**A SIMPLIFIED REVIEW OF LEFT VENTRICULAR TORSION**

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**Abstract**

Recent technological advancements have provided greater insight into left ventricular mechanics, in particular left ventricular torsion and untwisting. An understanding of left ventricular torsion and its measurement provides great insight into the function of the heart in various conditions. For instance, left ventricular torsion appears to be a more sensitive marker of cardiac adaptations to different conditions such as diabetes and aging in comparison to conventional global markers. Ultrasound derived measures of twisting forces within the heart are becoming the most widely used measurement due to the temporal resolution, affordability, portability, and ease of usage. The purpose of this review is to discuss pertinent information regarding left ventricular torsion and its measurement for the understanding of this somewhat obscure concept by non clinicians. It is also intended to demonstrate the widespread applicability of this concept including its use with elite athletes and patients with established chronic disease.

**Keywords: review, ventricular torsion, ventricular twisting**

**Introduction**

A simple explanation of the purpose of the heart muscle would be to pump oxygenated blood through the body. In doing this, the heart receives oxygen deficient blood returning from circulation, pumps this blood through the lungs, and then finally produces a high pressured pulse to eject the now fully oxygenated blood throughout all perfusable tissues. The main workhorse of the heart, and where the majority of heart mass is located, is the left ventricle. The left ventricle’s primary job is to eject enough blood at a high enough pressure to effectively perfuse the body with oxygenated blood. The heart beats an average of 25,000,000,000 (2.5 billion) times over a lifetime at a rate of 60 (rest) to 200 (exercise) beats per minute (Levine, 1997). To create pressures of 100-300 mmHg (Mundal et al., 1996) at such a high rate and for such a long period of time, the heart has evolved to become highly efficient at rapidly creating pressure within the left ventricle. Initially, the heart was believed to function in a diameter collapsing manner, similar to squeezing a soda bottle. Later studies revealed that in addition to this action, there is a linearly collapsing function, likened to also pushing down on either end of the bottle. Finally, in recent years, some laboratories (Han et al., 2008; Park et al., 2008; Tanaka et al., 2008) including our own (Esch et al., 2010; Esch et al., 2009; Esch & Warburton, 2009) have shown that there is a third twisting action (torsion) which also significantly contributes to ventricular contraction; similar to twisting an empty soda bottle with one hand on the top and the other on the bottom. Because there is relative lack of knowledge of the third important functional characteristic among the general population, this review is intended to highlight and simplify the key features of left ventricular torsional forces for non-clinicians and exercise professionals.

**How Left Ventricular Torsion is Generated**

The way rotational forces are created owes everything to the orientation of the fibres of the heart. Muscle fibres, when examined on an individual basis, are long cells that expend energy in order to shorten. Our body has evolved to maximally exploit the shortening ability of muscular cells (through various arrangements and synchronies) to achieve an almost infinite amount of movements and functions. Muscle fibres can only shorten along their longitudinal axis, essentially working to pull the terminal ends closer together. When many muscle fibres are orientated in parallel, the result is an increased contraction force. As opposed to the parallel fibre arrangement of most other muscles in the body, the heart muscle is unique in that the fibres are configured in an array of orientations (Streeter et al., 1969). It appears that certain portions of the heart wall possess differently orientated muscle fibres. For example, the outer wall has muscle fibres (U in Figure 1) aligned at -60 degrees to the circumferential angle of the ventricle, while the inner wall muscle fibre orientation (α in Figure 1) is at +60 degrees (Figure 1). The muscle fibre orientation at the intermediate layers gradually transition from the relatively extreme angles found at the inner and outer walls (Figure 1). When considering the muscle fibre layout of the left ventricle, it is logical that numerous force angles are involved during ventricular contraction and that this results in several notable plains of motion. Muscle fibres aligned parallel to the long axis (ie. up and down) cause squeezing of the heart from bottom to top, while muscle fibres aligned along the width (e.g. left to right) cause squeezing side to side. Finally, the more recently identified torsional motion (like twisting a plastic soda bottle, see Figure 2) is caused by fibres aligned at oblique angles (Esch et al., 2010; Esch et al., 2009; Esch & Warburton, 2009).

