to reduced cardiac function. This phenomenon has been termed exercise-induced cardiac fatigue (EICF) and was initially published in 1987 by Douglas et al who determined the transient reduction in ventricular systolic and/or diastolic function was due to alterations in preload and impaired contractility (Douglas et al, 1987). The field has drawn a lot of attention due to clinical implications where repeated bouts of EICF may cause myocardial damage (O'Keefe et al, 2012), exacerbate existing cardiac conditions (D'Silva and Sharma, 2014) and in some cases sudden cardiac death (Albert et al, 2000). Over the past four decades an extensive portfolio of EICF has been documented through a wide array of studies focusing on different exercises, durations, intensities, and other additional physiological factors aided by advancing echocardiographic technology and methods.

**The left ventricle**

Early literature is focused on left ventricular (LV) structure and function as it was seen as the more 'important' side of the heart. Studies such as Douglas et al and Rifia et al looked at changes in LV dimensions, wall thicknesses, fractional shortening (FS) and ejection

fractions (EF) to highlight ventricular dysfunction (Douglas et al, 1987) (Rifia et al, 1999). Both studies observed twenty-one well trained athletes before and after taking part in the Hawaiian triathlon which consisted of a 3.9km swim, 180.2km cycle and 42.2km run completed in succession. Two-dimensional echocardiograms were conducted to obtain 4 chamber apical views 2-5days prior and immediately after the race. The combined results of both studies found a reduced end diastolic LV volume, FS and a significant drop of 24% in EF. It should be noted that the reduction in FS occurred in the absence of an increase in systolic blood pressure or wall stress. In Douglas et al a follow up scan was performed 28±9hrs after the post-race measurements to observe any dysfunction sustained during recovery. They found all measurements returned to baseline or close to pre-race levels except for end diastolic cavity size which remained reduced. Rifia et al provided little comment on the mechanisms that may be responsible for the drop in EF, however Douglas et al did suggest that the decreased FS despite the lack of change in SBP or wall stress indicated reduction in inotropy. In relation to the frank-starling mechanism, a reduced inotropy lowers stroke volume whilst raising LV end-diastolic pressures which is a clear sign of dysfunction immediately post exercise (Klabunde, 2021).

The methodology behind how EICF is measured has developed to be more detailed over time. Utilising EF to determine ventricular function has uses but there are numerous drawbacks. EF is heavily load dependent and so any changes in preload or afterload could impact the accuracy of the results. In addition, different results can be displayed across varying heart rates and beat to beat alterations. Result reproducibility is also an issue as inter and intra-observability of LVEF is high with a meta-analysis showing 13% of 5721 patients were reclassified from normal to mild LVEF in a clinical setting (Kalam et al, 2014). Global longitudinal strain (GLS) on the other hand is a powerful tool that encompasses LV deformation with strain analysis. During systole and diastole, the myocardium deforms in three planes of motion, longitudinal, circumferential and radial. These measured individually have proven fairly inaccurate due to regional noise, however GLS takes an average strain from multiple regions which reduces this noise and creates a reliable and sensitive marker for LV function (Potter and Marwick, 2018). The method also functions on a partially automated system which improves the reproducibility of data and reduces the variability in results between different observers (Kalam et al, 2014). As a result, GLS

has been used alongside EF in more recent studies to provide a more robust assessment of ventricular function (Schellenberg et al, 2023).

Another meta-analysis detailed the overall findings of studies that measured longitudinal, circumferential and radial strain across multiple exercise types and durations (Lord et al 2018). All exercises were endurance based and were >120mins in duration. All three strains have displayed significant reductions post exercise alongside systolic strain rates (SSR), however it should be considered that a limited number (n=4) of studies have documented radial and/or circumferential strains. LV twisting/torsion is a key mechanism that utilises energy stored during systole to enhance diastolic filling. As the ventricle relaxes it untwists and creates a rapid change in pressure gradient that acts as a vacuum, 'sucking' blood from the atria into the empty cavity. This mechanism relies on both the ability to twist but also the ability to maintain structural integrity. Multiple studies have shown that after prolonged exercise the degree of LV torsion is significantly reduced which directly impacts early diastolic filling (EDF) (Oxborough et al, 2011, La Gerche, 2012, Lord et al, 2016). This may be due to an inhibition of LV relaxation rates that impacts both the amplitude and speed of twisting. This reduction in EDF is compensated to a degree by an increase in atrial contraction as displayed by an increase in A velocity in the presence of reduced peak E velocities (Oxborough, 2010). However, this requires the LA to have a more active contribution to EDF which is not as effective or efficient as LV torsion. In summary, the literature shows prolonged exercise can result in EICF in the LV through increased myocardial strain, resulting in a reduction in function through impaired contractility and twist mechanics.

**The right ventricle**

In recent years however there has been a shift of focus onto the right ventricle. Although the literature displays dysfunction in both left and right ventricles there is a consensus that the right ventricle is more impacted proportionally than its larger counter part due to differing anatomical and functional properties (La Gerche, 2017). Anatomically, the RV has three main components which form a crescent shaped structure: the inlet, the trabeculated apical myocardium and the outlet/infundibulum (Sanz et al, 2019). The inlet is located at the apex of the heart and is made up of the tricuspid valve, tendinous cords and the supporting papillary muscles which prevent inversion or prolapse of the valve

cusps (Rich and Khan, 2022). The inlet is lined by trabeculae carneae, raised projections of contractile myocytes which become progressively more trabeculated towards the apical part. The trabeculated apical myocardium is comprised of multiple intersecting muscle bundles from which key structures arise including the moderator band and the parietal band (Wang et al, 2019). Despite the heavy trabeculation, the normal RV myocardium is 2-5mm in thickness which is significantly thinner than the LV myocardium which averages between 4.5mm and 8.3mm (Walpot et al, 2019) making it more vulnerable to high pressures/volumes. The RV also has a reduced capacity for contractility between 20-33% of that of the LV (La Gerche et al, 2014). Finally, the outlet/infundibulum is a smooth muscular tube/sleeve which is typically free of any trabeculations (Ho and Nihoyannopoulos, 2006) and provides support to the pulmonary valve. As the muscle attaches to the valve the wall thickness decreases to 1.5mm (Wang et al, 2019) and the size of this segment is independent from the rest of the RV, attributing roughly 20% of end-diastolic volume (Sanz et al, 2019). In previous studies it has been difficult to assess RV structure and function due to its anatomical position and complex internal structure. However, assessing RV longitudinal strain /strain rate using speckle tracking echocardiography has been highlighted as an effective and accurate way of determining RV function as it is sensitive to subclinical myocardial dysfunction in addition to a strong correlation with RV ejection fractions and overall function (Smolarek et al, 2017).

In addition to these anatomical differences the RV is also coupled with the pulmonary system which also functions slightly differently to the systemic system the LV is coupled with. The pulmonary system is a low pressure- high compliance system with a main function of allowing large volumes of blood to circulate to the lungs with as little resistance as possible. It consists of a larger abundance of branched vessels and the comprising arteries and arterioles have a lower basal tone and thinner walls compared to those in the systemic system (La Gerche et al, 2014). This allows for lower resistance through a broader vascular network and increased compliance in the vessels at rest. This does however mean that the pulmonary system is working at the lowest resistance possible at rest, which leads to some issues during exercise. When exercising both pulmonary and systemic pressures increase due to the increased blood circulation, however the pulmonary system has a reduced rate of vasodilation due to it already

functioning at such a low resistance (Oxborough et al, 2011). This results in a larger relative pressure increase in the pulmonary system compared to the systemic which the RV is exposed to.

As a result, a study looking into the relative changes in load between LV and RV during endurance/ultra endurance exercise found LV wall stress increased by 14% compared to a 125% increase in RV wall stress (La Gerche et al, 2011). The combination of a thinner myocardial wall and a relatively greater increase in pressure leads to disproportionate wall stress and afterload on the RV (La Gerche et al, 2017). Interestingly, there does appear to be adaptations in the RV to strenuous exercise where experienced marathon runners have a lower fractional area change compared to untrained counterparts. A study by La Gerche et al (2011) observed thirty-nine endurance athletes and fourteen age and sex matched nonathletes complete a marathon. There was an inverse correlation between finishing time and RV fractional area change suggesting a more effective response to the exercise in trained individuals.

Another study by Lord et al detailed the impact a 100mile run had on the RV from both an Echo and ECG point of view (Lord et al, 2015). Fifteen participants took part in the race and completed a pre and post cardiac assessment, nine of whom attended a follow up assessment six hours afterwards. From an echo perspective, the results showed a 10% increase in RV systolic area, a 10% decrease in RV FAC post-race and a correlating 10% reduction in RV lateral wall strain. In systole, RV strain was significantly reduced during the 60-100% portion of contraction. In early diastole (5-50%) there was a significant reduction in strain rate ($P<0.05$) and peak strain rate in 65-85% was significantly different. There was also a significant delay in deformation throughout causing a reduced and delayed early diastolic peak strain rate (SRE). In the athletes who were re assessed 6hrs after, both the reduced RV lateral wall strain and SRE were still present. From an ECG perspective there were increases in the prevalence of T wave inversion in V1 (47-80%), J point elevation in V1(27-60%), early repolarisation (33-53%), partial right bundle branch block (27-40%), lengthening QTc interval (0-6%) and a summated R wave in V1 and S wave in V5 (19%). These suggest that prolonged exposure to RV afterload from exercise leads to electromechanical delay and reduced RV function.

**Impact of exercise duration and intensity**

There is also some evidence that duration of exercise as well as intensity has an impact on ventricular structure and function. A study looked at the impact exercise duration and intensity had on patients with arrhythmogenic cardiomyopathy (Lie et al, 2018). Patients who took part in longer duration exercises reported reduced RV function in parallel with increased RV dilation when compared to shorter durations. It's been suggested that this is due to the RV having a limited capacity for maintaining function when workloads are increased, again linked to the disproportionate workload it is placed under compared to the LV (La Gerche, 2012).

The impact of exercise duration is further highlighted by La Gerche et al when they observed forty well trained athletes competing in varying endurance races (La Gerche et al, 2012). The events consisted of a marathon (7), endurance triathlon (11), alpine cycling race (9) and an ultra-triathlon (13) which took 3hrs, 5.5hrs, 8hrs and 11hrs on average to complete respectively. Echocardiographs looking at RV ejection fraction (RVEF), RV fractional area change (RVFAC) and RV global strain rate (RVGSR) were captured at baseline, immediately post-race and a week after completing the event. All events showed significant decreases post-race in these variables with the exception of RVGRS after the endurance triathlon which increased. The degree of RV dysfunction through reduced RVEF was inversely correlated with increasing race duration ($r=-0.501$, $P<0.0001$) which further contributes to this theory.

However, there is debate over whether duration has an impact on EICF. Hassan et al (2006) observed thirty-nine athletes complete an ironman triathlon consisting of a 3.8km swim, 180km cycle and a 42.2km run (226km total) which took 712 ± 96mins to complete. Post race analysis of LV function found no significant changes in EF or end systolic pressure/volume ratio (ESPV). This suggests that extended durations of exercise do not necessarily impact EICF. A further study by Coates et al (2020) observed fourty-nine recreational runners complete a 25, 50, 80 and 160km trail run. Results displayed a reduced LV diastolic function with decreased E/A ratio (1.9 ± 0.6 to 1.4 ± 0.5) and LV filling pressure E/e' (6.3 ± 1.9 to 5.6 ± 1.6). RV systolic function as TAPSE was decreased by 2.9 ± 0.4 to 2.7 ± 0.5, however RV FAC remained unchanged at 40.4 ± 6% to 38.1 ± 8%. There was no relation between running duration and any cardiac value. In addition, EICF has also been shown to occur over much shorter durations. Kleinnibbelink et al (2021)

analysed RV and LV function and mechanics of twenty-one athletes after 45mins of high intensity cycling. The results displayed a significant decrease in LV EF and LV GLS as well as RV FAC, TAPSE and RV FWS. This reduction in function across both ventricles over a relatively short bout of exercise further implies exercise duration may not be a determining factor in EICF. The study may also hint at intensity having a larger impact on EICF than duration (Donaldson et al, 2019).