**How Left Ventricular Torsion leads to Increased Pressure and Efficiency**

*Increased Ventricular Filling*

To eject adequate blood at a sufficient pressure, the left ventricle maximizes the efficiency of both contraction and relaxation. During the relaxation phase of the heart cycle the left ventricle actively rests – that is, proteins that were actively squeezed together during contraction now passively expand and push the ventricular chamber open (think squeezing a stress ball tightly then releasing). Having the chamber actively expand creates a vacuum in the left ventricle which sucks blood from the left atrium without expending energy. This early filling, which avoids active pumping of blood into the ventricle, saves energy for later in the cycle when the vacuum is lost. Ventricular torsion plays a large role ensuring effective ventricular function though maximizing suction (better known as the mitral gradient) during rest. This contribution of torsion or untwisting (the resting phase of torsion) to left ventricle filling has been illustrated in studies showing that rapid untwisting rates are essential for reducing left ventricular pressure during diastole (Notomi, Martin-Miklovic et al., 2006). Furthermore, persons with delayed untwisting have been shown to have a lower pressure gradient between the atrium and ventricle driving the ejection of blood (Fuchs et al., 2004; Notomi, Martin-Miklovic et al., 2006; Notomi, Srinath et al., 2006). It appears that untwisting occurs very rapidly after contraction and may occur before other suction creating forces develop.

*Increased Ventricular Contraction Efficiency*

In order to appreciate the importance of efficient left ventricle resting time (for achieving greater ejection volumes and pressures), one more concept must be understood. Efficient left ventricular relaxation and untwisting is essential for maintaining high ejection volumes and pressures. This association between the relaxation phase and contraction phase occurs because the heart contracts to a greater extent the more it is distended or stretched during rest. A good analogy to understand this concept is blowing up a balloon and letting it go. If a standardized balloon is inflated to a low volume and released, the air that rushes out as the balloon soars around the room not only has a lower volume but a lower pressure than if the same balloon is inflated to a larger volume. This is due to a far greater amount of stored elastic energy in the walls of the balloon that is more inflated and more stretched. The same forces are in effect in the left ventricle; this is called the Frank-Starling law. As previously, discussed, suction created during diastole by untwisting allows for enhanced ventricular filling by preserving atrial energy expenditure until later in the cardiac cycle. Therefore, by creating a beneficial atrial-ventricular pressure gradient and increasing ventricular filling, torsion/untwisting plays a large role improving ventricular efficiency during contraction throughout both ventricular filling and ejection(Nikolic et al., 1995).

An additional method by which torsional forces are thought to improve contraction efficiency is by equalizing muscle fibre shortening between the outer and inner layers of the left ventricle. For example, imagine the left ventricle as a structure as a hollow tubular shape with a thick outer wall (picture a rolled up yoga mat). When the mat is then bound tighter, for a given decrease in the hollow radius, the inside layer of the mat gets much shorter as compared to the outside layer. This seemingly minor imbalance in the shortening length over one beat would be compounded over the course of a lifetime. Thankfully, the heart is designed to overcome this issue. To help us explain, let’s re-explore the example of an empty soda bottle. Imagine yourself twisting a soda bottle, realize that you can twist the bottle in two different ways, that is, your top hand can rotate clockwise and bottom hand counter-clockwise or vice versa. After dissecting numerous hearts, researchers now know that the outer layer of the heart is aligned to rotate the heart in the first mentioned direction while the inner layer is oriented to rotate the opposite way. To determine the outcome of these two layers seemingly working against each other, the tiebreaker goes in favour of the outer layer of the left ventricle, as it lies further away from the axis of rotation, so it generates the most force and dominates the characteristic twist (top hand counter clockwise, bottom hand clockwise), while the inner layer works in futile opposition. Therefore, because the twisting motion is dominated by the orientation of the outer ventricular muscle layer, the outer muscle later contracts more in response to torsion than the inner layer (which is partially opposed from its preferred motion). Without torsion, the inner layer of the heart would be unevenly producing the majority of the force during ventricular ejection. The torsional motion of the ventricle is dominated by, and relies upon, the force production of the outer layer. In combination, these two forces ensure equal force production from both the inner and outer layers of the left ventricle during contraction (Figure 3).