The impact of exercise intensity has been documented by Banks et al who observed changes in both LV and RV function in 18 participants during two separate 150min cycle tests at 60% and 80% of maximal aerobic power (Banks et al, 2010). The study used echocardiographs to measure LVEF, ventricular strain and tissue velocities pre, 15mins into and post cycle. The results showed a significant decrease of 5.8% in LVEF (69.3%±1.3 to 63.5%±1.3) through a reduction in LVEDV (66.9±2.3ml to 52.9±2.7ml) at 80% intensity but non at 60% intensity. Similar findings were presented in the strain data with 80% intensity causing a 1.2% increase in LV S (-23.5%±0.6 to -22.2%±0.6) and 3.3% increase in RV S (-26.3%±0.6 to -23.0%±0.6) and 60% causing no significant changes. This suggests that at higher intensities both ventricles are more susceptible to strain, and that this is more prominent in the RV than the LV.

Another study comparing impact of intensity on cardiac function was conducted on ten recreationally active males with a 90min heavy intensity vs 120min moderate intensity cycle (Stewart et al, 2016). Participants completed both exercises which required the same overall mechanical and myocardial work. The results found the 90min cycle displayed reductions in both LV and RV GLS whereas the 120min cycle only resulted in impaired RV GLS during and 24hrs post exercise. In addition, the degree of RV systolic dysfunction was greater after the heavy intensity cycle which further supports the theory that exercise intensity has an impact of degree of EICF. It also supports the theory that the RV may be more susceptible to EICF than the LV as it is present in both exercises. Overall there appears to be opposing evidence on whether duration directly impacts EICF and that exercise intensity may have be a larger contributor.

**Ventricular inter-dependence**

There have been some studies that have highlighted an inter-dependence between the right and left side of the heart. A study by Oxborough et al examined seventeen healthy males prior to, immediately after and 6 hours post completing a marathon (Oxborough et al 2010). The aim of the study was to determine shifts LA mechanics and how that impacted LV function using myocardial speckle tracking. All participants completed the race and all three examinations. The results showed significant reductions in LA end systole (13%) and diastole (5%) volumes, LA anterior-posterior diameter (5%) and a subsequent depression in LA reservoir and conduit volumes (18% and 11% respectively) post-race. In addition, RVFAC was reduced by 9% and LV untwisting/torsion occurred both at a reduced rate and later during early diastole. The reduced rate and delay of untwisting in the LV suggests an impairment in LV relaxation which has a direct impact on LA deformation during early diastole as it alters the pressure gradient between the chambers, forcing the LA to take a more active role in LV filling as shown by a 40% increase in atrial booster pump volumes. The reduction in RVFAC and the correlating drop in LA and LA reservoir volumes suggests a decrease in RV stroke volume caused by increased pulmonary artery pressures that impacts LA filling. It was noted that further assessment of RV function would be required to confirm this.

There is additional evidence for an inter-ventricular dependence through the impact of strain on the septal wall. Stewart et al measured LV and RV function including strain before and after a 90min high intensity cycling trial in twenty-three recreationally active males (Stewart et al, 2017). Measurements were taken at rest and during a low intensity recovery cycle which controlled for post-exercise hypotension. The results found a reduction in both LV and RV GLS (LV: −18.4 ± 0.4% vs. −17.7 ± 0.4%; RV: −27.6 ± 0.7% vs. −26.4 ± 0.6%) at rest which was further enhanced during the low intensity recovery cycle (LV: −21.3 ± 0.4% vs. −19.2 ± 0.5%; RV: −28.4 ± 0.8% vs. −23.5 ± 0.9%). At rest, segmental longitudinal strain was decreased in RV free wall (4%), septum (4%) and anterior septum (3%) but no changes were found in the posterior or lateral segments. Again, during the low intensity recovery cycle these were augmented with RV free wall decreasing by 17%, septum by 12% and anterior septum by 9% whilst posterior and lateral remained unchanged. These alterations in septal and anterior septal correlated with the reduction in LV GLS as LV also displayed no alterations in lateral or posterior segments. The increase

in RV wall stress is translated through the septum, showing highest levels of myocardial strain at the insertion points of the RV into the intraventricular septum. This shows that RV dysfunction and the subsequent strain on the septum may lead to reductions in LV strain without a loss of LV myocardial contractility.

Another example would be when fifteen elite level runners took part in the study where they had an ECG and echocardiogram pre, post and 6 hours after a 100-mile race (Lord et al, 2016). The results found a reduction in both LV and RV longitudinal strain (9% and 10% respectively) and an impairment in LV filling. RV peak strain was reduced, and RV area increased, however RV SV remained the same suggesting alterations were due to an increase in blood volume as appose to RV dysfunction. In addition, there was no dysfunction found in the LA suggesting RV filling remained enhanced and stable. In contrast, LV did show signs of dysfunction with reductions in twist (46%), basal and apical rotation (39%) and systolic and diastolic twist rates (46%) in addition to a decrease in LV basal, mid and apical circumferential strain (19%, 14% and 15% respectively). This suggests that the LV dysfunction was a result of reduced myocardial relaxation leading to reduced filling and not due to RV dysfunction. There was a decline in afterload resulting from a reduction in LV wall stress and BP, however this was not accompanied by an increase in myocardial strain but a decrease further suggesting a loss of intrinsic function.

**Swimming**

Although the current depth of EICF literature is vast there are still some areas that are lacking in research, for example water-based exercise assessment. Swimming is a widely popular sport both as a standalone sport and as part of triathlon and open water. Swimming in each of these categories has multiple events for athletes to specialise in based on distance and stroke which naturally impacts the cardiovascular adaptations they will accrue. However, for the purpose of this project, we will consider swimming a distance aerobic based sport. At a basic exercise level, swimming is classified as a high isometric and moderate isotonic exercise (Mitchell et al, 2005) that requires the athlete to engage both their upper and lower limbs to propel themselves through water, putting it somewhere between rowing and running. To give an idea of what kind of training stimulus swimmers are subjected to, 18 elite level distance swimmers were observed over a regular swimming season and averaged 58km per week (Pollock et al, 2019). This in

comparison to a land-based exercises such as running is far shorter with elite level runners completing between 120-200km per week (Tjelta, 2016). With that said, swimming requires approximately four times the metabolic cost of running when distances are matched due to the use of multiple muscle groups and degree of resistance from drag (Riding et al, 2016), so the stimulus is rather similar.

Unlike other sports however there are some novel physiological and environmental challenges that impose additional pressures on the heart including water immersion, body position, breath holding and a change in gravity. Being immerged in water applies a compressive force on the body which causes a shift in internal pressure, causing blood from the limbs to be driven towards thoracic cavity (Lazar et al, 2013). This augmentation in venous return increases preload. Applying the Frank-starling mechanism, this increase in preload stretches the myocardial fibres and allows them to contract more forcefully, leading to an enhanced SV. This venous return is further enhanced by the supine body position the athletes swim in and the use of the legs to kick through the water. The combination of these factors dramatically increases SV, leading to a 30-60% increase in cardiac output (Riding et al 2016) which is a significant stress on the heart. There is some balance in this response however as the subsequent rise in blood pressure triggers baroreceptors that decrease heart rate. Water immersion also lessens systemic vascular resistance brought on by a combination of nitric oxide released in response to the hydrostatic pressure (Sun et al, 2004) and a lower catecholamine surge similar to that seen in runners (Guezennec et al, 1998) which may increase LV workload whilst swimming.

Swimming also includes voluntary breathe holding which has a couple of physiological impacts. A study was conducted on twenty-one divers to determine the impact of a maximal breath hold on cardiac function both in air and submerged in water (Marrabotti et al 2013). Measurements were taken at 1/3, 2/3 and at maximum breath hold. The results showed linear significant increases in LV volume ($P<0.001$), LV SV ($P<0.001$) and RV diameter ($P<0.001$) but a significant reduction in LV EF ($P<0.033$) for both conditions alongside an increase in early diastolic velocities ($P<0.005$) and a reduction in deacceleration time ($P<0.001$) during LV filling. This suggests that breath holding causes an impairment in LV filling despite enlargement in LV dimensions. Breath holding may also

put the athletes in the condition of intermittent hypoxia if durations are prolonged. Hypoxia impacts the systemic and pulmonary systems in different ways. It leads to a reduction in systemic vascular resistance which induces a decrease in LV afterload (Kleinnibbelink et al, 2020). On the other hand, it causes pulmonary vasoconstriction which naturally causes a rise in pulmonary artery pressures (PAP) and pulmonary vascular resistance (PVR). The resulting reduction in pulmonary vascular distension, when present in exercise, prevents vasodilation to combat the rising PAP, putting a disproportionate strain on the RV. As a result, hypoxia has been found to directly impact RV dimensions and increase systolic pressures (Naeije et al, 2013) so a build-up of recurring hypoxic phases may have a lesser but similar impact. There have been studies that suggest hypoxia doesn't have any additional impact on heart function if the exercise already stimulates cardiac fatigue. A study by Kleinnibbelink et al looked at the impact of hypoxia during a 45min high intensity run (Kleinnibbelink et al, 2021). Twenty-one participants ran in both a normal (20.9% oxygen in inspired air) and hypoxic (14.5% oxygen in inspired air) environment and a pre and post exercise echocardiogram was performed to measure RV and LV mechanics and function. They also performed a stress echocardiograph at pre and post, removing the disparities that occur with varying HR and sympathetic withdrawal. The results found significant reductions in RV s', RV fractional area change and RV free wall strain as well as LV EF and LV global strain in both conditions at pre and post which were more pronounced during stress echocardiographs. However, there was no significant difference between the degree of dysfunction in both the conditions. The only significant alterations exercising in the hypoxic environment resulted in was a larger RA size, shorter pulmonary acceleration time and a reduction in late diastolic uncoupling. This suggests that although conditions may cause dysfunction individually, they may not always lead to greater dysfunction that is already present.

The change in gravity mainly impacts the novel theory that the RV functions similar to a hydraulic ram (Sengputa, 2013). A hydraulic ram uses gravity to build kinetic energy as water flows down a supply pipe, the waste valve of which closes suddenly due to an increase in drag. When the valve shuts there is a rapid build-up of pressure which directs the water through a delivery pipe leading into a chamber with air. As water is forced into the chamber the air is compressed and eventually expands to force the water out through

an outflow pipe at a high pressure. When the pressure curves of both a hydraulic ram and the RV are observed there are compelling similarities that suggest the RV may function in the same way. With this in mind, the artificial change in gravity that swimming in water creates may impact the function of this mechanism.

There are limited previous studies looking at the impact of swimming as an individual sport on cardiac structure and function. Martinez et al analysed thirty-three athletes before and after completing a 9.5km open water race with a focus on the RV using echocardiography and measuring cardiac Troponin and leukocytes (Martinez et al, 2019). All participants completed the race with a mean finishing time of 174 ± 27.2 mins. The overall results displayed a dilation in RV but with no significant impairment to RV function or mechanics. However, it was noted that eight participants displayed a decrease in RV global strain. These athletes had larger apical RV segments than the others before the race and displayed larger basal dilation post-race. The swimmers were also equally divided between those who displayed decreased (n=16) and increased (n=17) SV post-race. The decrease in SV was the result of inadequate cavity dilation and showed a general tendency for lower RV deformation. The increased SV swimmers presented larger increases in RV size than their counter parts, suggesting a better adaptation during the exercise. RV dilation was present in the absence of LV dilation which supports the theory that there is a disproportionate load on the RV but that the stimulus was not sufficient to cause any dysfunction.

Like in the rest of the literature there are contrasting results. A study by Gajda et al observed fourteen well trained but non elite swimmers complete a 500km swimming relay in the Warta River in Poland over 91 hours (Gajda et al, 2019). Swimmers took turns completing 5km swims which took between 44:46mins and 60:02mins every repetition, completing roughly 35km each. Of the fourteen swimmers who took part twelve completed the required three echocardiograms conducted the day before the relay, immediately after completing their last swim and 48 hours after the event. The results indicated no signs of cardiac fatigue were present during or after the relay, with no decreases in any functional or mechanical indices in either ventricles or atria. In fact, 48 hours post exercise there was an increase in RV FAC, LVEF, myocardial systolic peak velocities and LV shortening fraction. This may be due to the conditions of the study as it

does not take into account the impact of river currents or long rest periods between relay legs.

Finally, there is also the potential impact of swimming-induced pulmonary edema (SIPE) characterised as varying degrees of dyspnea which can develop into coughing, chest pain or tightness, haemoptysis and eventually hypoxia (Smith et al, 2018). A meta-analysis looked at thirty-eight cases of SIPE across seventeen different articles (Grünig et al, 2017). Of the seventeen articles analysed, thirteen were focused on surface swimmers. The cause of each case varied based on the individuals status and environment but the vast majority of SIPE cases were brought on during either moderate-high intensity swimming or swimming in cold water. Of all thirty-eight cases, 79% reported a shortness of breath, 71% reported a cough and 68% displayed haemoptysis. Post SIPE analysis found subjects suffered from a degree of hypoxia with reduce oxygen saturation levels (73%) and unilateral/bilateral crackles (47%) which indicated excess fluid build-up in the lungs. It's been suggested that this may be caused by a combination of increased venous return, pulmonary artery pressures and cold-water immersion and has been linked to RV function as two athletes presented right side heart failure alongside pulmonary edema on land during a 90km running section of a triathlon (McKechnie et al, 1979). The majority of subjects returned to normal between 48hrs of recovery, but between October 2008 and November 2015 there were 42 deaths during the swimming section of triathlons, with 23 of the post-mortem cases pointing towards SIPE susceptible markers (Moon et al, 2016).

It is therefore important as highlighted by Claessen and La Gerche (2016) to assess the implications of these acute exercise changes at a clinical level, helping to underline any links between exercise-induced myocardial changes and damaging conditions such as arrythmias.

With all of this in mind, this project aims to provide an insight into how different durations of prolonged swimming impact heart structure and function with a key focus on the right ventricle and subsequent effects on the left side of the heart. The combined assessment of the structural and mechanical properties of the heart should provide an in-depth analysis of this and provide some insight into the underlying mechanisms that cause any observable alterations. There will also be a short assessment on lung function to determine if any symptoms of SIPE occur during the tests. Although the limited previous

literature suggested prolonged swimming does not usually display signs of dysfunction within the ventricles, we hypothesise that there will be evidence of EICF in both ventricles due to the intensity and duration of the exercise displayed by significant reductions in systolic and/or diastolic functional indices.

## Chapter 3- General methods

As both of the following empirical studies are drawn from the same data collection, this chapter will detail the shared methods of both studies. Ethics approval was obtained from the ethics committee of Liverpool John Moores University (REC reference: 23/SPS/005)

### Sampling

Using data from Qasem et al (2018) on RV structural and functional indices a statistical sample size estimation test was conducted and the sample size required was seventeen. Participants were recruited from the city of Liverpool performance swimming squad and the university of Liverpool and Liverpool John Moores university swimming teams through a recruitment poster (See appendices). Participants were required to be between the ages of 16-35yrs and partake in a minimum of 3 hours structured swimming training on a weekly basis. Participants were excluded from the study if they had a history of CVD, were pregnant, diabetic or were currently taking cardiovascular medication. Fifteen participants (11 male swimmers and 4 female swimmers aged 20 ± 2 yrs, height:178 ± 22cm, body mass:74.2 ± 14.2kg) were recruited. Participants had been training for an average of 5±1yrs and completed 11±5hrs of structured swimming training (swim and gym) per week, with a minimum of 3 hours swimming. Participants were predominantly white Caucasian (n=14) with a single athlete being of Asian ethnicity.

### Design

Each participant completed a repeated measures test where they attended an hour session and a separate two-hour session within two weeks of each other at the Wavertree aquatics centre. In these sessions the participants had a standard 12-lead ECG and echocardiogram before and after (following 5 minutes of rest) conducting a prolonged (30min or 60min) swim at moderate intensity (i.e. a pace that could be held throughout the duration of the swim). All participants refrained from exercise at least 6 hours before the intervention and did not consume alcohol or caffeine 24 hours prior to the intervention. Upon arrival the participants were briefed on the design, procedures and expectations and provided informed consent.

### Procedures

Upon arrival, participants were given a brief on what the aims of the study were and what was involved. They were given a participant information sheet and asked to complete a basic cardiovascular health questionnaire (see appendices) and sign a written consent form. Participants then had anthropometrics recorded using standard calibrated scales (Seca 803 Clara, HaB direct, Birmingham, UK) and a stadiometer (Stadiometer, HaB direct, Birmingham, UK). Manual blood pressure was recorded using a cuff and stethoscope. In addition, each participant completed a spirometry test where they completed three maximum effort exhales, and their average forced voluntary expiration in 1s (FEV1) and forced vital capacity (FVC) were recorded.

The participants then underwent a standard 10-second, 12 Lead Resting ECG (Seca CardioPad 2, Seca UK, Birmingham, UK) in a supine position to exclude any undiagnosed pathologies. This was followed by a resting transthoracic echocardiogram conducted by one of two experienced sonographers on a commercially available ultrasound system (Vivid IQ; GE Medical; Horten, Norway). Participants were asked to lay in a left lateral decubitus position and all images were acquired in accordance with British Society of Echocardiography guidelines (Robinson et al 2020). Images were collected in the following order: parasternal long axis, parasternal short axis, apical four chamber, apical two chamber, apical three chamber and sub-costal and supra-sternal. From these images 2D, Doppler, tissue Doppler imaging (TDI) and m-mode were collected. Images were also optimised with frame rates (40-90 frames per second (FPS)), depth and gain to maximise endocardial delineation for STE of both LV and RV. Analysis of the images was conducted offline using commercially available software (EchoPac version 6.0; GE Medical, Horten, Norway) by the researcher. The specific echocardiographic measurements are detailed in the relevant empirical study chapters.

Participants were then asked to complete either a 30min or 60min continuous swim at a comfortable pace based on their individual ability. Pool conditions were maintained for all participants with the pool being long course (50m) at a constant depth of 1.8m and an average water temperature of 27°C. All swims were front crawl based, and participants were required to swim continuously with occasional brief breaks to take on fluid (ad libitum). If participants did stop the time was paused for the duration until the participant began the exercise again. Distance was recorded by the researcher using a tally method.

After completing the swim participants were asked to dry themselves with a towel and rested for 10mins before the process was repeated in the same order as previously detailed.

**Statistical analysis**

All data are presented as mean ±. SD. Statistical analyses were performed in SPSS (IBM, New York, USA) to establish normal distribution using a Kologorov Smirnov test and where appropriate a 2-way within subjects ANOVA (condition vs time) was utilised to compare pre and post readings of key measurements across the 30min and 60min swims. ANOVAs included descriptive statistics, estimated marginal means and comparison of main effects adjusted to the Bonferroni confidence interval. In the results, pre and post is referred to as condition and time relates to 30min or 60min swim. A Pearson's correlation analysis was performed on the delta values of significant structural and functional indices between the LV and RV. No statistically significant correlations were found between any relevant indices. All data was normally distributed. Statistically significant changes were reported with a 95% confidence interval ($P \leq 0.05$). Ten pre and post data sets were additionally analysed by an experienced sonographer and a coefficient of variance was calculated to determine inter-observer variability. This was done to aid analysis comprehensiveness. All data was within 10% variance between observers.

## Chapter 4- Impact of prolonged swimming on the left ventricle

Left ventricular structure and function has been the focus of the majority of the previous literature as discussed in Chapter 2. These studies have demonstrated that over prolonged periods of moderate-high intensity exercise there are signs of a reduction in LV function through reduced diastolic function, GLS and ejection fraction (EF). This has been recorded over multiple land-based sports such as cycling and running with a focus on endurance to ultra endurance distances/races (Oxborough et al, 2010) but there is little based on solely swimming. Exercising in a supine position along with external hydrostatic pressure increases venous return, which may alter preload/afterload (Lazar et al, 2013). When this is combined with a prolonged bout of moderate intensity exercise that utilises multiple muscle groups there is potential for the RV to be subjected to a greater volume and pressure-based stimulus. The subsequent study is conducted on a 30min and 60min swim. Although it may not appear to be as demanding as previous events such as marathons or ironman triathlons, the demands of swimming are far greater than land-based exercise with the metabolic cost being four-fold that of running when intensities and distances are matched (Riding et al, 2016). The aims of this study therefore are to determine the effect of prolonged swimming from different durations, on LV structure and function. This aim leads to the specific hypothesis based on the physiological challenges that swimming poses in addition to the duration of the exercise will result in EICF, leading to measurable reductions in function and/or an alteration in LV structure. We also hypothesise that the degree of dysfunction will be greater in the 60min swim compared to the 30min swim.

### Methods

The specific study design, sample population and protocol are detailed in Chapter 3.

### Left sided echocardiogram protocol

All measurements were made in accordance with British Society of Echocardiography guidelines (Wharton et al, 2015). Left ventricular structure was determined by end diastolic diameter (LVED), and septal (IVSd) and posterior wall thicknesses (PWDd) were captured in a parasternal short axis view. LV end-diastolic dimensions, LVEDV and LVESV were measured from an apical four-chamber view. Pulsed wave Doppler in apical three and four-chamber views were conducted to measure LV diastolic function. LVEF was

calculated using the Simpsons biplane method to derive all volumes including the left atrium. Tissue doppler was also implemented on the apical four-chamber view through a 2mm sample volume placed on the septal aspect of the mitral valve annulus to measure early diastolic myocardial velocity (E'). This was repeated on the lateral wall. LVGLS was measured from the apical four-chamber, two chamber and three chamber view using the EchoPac software. After highlighting the endocardial border of the LV, the software automatically traces the border and divides it into six sections making a total of eighteen segments, this can then be adjusted manually either through changing the range of interest to determine wall thickness or altering the contour to ensure optimal tracking (Lee and Park, 2018).

**Results**

Fifteen swimmers completed the 30min swim and thirteen completed both the 30min and 1hour swims with pre and post exercise measurements. Unfortunately, strain data from two participants was unusable due to suboptimal image quality, leaving eleven usable data sets. All results were displayed as means ± standard deviation (SD) and presented in table 1.1 to 1.3. Significant differences were reported as ($P \leq 0.05$) and separately displayed in graphs. Subjects swam 1950m±250m in the 30min session and 3800m±350m in the 60min session.

There were no significant changes to height or weight post swim over either duration ($P > 0.05$). Heart rate (HR), systolic blood pressure (SBP) and diastolic blood pressure (DBP) significantly increased post-swim for both durations ($P < 0.01$, $P = 0.07$ and $P = 0.03$ respectively). There were no significant changes to FVC or FEV1 over either duration ($P > 0.05$).

| Parameter | 30min | | 60min | | P Value | | |
|---|---|---|---|---|---|---|---|
| | Pre | Post | Pre | Post | Condition | Time | Interaction |
| Height (cm) | 178 ± 10 | 178 ± 10 | 181 ± 6 | 181 ± 6 | N/A | N/A | N/A |
| Weight (kg) | 74.2 ± 9.1 | 74.1 ± 9 | 74.3 ± 9 | 74.3 ± 8.9 | 0.44 | 0.60 | 0.49 |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| Heart rate (bpm) | 66 ± 10 | 91 ± 13* | 67 ± 11 | 91 ± 15* | 0.001* | 0.76 | 0.17 |
| SBP (mmHg) | 116 ± 12 | 120 ± 13* | 115 ± 8 | 126 ± 14* | 0.007* | 0.20 | 0.09 |
| DBP (mmHg | 65 ± 9 | 71 ± 10 | 69 ± 8 | 71 ± 7 | P=0.89 | 0.66 | 0.71 |
| FVC (L) | 5.3 ± 0.9 | 5.5 ± 0.7 | 5.5 ± 0.6 | 5.3 ± 0.5 | P=0.83 | 0.69 | 0.04* |
| FEV1 | 4.6 ± 0.6 | 4.6 ± 0.6 | 4.6 ± 0.7 | 4.6 ± 0.6 | P=0.53 | 0.86 | 0.97 |

**Table 1.1** anthropometrics, blood pressure and spirometry data. *Denotes P≤0.05

There were no significant changes to LV structural indices with exception of for LVEDV (see table 1.2) There was a significant main effect for condition ($F_{1, 12}$=42.85, P<0.001). Overall, the pre-exercise LVEDV was higher than post exercise. There was no significant main effect for duration ($F_{1, 12}$=0.79, P=0.39). There was no significant interaction ($F_{1, 12}$<0.001, P=0.99).

| Parameter | 30min | | 60min | | P Value | | |
|---|---|---|---|---|---|---|---|
| | Pre | Post | Pre | Post | Condition | Time | Interaction |
| LVd (mm) | 50 ± 4.6 | 49 ± 5.1 | 49 ± 4.9 | 48 ± 4.3 | 0.10 | 0.40 | 0.45 |
| LVs (mm) | 33 ± 4.6 | 32 ± 5.1 | 33 ± 3 | 33 ± 3.8 | 0.49 | 0.77 | 0.42 |
| LVEDV (ml) | 142 ± 28 | 130 ± 31* | 141 ± 36 | 128 ± 33* | <0.001* | 0.39 | 0.99 |
| MWT | 8.2 ± 1.1 | 8.6 ± 0.9 | 8.2 ± 1.1 | 8.2 ± 1.1 | 0.25 | 0.36 | 0.19 |

**Table 1.2** highlights the main structural indices of the LV. *Denotes P≤0.05

There were significant reductions in LV function (see table 1.3). In LVEF, there was a significant main effect for condition ($F_{1, 12}$=40.22, P<0.001). Overall, the pre-exercise LVEF was higher than post exercise (see figure 1.2). There was no significant main effect for

duration ($F_{1, 12}$=1.56, P=0.24). There was no significant interaction ($F_{1, 12}$=48.08, P=0.19). In LVGLS, there was a significant main effect for condition ($F_{1, 10}$=51.30, P<0.001). Overall, the pre-exercise LVGLS was higher than post exercise (see figure1.3). There was no significant main effect for duration ($F_{1, 10}$=4.78 P=0.32). There was no significant interaction ($F_{1, 10}$=1.9, P=0.2). In trans mitral deceleration time (TMD), there was a significant main effect for condition ($F_{1, 11}$=9.67, P=0.01). Overall, the pre-exercise TMD was lower than post exercise (see figure 1.4). There were no significant changes in any remaining functional indices and duration did not impact any indices. (see table 1.3)

| | 30min | | 60min | | P Value | | |
|---|---|---|---|---|---|---|---|
| Parameter | Pre | Post | Pre | Post | Condition | Time | Interaction |
| LVESV (ml) | 64 ± 13 | 70 ± 18 | 65 ± 21 | 68 ± 25 | 0.13 | 0.99 | 0.72 |
| LVEF (%) | 57.1 ± 3.5 | 50.2 ± 4.4* | 57.2 ± 3.8 | 54.7 ± 5.2* | ≤0.001* | 0.24 | 0.19 |
| LVGLS (%) | -19.1 ± 1.8 | -16.7 ± 1.7* | -18.2 ± 1.9 | -16.5 ± 1.5* | ≤0.001* | 0.32 | 0.20 |
| TME (m/s) | 0.84 ± 0.13 | 0.77 ± 0.13 | 0.78 ± 0.16 | 0.74 ± 0.15 | 0.14 | 0.24 | 0.88 |
| TMA (m/s) | 0.42 ± 0.09 | 0.54 ± 0.16* | 0.47 ± 0.09 | 0.59 ± 0.16* | 0.03* | 0.36 | 0.72 |
| TMD (m/s) | 198 ± 32 | 152 ± 52* | 174 ± 31 | 132 ± 41* | 0.01* | 0.009* | 0.62 |
| M S' (mm/s) | 10 ± 2.4 | 10 ± 2.5 | 11 ± 2.3 | 11 ± 2.4 | 0.40 | 0.86 | 0.41 |
| M E' (mm/s) | 13 ± 3 | 14 ± 3 | 14 ± 2.8 | 13 ± 3.5 | 0.66 | 0.77 | 0.18 |
| M A' (mm/s) | 9 ± 2.6 | 6 ± 1.8 | 8 ± 1.6 | 9 ± 1.9 | 0.49 | 0.05* | 0.002* |
| L S' (mm/s) | 13 ± 3.2 | 11 ± 1.8 | 13 ± 3.4 | 12 ± 2.7 | 0.03* | 0.52 | 0.74 |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| L E' (mm/s) | 19 ± 3.9 | 18 ± 3.1 | 17 ± 4.4 | 16 ± 4.1 | 0.19 | 0.71 | 0.77 |
| L A' (mm/s) | 7 ± 1.3 | 6 ± 1.9 | 7 ± 1.7 | 6 ± 1.8 | 0.091 | 0.93 | 0.93 |

**Table 1.3** displays all functional indices of the LV. *Denotes P≤0.05.

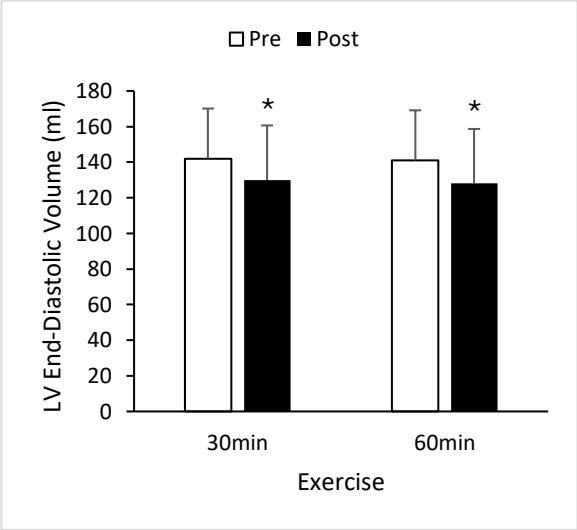


**Figure 1.1** impact of both 30min and 60min swims on LVEDV. * Denotes P≤0.05 for condition.

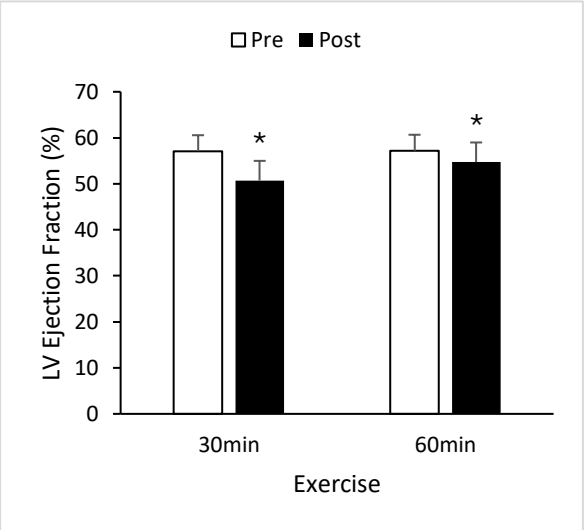


**Figure 1.2** impact of both 30min and 60min swims on LVEF. * Denotes P≤0.05 for condition.

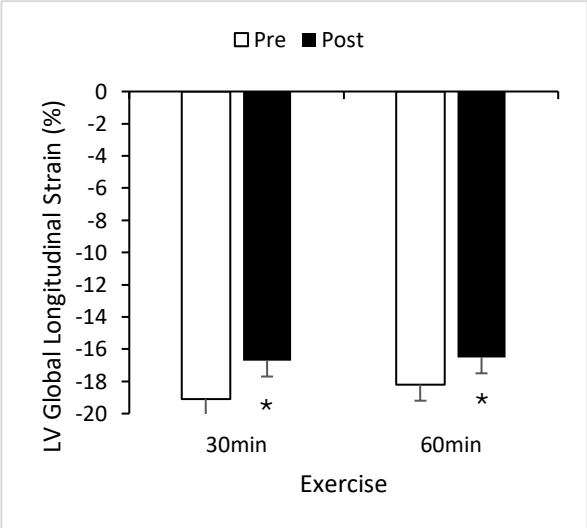


**Figure 1.3** impact of both 30min and 60min swims on LVGLS. * Denotes P≤0.05 for condition.

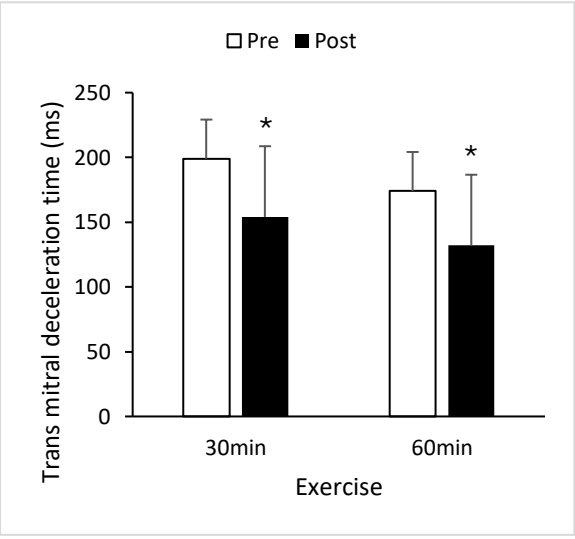


**Figure 1.4** impact of both 30min and 60min swims on TMD. * Denotes P≤0.05 for condition.

**Discussion**

The key findings of this study were that after both 30mins and 60mins of moderate intensity swimming exercise there was 1) a significant reduction in LVEF suggesting the presence of EICF, 2) a significant reduction in LVEDV but with preserved LVESV, 3) a reduction in LV function through an increase in LVGLS and, 4) there was no significant impact of duration on any indices.

The results show an alteration in both structure and function indicative of EICF after both durations of swimming exercise. The reduction in LVEF and LVGLS show a significant negative impact on LV systolic function and the reduced LVEDV displays an altered ventricular structure during diastole. This correlates with the current literature for land-based exercise to a degree. LV systolic function has been shown to decrease through myocardial fatigue post prolonged exercise (Lord et al, 2018), however fatigue during systole is usually preceded by fatigue during diastole as the initial stretch determines contraction capabilities according to the Frank-Starling mechanism (Delicce and Makaryus, 2017). The data shows no evidence of diastolic dysfunction, only a reduced blood volume which may be affected by other mechanisms as detailed later. There was no significant evidence that duration impacted the degree of dysfunction present which aligns with the works Haasan et al (2006) of but opposes those of La Gerche et al (2006) as discussed in Chapter 2. Studies looking into the impact of exercise duration and intensity generally display a correlation between degrees of dysfunction and increasing exercise duration/intensity (Donaldson et al, 2019). We can determine from the average distance covered in both swims that the participants-maintained exercise intensity as the 60min distance is almost double that of the 30min (1950m vs 3800m), yet there was no significant difference in the dysfunction present between the two durations. Therefore, this study supports the theory that duration may not be a contributing factor. It also highlights that intensity may have more of an impact on EICF than duration (Classen and La Gerche, 2016). This suggests that once EICF occurs after a certain duration the degree of dysfunction is maintained unless there is an alteration in intensity. However, it does also show that the dysfunction post swim is present over both durations exercised (30min vs 60min).

There was also no impact on lung function from either duration of swimming exercise as shown by the sustained FEV1 and FVC meaning SIPE was not induced. Although it is uncommon to observe SIPE in general, this may be due to the temperature of the water and the location of the testing. SIPE is most commonly observed in open water swimmers where the water conditions are cold and with naturally more waves, creating a more difficult environment to breathe normally than in a standard swimming pool (Grünig et al, 2017).

A significant finding is the degree of dysfunction that occurs after such a short duration of exercise. In previous land-based literature, the majority of exercise durations that cause EICF are far greater than 30mins with the literature revolving mainly around ultra-endurance events such as long-distance cycling, marathons or ironman triathlons. Although the metabolic demand of swimming exercise is considerably larger (Riding et al, 2016), these events require multiple hours to complete at the same intensity if not higher. Oxborough et al (2011), demonstrated that after completing a 161km ultramarathon LVGLS was reduced by 2% (-18.3% to -16.3%) and LVEF decreased by 5% (65% to 60%) which is similar to the 2.4% (-19.1% to -16.7%) and 6% (57% to 51%) decrease respectively after 30mins of swimming. This relatively similar degree of LV dysfunction between a 1900m swim and a 161km cycle suggests that the physiological challenges of swimming exercise exacerbate the effects of EICF as hypothesised. There are some studies that look at land-based exercise over a shorter duration such as Kleinnibbelink et al who observed EICF after 45mins of high intensity running (Kleinnibbelink et al, 2021). In a resting post exercise echo, LVGLS was unchanged (-20%) and LVEF decreased by 2% (58% to 56%) which then reduced by 2% (-23% to -21%) and 5% (63% to 58%) respectively during a stress echo. This study further supports swimming exacerbating EICF as the shorter duration and lower intensity swim still resulted in greater reductions in LVGLS and LVEF, only becoming comparable between a rested echo post swim and a stress echo post run.

Although the results comply to a degree with the current literature on land-based cardiac fatigue, they contrast with the previous literature focused on swimming. Gajda et al who measured LVEF and LV fractional shortening after a 500km relay swim found only increases in LV function with pre LVEF being (64.9%), peak effort (64.2%) and recovery 48hrs post (71.6%) showing no signs of dysfunction (Gajda et al, 2019). However, this may

be due to the environment in which the study took place. Swimmers completed shifts of 5km swims in the Warta River in Poland, posting times between 44:46mins and 60:02mins per swim. Given in the 2022 world aquatics championships the 5km gold was won in a time of 52:48mins and the swimmers in this study were described as 'non elite' swimmers aged 16-67yrs, it is reasonable to suggest the swimmers were aided by the current of the river. This potentially reduced the intensity/duration of each swim. The 'shift' method of the relay may have also contributed to this. Each swimmer took turns to complete the 5km shifts, meaning they had long periods of recovery between bouts. In addition, only one echocardiograph was collected for each swimmer at the end of their last shift and not after each 5km bout. This potentially means only the impact of the final 5km of each participant was observed, perhaps explaining why no signs of dysfunction were found after a much greater workload than the present study.

**Mechanisms**

Potential mechanisms contributing to the EICF in this case would be 1) an increase in afterload raising vascular resistance, 2) fatigue within the LV myocardium causing a loss of inotropy and/or, 3) a reduction in preload either brought on by myocardial fatigue or reduced ventricular filling.

Afterload represents the relative resistance the ventricles need to overcome to maintain blood flow (Vest, 2019), with increased SBP and DBP creating a larger pressure gradient for the ventricle to overcome. This is partially shown by the correlation between increased DBP and reduced function in LVGLS after the 60min swim. Although, when isolated, this would result in a reduced EF and LVGLS, there would also be signs of ventricular dilation with increased LVESV and LVEDV due to the increase in venous return (Klabunde, 2004). The results found LVESV remained unchanged post exercise and LVEDV decreased, suggesting that although afterload may have contributed to LV systolic dysfunction it was unlikely to be the primary source of EICF.

A loss of inotropy within the LV myocardium could have affected ventricular stiffness, reducing systolic and/or diastolic function which synergises with the reduced EF, LVEDV and LVGLS. Hart et al observed fourteen trained individuals complete a marathon (Hart et al, 2006) and found evidence of reduced LV compliance during diastole. Doppler data

displayed E'/A' ratio, E/A ratio and early filling flow propagation velocity were decreased by 32%, 31% and 24% respectively, leading to reduced LV filling. The ventricular stiffness would also impact both LV dilation and twisting, reducing the degree of 'suction' of blood from the LA to into the LV further impairing ventricular filling (Lord et al, 2018). In this study however, the tissue Doppler data showed no significant reductions in tissue velocities from E' or A' and a decrease in trans mitral deceleration time which indicates a relative increase in diastolic function. The results provide no evidence for LV diastolic dysfunction other than a reduced diastolic volume. Although there may have been some systolic myocardial fatigue it is unlikely as this is preliminary to diastolic myocardial fatigue.

It is well established that preload directly impacts LV structure/function as higher preload stretches the myocardial fibres for increased force and therefore stroke volume (Delicce and Makaryus, 2017). The reduction in LVEDV indicates a reduced preload, potentially contributing to the reduced LV systolic function through overall smaller chamber size and decreased myocardial stretch. Preload is determined by the amount of blood in circulation through the ventricles, which can be impacted by multiple variables. There is evidence that suggests dehydration could cause a reduction in preload. Watanabe et al (2020) looked at the impact of dehydration on stroke volume and cardiac output during two hours of cycling using echocardiograms to measure ventricular indices and volumes. Eight trained cyclists and triathletes were recruited to take part in two separate two hour cycling tests at 50-55% of their maximal work rate, one in a hydrated state and one in a progressively dehydrated state. The study found a significant reduction in stroke volume in the dehydrated condition caused solely by a reduction in LVEDV (-31±4ml vs euhydration) as LVESV remained stable. It was proposed that the dysfunction was caused by a lower preload and that the source of this was from a reduced venous return and tachycardia-induced shortening in LV filling duration rather than a loss of diastolic/systolic function. Although the results of this study display a similar outcome to the current study, the ventricular dysfunction post swim occurs after just 30mins of exercise in a relatively cold environment which makes it highly unlikely that dehydration was a contributing factor to LV impairment in this case. Another possible mechanism would be LV dysfunction present was impacted by a preliminary reduction in RV function. A study by Lord et al

demonstrated reduced peak RV strain; RV longitudinal strain and RV dilation had a serial impact on LV filling after completing a 100-mile ultramarathon (Lord et al, 2016). They determined that reduced RV contractility was a result of altering RV structure as appose to myocardial dysfunction, potentially leading to underfilling of the LV accompanied by reduced LV relaxation and twist/untwist. Previous literature has detailed reductions in LV dimensions with preserved indices of LV systolic function post strenuous exercise in the presence of reduced RV function/dimensions (Naije et al, 2017).

These potential contributing factors are interdependent, with alterations in any individual factor having a serial impact on the remaining (Klabunde, 2004). However, we can speculate that a reduced preload from underfilling may have been the driving factor due to the reduced LVEDV in the absence of diastolic myocardial fatigue or reduced LVESV.

**Conclusion**

To conclude, 30mins and 60mins of moderate intensity swimming exercise leads to left ventricular EICF in the form of reduced systolic function and reduced diastolic volume, the degree of which is similar to land-based exercises that require considerably more workload. Given the short duration over which the dysfunction in this study has occurred it is reasonable to speculate the environmental and physiological challenges of swimming have exacerbated the effects of EICF. The EICF had multiple contributing factors, but a reduced preload potentially brought on by a preliminary failure in RV mechanics may be the driving factor. The additional challenges, particularly the change in gravity, voluntary breath holding and the supine positioning, have the potential to impact the RV more due to its susceptibility to changes in pressure and volume. Therefore, the next chapter will focus solely on the RV, any signs of dysfunction there in and how the dysfunction has a serial or parallel impact on LV filling.

## Chapter 5- Impact of prolonged swimming on the right ventricle

The primary area of focus for this study is to observe any alterations that occur to the RV both functionally and structurally after prolonged swimming. From a physiological perspective, the RV is more susceptible to changes in pressure and volume due to a relatively thinner myocardium and it's pairing with the pulmonary system (Oxborough et al, 2011). The thinner myocardial wall produces less contractile force than the LV myocardium and so is less able to cope with increases in blood volume/ pressure brought on by exercise (La Gerche et al, 2014). The pulmonary system is also unable to adapt to increases in pressure compared to the systemic system as it has little margin for dilation to reduce vascular resistance (Oxborough et al, 2011). The combination of these factors leads to the RV being subjected to relatively higher workloads than the LV, making EICF more likely to be present during land-based exercise. Swimming adds multiple challenges which directly impact the RV through raising both volume and pressure. Exercising in a supine position increases venous return, raising blood volumes that may increase work for the RV (Lazar et al, 2013). Pulmonary artery pressure is also increased due to the voluntary breath holding which could again put the RV under more strain (Riding et al, 2016). Finally, the theory that the RV utilises a hydraulic ram system to aid function relies heavily on gravity, which is artificially altered when exercising in water (Sengputa et al, 2013). This would force the RV to take a more active role in maintaining function, perhaps leading to an early onset of EICF. The results of the LV study would suggest that the RV has been affected by EICF and that the changes in structure and function were linked between the ventricles. The aim of this study therefore is to determine what effect prolonged swimming has on right ventricular structure and function, if there is evidence of dysfunction and whether there is any association to changes in the LV. The hypothesis of this study was that due to the physiological challenges swimming poses in combination with the extended duration of the swimming exercise, there would be a measurable degree of RV dysfunction through significantly reduced RVGLS/RVFWS and/or RV volumes which had a serial impact on LV filling.

### Methods

The specific study design, sample population and protocol are detailed in Chapter 3.

**Right sided echocardiogram protocol**

RV focused apical four-chamber view was used to determine RV size at inflow and a parasternal long axis view was used to assess the outflow tract (Zaidi et al, 2020). RV areas were calculated by tracing around the endocardium during peak end-diastole and end-systole which were later used to determine RV fractional area change (RVFAC) using the calculation (RVEDA-RVESA) (Oxborough et al, 2011). Tricuspid annular plane systolic excursion (TAPSE) was measured from the lateral tricuspid annulus to assess longitudinal RV systolic function by measuring the distance between end diastolic and peak systolic points (Lee and Park, 2018). RV free wall strain (RVFWS) and RV global strain (RVGLS) were measured from the RV focused apical four-chamber view using the EchoPac software. After highlighting the lateral, septal and apex of the tricuspid annulus the software automatically traces the endocardial border and divides it into six sections, this can then be adjusted manually either through changing the range of interest to determine wall thickness or altering the contour to ensure optimal tracking (Lee and Park, 2018). RV pulse-wave tissue doppler was also implemented using a 2mm sample volume within the tricuspid annulus to assess tissue velocities including R S', E' and A' respectively (Oxborough et al, 2011).

**Results**

All results were displayed as means ± standard deviation (SD) and presented in table 2.1 and 2.2. Significant differences were reported as ($P \leq 0.05$) and separately displayed in graphs.

There were no significant changes in RV structure post exercise except for RVDA. There was a significant main effect for condition ($F_{1, 12}=6.21$, $P=0.028$). Overall, the pre-exercise RVDA was higher than post exercise. There was no significant main effect for time ($F_{1, 12}=1.66$, $P=0.22$). There was no significant interaction ($F_{1, 12}=0.33$, $P=0.58$).

| Parameter | 30min | | 60min | | P Value | | |
|---|---|---|---|---|---|---|---|
| | Pre | Post | Pre | Post | Condition | Time | Interaction |
| RVOT PLAX (mm) | 30 ± 5.1 | 29 ± 6.5 | 29 ± 5.1 | 30 ± 4.6 | 0.74 | 0.67 | 0.45 |
| RVOT1 (mm) | 28 ± 3.6 | 29 ± 5.6 | 28 ± 4.5 | 27 ± 5.9 | 0.75 | 0.40 | 0.38 |
| RVOT2 (mm) | 20 ± 4.3 | 19 ± 3.1 | 21 ± 3.8 | 22 ± 5.3 | 0.54 | 0.43 | 0.46 |
| RVD1 (mm) | 37 ± 5.7 | 37 ± 5 | 36 ± 4.2 | 36 ± 5 | 0.33 | 0.17 | 0.53 |
| RVD2 (mm) | 28 ± 5.1 | 28 ± 6.3 | 27 ± 3.5 | 26 ± 3.4 | 0.84 | 0.14 | 0.34 |
| RVD3 (mm) | 76 ± 9.3 | 75 ± 11 | 78 ± 8.1 | 77 ± 10.5 | 0.43 | 0.29 | 0.83 |
| RVDA ($mm^2$) | 23.5 ± 3.9 | 22.5 ± 5.6* | 23 ± 4.6 | 22 ± 4* | 0.028 | 0.22 | 0.60 |
| RVSA ($mm^2$) | 14.1 ± 4.6 | 12.8 ± 3.7 | 12 ± 2.4 | 13 ± 2.7 | 0.27 | 0.11 | 0.07 |
| IVC (mm) | 18 ± 4 | 18 ± 3.2 | 18 ± 4 | 17 ± 2.8 | 0.83 | 0.87 | 0.62 |

**Table 2.1** structural indices taken from the right side of the heart. * Denotes P≤0.05.

There were significant changes to RV function post exercise. In RVFAC, there was a significant main effect for condition ($F_{1, 12}$=23.94, P<0.001). Overall, the pre-exercise RVFAC was lower than post exercise (see figure 2.1). There was no significant main effect for time ($F_{1, 12}$=0.003, P=0.96). There was no significant interaction ($F_{1, 12}$=1.17, P=0.3). In RVDA, there was a significant main effect for condition ($F_{1, 12}$=6.21, P=0.028). Overall, the pre-exercise RVDA was lower than post exercise (see figure 2.2). There was no significant main effect for time ($F_{1, 12}$=1.66, P=0.22). There was no significant interaction ($F_{1, 12}$=0.33, P=0.58). In RVFWS, there was a significant main effect for condition ($F_{1, 9}$=11.95, P=0.007). Overall, the pre-exercise RVFWS was lower than post exercise (see figure 2.3). There was no significant main effect for time ($F_{1, 9}$=1.24, P=0.30). There was no significant interaction ($F_{1, 9}$=0.002, P=0.97). In RVGLS, there was a significant main effect for condition ($F_{1, 9}$=22.51, P=0.001). Overall, the pre-exercise RVGLS was lower than post exercise (see figure 2.4). There was no significant main effect for time ($F_{1, 9}$=0.67, P=0. 44). there was no significant interaction ($F_{1, 9}$=0.001, P=0.98). In RV S', There was a significant main effect for

condition ($F_{1, 11}$=5.1, P=0.045). Overall, the pre-exercise RV S' was higher than post exercise (see figure 2.5). There was no significant main effect for time ($F_{1, 11}$=4.61, P=0. 55). There was no significant interaction ($F_{1, 11}$=0.007, P=0.94). In TAPSE, there was a significant main effect for condition ($F_{1, 11}$=8.1, P=0.016). Overall, the pre-exercise TAPSE was higher than post exercise (see figure 2.6). There was no significant main effect for time ($F_{1, 11}$=0.005, P=0. 94). There was no significant interaction ($F_{1, 11}$=0.14, P=0.72). There were no significant changes to any other functional indices.

| Parameter | 30min | | 60min | | P Value | | |
|---|---|---|---|---|---|---|---|
| | Pre | Post | Pre | Post | Condition | Time | Interaction |
| TAPSE (mm) | 20.7 ± 3.4 | 19.4 ± 4.3* | 19 ± 3.2 | 17 ± 3.2* | 0.045 | 0.06 | 0.94 |
| R S' (mm/s) | 14 ± 1.5 | 13 ± 1.8 | 15 ± 1.9 | 13 ± 3.2* | 0.016 | 0.94 | 0.72 |
| R E' (mm/s) | 13 ± 2.9 | 14 ± 2.0 | 14 ± 2.4 | 14 ± 4.2 | 0.59 | 0.89 | 0.92 |
| R A' (mm/s) | 10 ± 2.4 | 9 ± 3.8 | 10 ± 3.1 | 9 ± 4.6 | 0.73 | 0.96 | 0.90 |
| RVFWS (%) | -24.9 ± 2.9 | -22.4 ± 3.6* | -25 ± 4.6 | -22.9 ± 3* | 0.004 | 0.16 | 0.34 |
| RVGLS (%) | -21.6 ± 2.1 | -19.3 ± 2.6* | -21.7 ± 3.3 | -20.5 ± 2.7* | 0.003 | 0.37 | 0.20 |
| RV Basal | -25.3 ± 3.8 | -23.5 ± 4.6 | -23.9 ± 5.0 | -20.8 ± 6.9 | 0.18 | 0.22 | 0.97 |
| RV Mid | -28.6 ± 5.6 | -26.2 ± 6.0 | -28.6 ± 3.1 | -25.9 ± 3.4 | 0.13 | 0.92 | 0.89 |
| RV Api | -23.9 ± 7.1 | -20.9 ± 6.2 | -26.1 ± 3.8 | -23.2 ± 4.0 | 0.17 | 0.18 | 0.48 |
| RVFAC (%) | 43 ± 5.3 | 40 ± 9.8* | 45 ± 6.5 | 38 ± 5.3* | ≤0.001 | 0.96 | 0.30 |

**Table 2.2** highlights the main structural indices of the RV. * Denotes P≤0.05.

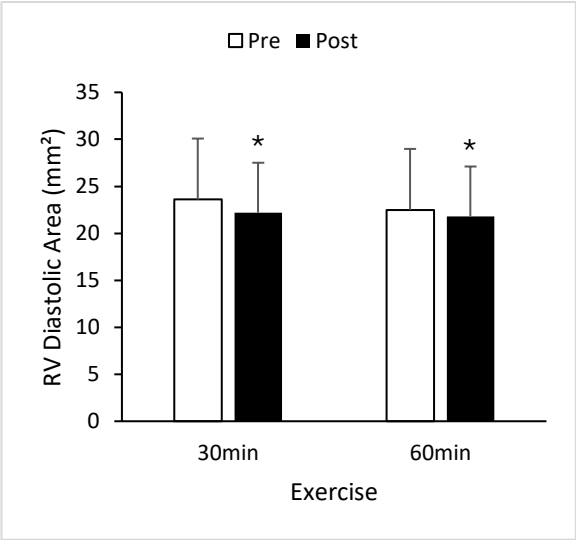


**Figure 2.1** displays the impact of both 30min and 60min swims on RVFAC. * Denotes P≤0.05 for condition.

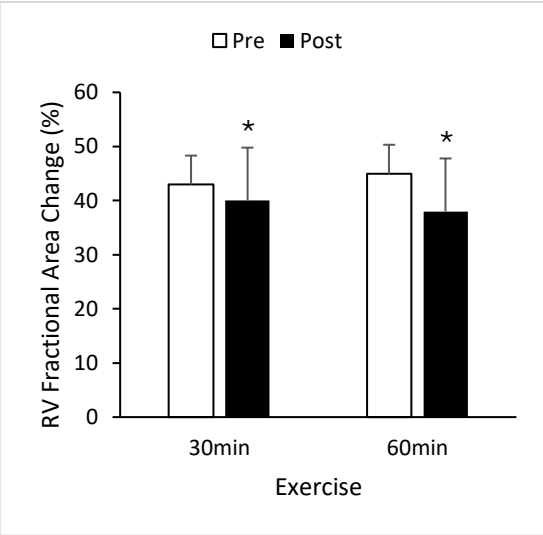


**Figure 2.2** displays the impact of both 30min and 60min swims on RVDA.* Denotes P≤0.05 for condition.

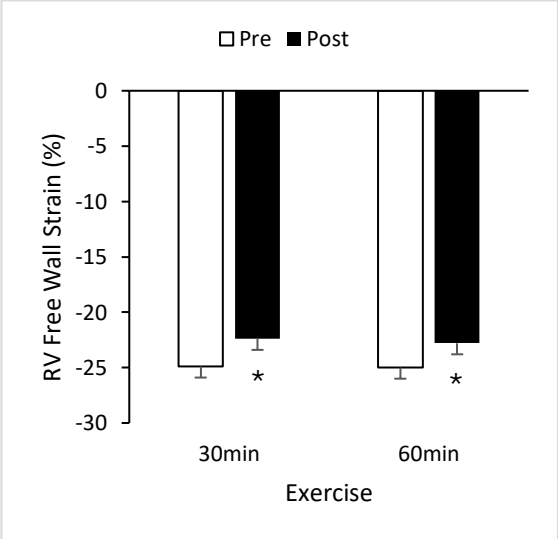


**Figure 2.3** displays the impact of both 30min and 60min swims on RVFWS. * Denotes P≤0.05 for condition.

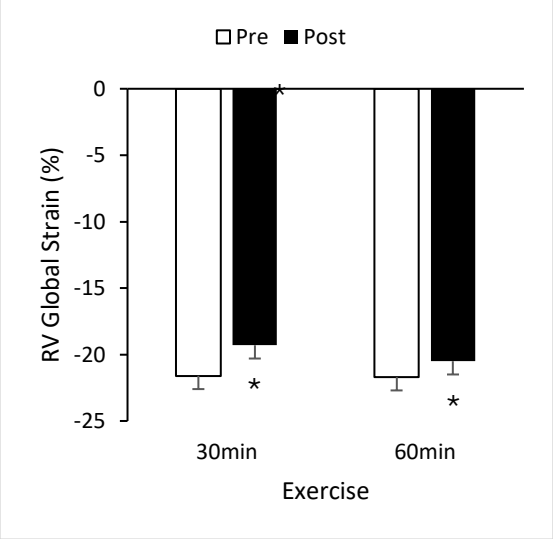


**Figure 2.4** displays the impact of 30mins swimming on RVGLS. * Denotes P≤0.05 for condition.

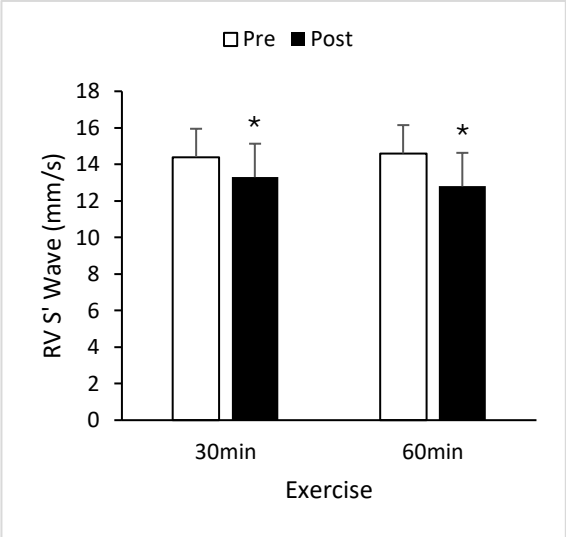


**Figure 2.5** displays the impact of both 30min and 60min swims on RV S'. * Denotes P≤0.05 for condition.

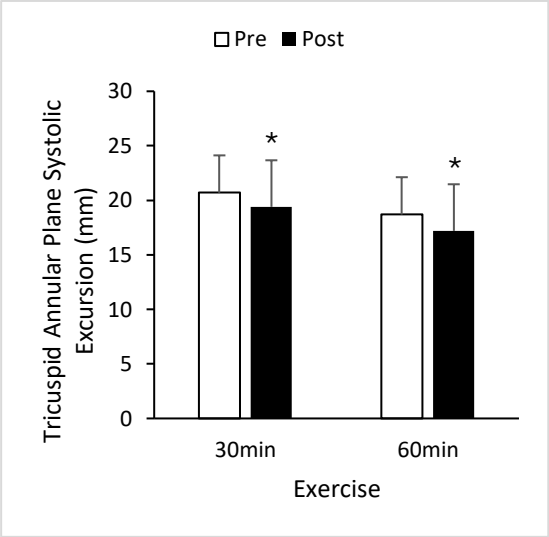


**Figure 2.6** displays the impact of both 30min and 60min swims on TAPSE. * Denotes P≤0.05 for condition.

**Discussion**

The key findings of this study were that after both 30mins and 60mins of swimming exercise there was 1) a significant reduction in RVFAC suggesting the presence of EICF, 2) a significant reduction in RVDA but with preserved RVSA, 3) a significant reduction in RV systolic function through reduced RVGLS, RVFWS, TAPSE and R S', 4) no significant correlations between RV and LV functional or structural indices and, 5) there was no significant impact for time on any of the indices.

The results show an alteration in both structure and function indicative of EICF after both durations of swimming exercise. RV function was reduced in systole denoted by reduced RVFWS, RVGLS, TAPSE and R S'. Structure had also been altered in diastole through reduced RVFAC and RVDA but with unchanged RVSA. There was no significant impact for duration on the degree of EICF observed, again contradicting previous literature (La Gerche, 2012).

The degree of systolic dysfunction in the RV is comparable to that of much longer/more intense land-based exercises. A reduction in RVGLS was recorded by Stewart et al after a 90min high intensity cycle (Stewart et al, 2017). The study showed a reduction in function of 1.2% in RVGLS (-27.6% to -26.4%) at rest post exercise compared to 2.3% after 30mins and 1.2% after 60mins of moderate intensity swimming exercise. The reduction in function occurred both longitudinally (RVFWS, RVGLS) and radially (RVFAC) showing whole ventricular reduction in function (Lee and Park, 2018). This again is displayed in Oxborough et al where a reduction in function of 3% in RV strain (-27% to -24%) was found post 161km ultramarathon (Oxborough et al, 2011). This further supports the theory that the physiological challenges of swimming augment the effects of EICF as a larger decrease in function occurs relative to intensity and duration than land-based exercises. The correlation analysis found no links between the delta changes in any RV or LV indices. However, the degree of reduced systolic function between the ventricles is comparable, with LVGLS reducing by 2.4% and RVGLS by 2.3% after 30mins and 1.7% and 1.2% after 60mins respectively. This contrasts with the previous literature as the RV does not display a disproportionate degree of fatigue (La Gerche, 2013), possibly as the dysfunction doesn't appear to be primarily driven by myocardial fatigue. Further analysis

of the basal, mid and apical segments of the RV free wall showed no significant signs of fatigue, further suggesting the RV was not subject to a disproportionate stress/load.

The dependence between the RV and LV was key to this study. Ventricular intercoupling has been well detailed in the literature (Stewart et al, 2017, Lord et al, 2015, Oxborough et al, 2011) concluding that a preliminary dysfunction in the RV may lead to dysfunction in the LV due to underfilling. Previous studies have documented RV dilation alongside reduced RV contractility post prolonged exercise (Elliot and La Gerche, 2015) due to sustained volume/pressure overload increasing wall stress (Lord et al, 2016). This dilation causes the septal wall to flatten (Oxborough et al, 2011) which has a serial impact on LV dimensions, reducing LVEDV but maintaining function. In this study the opposite occurred, with RVDA decreasing and RVSA remaining unchanged, causing a drop in RVFAC potentially due to reduced ventricular filling. Ventricular intercoupling studies have also shown an increase in RV strain followed by a subsequent increase in LV strain and reduced EF. In this study, no evidence of reduced diastolic function was found as RV E' remained unchanged. This alteration in both ventricles during diastole with no evidence of myocardial fatigue further suggests the changes are load dependent but across both ventricles not just the LV.

**Mechanisms**

The changes to RV structure and function mirrored those found in the LV, with a reduced systolic function and diastolic structure but with sustained systolic structure and diastolic function. The lack of diastolic fatigue combined with a reduced cavity size would suggest that RV myocardial fatigue and subsequent intrinsic failure of RV mechanics were not responsible for LV underfilling. There may be some contribution from systolic myocardial fatigue and increased afterload but there is little evidence to suggest these were the main causes. Instead, the EICF found in the RV may have also be load dependent, brought on by an overall reduction in ventricular filling and therefore preload.

A reduced preload could have multiple contributing factors. Dehydration can impact blood volume, although we do not believe this occurred for the reasons listed previously in chapter 4. It may have been due to increased heart rate. Heart rate post exercise is significantly increased which shortens diastolic filling time (Mbaissouroum et al, 1993),

potentially reducing diastolic volumes/dimensions. However, increases in heart rate are frequently documented in the literature but are not usually correlated with reduced diastolic volumes as exercise stimulates increased blood supply.

Another potential theory is that the unique properties of swimming may have been altering preload. The exercise was conducted in a supine position and utilises body wide muscle groups, which under regular circumstances would increase venous return and therefore preload. However, during swimming the heart is positioned under the water whilst the limbs are regularly positioned above/higher in the water. This may alter the way that blood is distributed amongst the vasculature and potentially impact venous return. In addition, due to the intermittent breath holding there could have been increases in pulmonary artery pressure and corresponding increases in intrathoracic pressure. According to Laplace's law (Li, 1985), wall stress= (pressure*radius)/(wall thickness*2). Increased intrathoracic pressure causes a reduced venous return as blood is redistributed to the periphery (Tyberg et al, 2000). This reduces the end-diastolic pressures of both ventricles resulting in reduced wall stress and end-diastolic volumes. There were also no changes to wall thickness post swimming exercise and afterload was raised so the myocardium may have attempted to balance the increased wall stress by reducing chamber radius. This potential redistribution in blood volume may also be impacted by the changes in gravity and hydrostatic pressure, perhaps further influencing venous return and leading to reduced ventricular filling.

**Conclusion**

To conclude, swimming for 30mins and 60mins leads to RV EICF in the form of reduced systolic function and reduced diastolic area, the degree of which is similar to land-based exercises of much greater workload which further suggests the physiological challenges of swimming may exacerbate the effects of ECIF. The results partially agree with the land-based literature as systolic function was compromised, however diastolic function remains unchanged, and duration had no impact. Although the RV is responsible for LV filling there is little evidence to show that RV dysfunction is the cause for LV underfilling in this study. There were no correlations between RV and LV structural or functional indices and neither ventricle displayed signs of diastolic dysfunction, showing a lack of myocardial fatigue. We can speculate that the dysfunction may be derived from an overall reduction in ventricular

filling, explaining the reduced systolic function and diastolic parameters. The mechanisms behind this were difficult to determine, but it may have been due to altered blood distribution from pressure/gravitational changes present during swimming.

**Chapter 6- Overall findings and discussion**

The aims of these studies were to explore what impact prolonged swimming at a moderate intensity had on cardiac structure and function, with a focus around EICF and whether the physiological pressures of swimming would augment any indicators of dysfunction. We defined EICF as a reduction in systolic/diastolic function post exercise in the LV or RV.

The first study found that after both 30mins and 60mins of swimming exercise, there was evidence of reduced systolic function and a reduced diastolic volume. The reduction in systolic function was comparable to that of far more demanding terrestrial exercises previously detailed in the literature, suggesting the physiological pressures of swimming could exacerbate EICF. However, the results showed a distinct lack of impairment to diastolic function, which usually precedes systolic dysfunction due to the order of the frank starling mechanism as the degree of initial stretching/relaxation impacts the myocardium's ability to contract (Delicce and Makaryus, 2017). This suggested that the EICF was not derived from myocardial fatigue but perhaps is load dependent. Although there was an increase in afterload, LV dimensions would suggest a reduced preload from LV filling could be the main driver for the EICF.

LV filling is heavily dependent on RV mechanics and so the second study sort to determine the impact on the RV, any signs of EICF present and the subsequent impact on LV filling. The results found systolic function and diastolic dimensions were reduced after both 30mins and 60mins of swimming, comparable to that found in land-based studies (Oxbourough et al, 2011). There was no evidence of reduced diastolic function, only a reduced diastolic area which contradicted other literature (Elliot and La Gerche, 2016). Although the RV contributes heavily to LV filling, no correlations were found between changes in RV mechanics and LV function or structure. From this we could speculate that the EICF found in the LV was not due to failing RV mechanics but an overall reduction in blood volume that impacted both ventricles. An increased HR may have also played a role with the reduced filling due to a reduced diastolic filling time (Mbaissouroum et al, 1993).

Both studies found no impact of duration with no difference in degree of EICF between 30mins and 60mins of swimming. This contradicted some of the literature around

duration being a contributing factor (La Gerche et al, 2012) but also aligned with other literature that intensity may be a greater determinant for EICF (Banks et al, 2010). These studies also contradicted the previous literature around cardiac fatigue in swimming which displayed either no signs of change to ventricular structure and function or an improvement in ventricular function post swim (Gajde et al, 2019). This is likely due to the intensity of the exercise as when distance/duration were matched the subjects in the current study were working at a higher intensity.

Overall, chapters four and five provided an insight into the potential impact of prolonged swimming on ventricular structure and function. Bouts of 30mins and 60mins of moderate intensity swimming exercise display signs of EICF through reduced systolic function and diastolic structure. There may be some systolic myocardial fatigue, but it is unlikely as no diastolic fatigue was observed. Instead, we could speculate the EICF appeared to be derived from an overall reduction in blood volume causing reduced cavity dimensions and preload. The mechanisms behind this are hard to establish as right atrial and inferior vena cava dimensions were not documented but the unique mechanics around swimming may have caused a redistribution of blood to the periphery. Figure 5.1 summarises the key findings of both empirical studies.

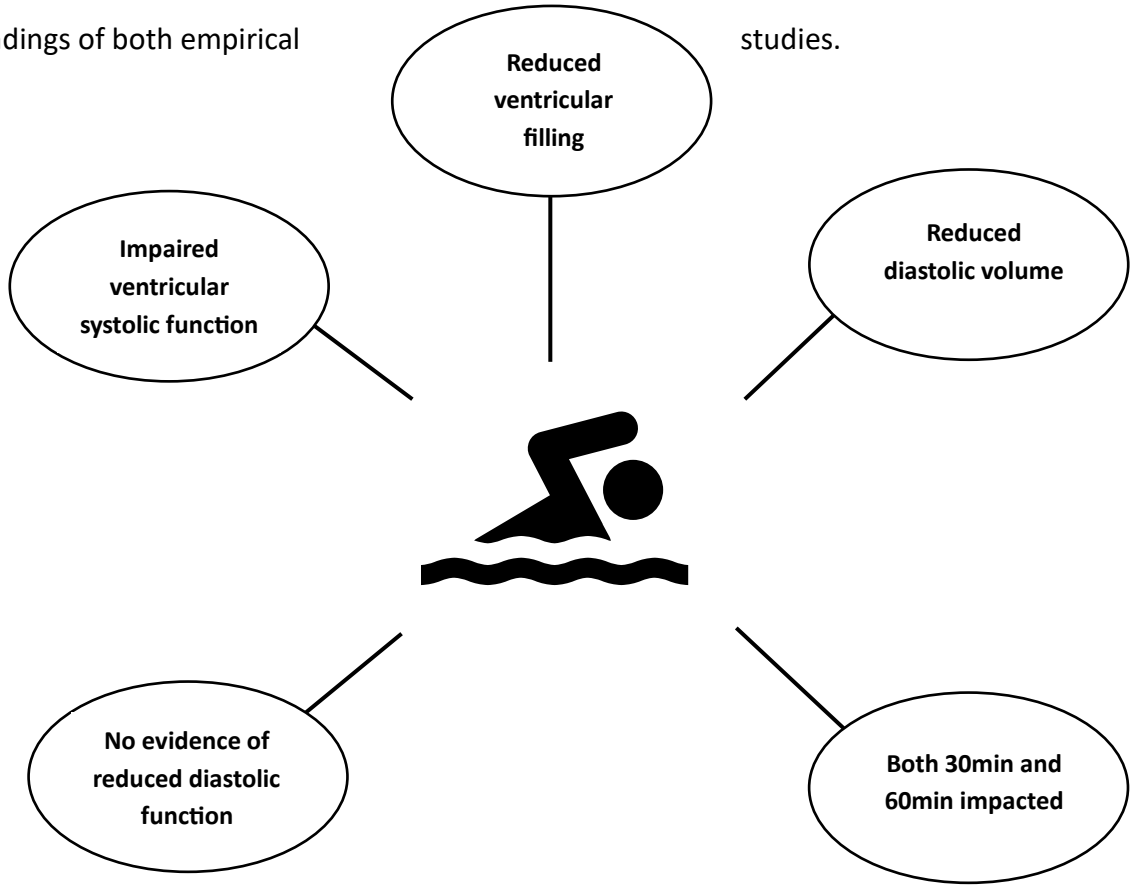

**Figure 5.1** displays an infographic summarising the key findings of both empirical studies

**Limitations and recommendations**

Due to the small sample size and complications with suboptimal image quality the findings of these studies lack the necessary power to draw solid conclusions on what mechanisms could be behind the EICF found. Initially the aim was to recruit 20 participants for both the 30min and 60min swims, however due to issues with pool time, staff availability and recruitment only fifteen subjects were recruited, of which only thirteen completed both swims. Data was further depleted due to suboptimal image quality, leaving eleven usable data sets. This resulted in the studies being underpowered. The age of the participants was also young (average of 20yrs±2), and the ethnic diversity of the participants was almost negligible with only one participant not being Caucasian. This means that the study doesn't fully represent the impact of prolonged swimming when applied to people of older ages or ethnic backgrounds. In addition, some participants were adolescence and so may have different cardiac development to the older participants due to influences from puberty (Goswami et al, 2014). In view of this, future research in this area should aim to accommodate a larger and more diverse cohort of individuals to ensure a better representation of the general population.

The results of both studies suggested a reduction overall ventricular filling and potentially blood volume. Considering this, future studies may benefit from observing right atrial volumes and TR to determine if the RV is impacted. In this study, there was not enough TR present to measure, however TR can be enhanced on images by using saline as highlighted in Lord et al (2023). It may also be interesting to measure blood plasma volume as an alteration in blood composition may be a contributing factor to the reduced ventricular filling (Coyle et al, 1989). To observe if there is dysfunction not caused by sympathetic withdrawal, future studies could also consider performing a stress echocardiograph at both pre and post as conducted by Kleinnibbelink and colleagues (Kleinnibbelink, 2021). This would allow for measurements to be taken at a similar HR, highlighting any dysfunction that occurs in the absence of varying ventricular filling times. It would also be interesting to see if there were changes to the vasculature, observing brachial and femoral arteries to test the theory of redistributed blood volume.

Future studies would also benefit from utilising other measuring techniques such as utilising 3D echocardiographs or a more in-depth analysis of LV and RV strain including circumferential and radial. In addition, although duration appeared to have no significant effect on the degree of dysfunction found, changing intensity may have an impact. If fatigue occurs 30mins into moderate intensity exercise, this may have an earlier onset at higher intensities or may display more prominent dysfunction if duration is maintained and intensity is increased (Banks et al, 2010). It may also be beneficial to directly assess the changes in pulmonary artery pressures and intrathoracic pressure to see what effect they have on blood circulation specifically in swimming. However, this may not be possible/practical as this would require subjects to swim whilst undergoing right heart catheterization where a catheter would be inserted into the pulmonary artery (Kovacs et al, 2014).

There has also recently been the introduction of measuring myocardial work as a tool to assess EICF. Myocardial work combines systolic blood pressure with LV GLS to create LV pressure-strain loops (Erevik et al, 2023). This method provides an additional assessment of myocardial inefficiencies prior to changes in LV global function and can detect pathological alterations caused by ischemia, perhaps providing a more in-depth assessment of LV function.

A final recommendation would be to have a follow up cardiac assessment during the recovery period of testing. Previous research has shown variations in the response of the heart over different recovery periods post exercise. For example, Gajda et al found a 6.9% increase in LVEF 48hrs into recovery after the 500km relay swim (Gadja et al, 2019). La Gerche et al on the other hand observed RV and LV function 6 to 11 days after completing various endurance races (La Gerche et al, 2012). The results found RV function to still be slightly but not significantly depressed (RVEF= 51±3.6% vs 50±3.8%, RVFAC= 51.5±6% vs 49.8±6.6%) and strain to be elevated (RV S= -27.2±3.4% vs -25.6±3%). LV function had slightly increased with LVEF (58.8±5.1%) surpassing baseline and immediately post exercise values (56.4±5.2% vs 57.5±5.6%) but strain was still elevated (-18.4±3.7% vs -17.7±2.3%). Provided both current studies found systolic dysfunction within the ventricles after only 30mins it would be interesting to see how long this is sustained for once

exercise has been concluded. Should the dysfunction after only 30mins of swimming remain present after a given time of recovery this would be a major finding.

**Conclusion**

This thesis aimed to explore what impact prolonged swimming had on cardiac structure and function with a focus on EICF. Based on the current literature around EICF in terrestrial based sports and the unique physiological challenges swimming poses (Riding, 2016), the hypothesis of the empirical studies in chapter 4 and 5 was that a relatively short duration of swimming at a moderate intensity would display signs of EICF. Chapters 4 and 5 highlighted the presence of EICF in both ventricles after 30mins and 60mins of swimming. However, the evidence suggested this was not caused by myocardial fatigue but rather an overall reduction in ventricular filling. The mechanisms behind the dysfunction were unclear, however it could be theorised that the unique positioning of the heart in relation to the limbs during swimming in addition to alterations in pressure may be redistributing blood and causing a reduction in venous return.

**Chapter 7- Appendices and references**

**3.1 The data collection sheet used during the studies in chapter 4 and 5**

## Prolonged swimming cardiac structure and function data collection sheet

| | |
|---|---|
| Study ID | Date |
| Test duration Mins | Distance covered M |

| | |
|---|---|
| Height | Cm |
| Weight | Kg |
| Blood pressure | mmHg |
| FVC | |
| FEV1 | |
| Post BP | mmHg |
| Post FVC | |
| Post FEV1 | |

### 3.2 The recruitment poster used during the studies in chapter 4 and 5

LIVERPOOL JOHN MOORES UNIVERSITY

# Acute impact of prolonged swimming on cardiac structure and function

## What effect does long distance swimming have on the heart?

We're looking for swimmers aged 16-35 to take part in a study looking into the effects long duration swimming has on the heart. The study aims to understand the structural and functional changes that occur after a 30min and 1-hour continuous swim. To do this, the study will use ECG and echocardiography to observe both the electrical activity of the heart and the structure of the heart chambers before and after swimming.

## What does the study involve?

Participants will be asked to attend 2 separate sessions where the following tasks will be completed:

- A 30 min or 1-hour continuous swim
- Anthropometrics- height and weight
- Blood pressure measurement
- Electrocardiogram- electrical activity of the heart
- Echocardiogram- ultrasound images of the heart
- Spirometry- breathing test

More information on what the tests are and how they are conducted is available on the participant information sheet.

## Benefits of taking part

- Participants will receive a full cardiac health screening

### Location

The study will take place at the Wavertree aquatics centre located at Wavertree Sports Park, Wellington Rd, Liverpool
L15 4LE

### Are you eligible?

You are eligible to take part if you are:

- Aged between 16-35
- Male or female
- Currently take part in swimming training ≥3hours per week

**You are not eligible for this study if you:**

- Have a history of cardiac disease
- Are taking cardiovascular medication
- Have diabetes
- Are pregnant

## Contact details

If you are interested in taking part in the study and would like more information, please contact Chris Lambert, the principle investigator of the study, via email at spsclamb@ljmu.ac.uk. You will be sent a detailed participant information sheet to look at and if you wish to take part, we will organise a preferable date and time to attend testing.

**3.3 Conformation email of ethical approval for the studies in chapter 4 and 5**

Dear Chris

**Chris Lambert, PGR - The swimmer's heart: impact of discipline and stroke type (David Oxborough/Robert Cooper)**
**UREC decision: Favourable ethical opinion**
**UREC reference: 23/SPS/071**
**Research Governance Assessment: Approved – the study may commence.**
On behalf of the University Research Ethics Committee (UREC), I am pleased to confirm a favourable ethical opinion for the above study on the basis described in the application form, protocol, supporting documentation and any clarifications received, subject to the conditions specified below.

Conditions of the favourable opinion

· You must ensure the information included in the participant facing documents are always current and informed by ongoing risk assessments and any changes to current practices (https://www.ljmu.ac.uk/ris/research-ethics-and-governance/research-ethics/university-research-ethics-committee-urec/ethics-application-form-and-templates)
· Where any substantive amendments are proposed to the protocol or study procedures further ethical opinion must be sought (https://www.ljmu.ac.uk/ris/research-ethics-and-governance/research-ethics/university-research-ethics-committee-urec/amendments)
· Any adverse reactions/events which take place during the course of the project are reported to the Committee immediately by emailing FullReviewUREC@ljmu.ac.uk
· Any unforeseen ethical issues arising during the course of the project will be reported to the Committee immediately emailing FullReviewUREC@ljmu.ac.uk

Please note that favourable ethics opinion is given for a period of five years. An application for extension of the ethical opinion must be submitted if the project continues after this date.

**Research Governance Approval.**
This email also constitutes LJMU Research Governance Approval of the above referenced study on the basis described in the ethics application form, protocol, supporting documentation and any clarifications received, subject to the conditions specified below.
Conditions of Approval

- Compliance with LJMU Health and Safety Codes of practice and risk assessment policy and procedures (https://www.ljmu.ac.uk/staff/hsu/codes-of-practice-and-guidance-notesand) and LJMU code of practice for research (https://www.ljmu.ac.uk/-/media/staff-intranet/research/ris/ris-documents/ljmu-code-of-practice.pdf)

- Ensure the study is covered by UMAL liability/indemnity (https://www.ljmu.ac.uk/staff/finance/insurance)
- Compliance with LJMU Data Protection Policies and procedures (https://www.ljmu.ac.uk/about-us/data-protection)
- Agreements/contracts are arranged as required (e.g. collaboration agreements, general agreements, data processing/sharing agreements, intellectual property rights agreements, financial provisions agreements/contracts, material transfer agreements etc.)
- Where relevant, appropriate gatekeeper / management permission is obtained at the study site concerned.
- The LJMU logo is used for all documentation relating to participant recruitment and participation e.g. poster, information sheets, consent forms, questionnaires.
- The study consent forms, study data/information, all documents related to the study etc. will be accessible on request to a student's supervisory team and/or to responsible members of Liverpool John Moores University for monitoring, auditing and data authenticity purposes.

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