**How Left Ventricle Torsion is Measured**

Ventricular torsion has been measured using three methods. All three techniques are similar in that they essentially involve labelling cardiac muscle fibres and tracking their motion. One method, called implanted myocardial marking, involves physically attaching metallic markers on muscle fibres during surgery and later using imaging techniques to track the markers during the heart cycle. Because of the extremely invasive nature of this technique it is rarely performed. The remaining two ways to measure ventricular torsion/ untwisting are called magnetic resonance imaging (MRI) with tissue tagging, and echocardiography with tissue tracking.

 The use of MRI for evaluating torsion of the left ventricle is the most validated technique and considered the most accurate (Notomi, Setser et al., 2005) . Areas of interest can be digitally “tagged” using specialized software and analyzed throughout the cardiac cycle. This technique allows a very high level of detail in that even the motion of the outer and inner layers of the left ventricle can be focused on and evaluated independently (Lumens et al., 2006; Osman et al., 1999; Zerhouni et al., 1988). Two main drawbacks of MRI in comparison to the echocardiography technique for evaluation of ventricular torsion are the reduced temporal resolution of the videos and the substantially higher cost of equipment. The echocardiography method for determining ventricular torsion involves the use of speckles (tiny unique image patterns created by structures smaller than the ultrasound wavelength) which are consistent during minor motion such as torsion (Helle-Valle et al., 2005; Notomi, Lysyansky et al., 2005; Notomi, Setser et al., 2005). When tracking speckles identified in the ventricular wall over a cardiac cycle and digitizing the motion, an estimate of torsional forces is produced (Akagawa et al., 2007; Esch et al., 2010; Esch et al., 2009; Esch & Warburton, 2009; Notomi, Lysyansky et al., 2005; Notomi, Setser et al., 2005). The results of speckle tracking echocardiography correlate well with the MRI technique (r=0.84;(Notomi, Setser et al., 2005). The main advantages of echocardiography include the relative portability (allowing for evaluating torsion in a variety of situations including exercise), lower cost, and higher temporal resolution. Disadvantages of ultrasound for evaluating torsion include the dependence of the results on accurate heart images and relatively vague land-marking (in comparison MRI images can be selected very precisely). A limited number of studies have measured ventricular torsion during exercise. All of the published investigations we are aware of have used echocardiography to evaluate ventricular torsion (Esch et al., 2009; Neilan et al., 2006; Notomi, Martin-Miklovic et al., 2006; Tischler & Niggel, 2003). The combination of portability and high resolution make echocardiography the most viable choice when imaging cardiac structures during exercise.

**Left Ventricular Torsion and Exercise**

**Acute exercise**

A number of physiological factors that influence ventricular torsion are augmented during exercise. The return of blood back to the heart through the veins, the ability of the cardiac muscle to generate force and sympathetic predominance (the excitatory reflex of the body) increase during exercise(Tischler & Niggel, 2003). In opposition however, afterload (the pressure the ventricle is pumping against) also increases and leads to somewhat decreased ventricular torsion. Although these factors indeed work against each other to some degree, it appears that the physiological changes leading to increased torsion offset the increase in afterload as several investigations have shown torsion to increase during exercise (Neilan et al., 2006; Notomi, Martin-Miklovic et al., 2006; Tischler & Niggel, 2003).

**Long term exercise training**

To understand fully how exercise training influences ventricular torsion, it helps to examine how physiological characteristics, which have been shown to influence torsion, respond to chronic exercise. Firstly, long term exercise training leads to increased whole body blood volume, which in turn leads to increased ventricular filling during the resting phase of the cardiac cycle. According to the aforementioned Frank-Starling law, increased filling during the resting phase increases ventricular ejection volume during contraction and increase left ventricular torsion (Dong et al., 1999; Warburton et al., 1999). Chronic exercise also leads to a reduced afterload (Gledhill et al., 1994), which increases torsional motion allowing for greater ejection volumes. Additionally, trained individuals have been shown to have a low resting heart rate (Katona et al., 1982) and this is associated with a decreased torsion in athletes at rest (Nottin et al., 2008). Also, because athletes have been shown to have significantly decreased peak heart rates as compared to untrained individuals(Whyte et al., 2008), it could be suggested that torsion is reduced in those who are trained in general. Indeed, torsion may be reduced in trained individuals, but only at rest, and this likely reserves more torsional motion for exercise, leading to increased exercise capacity and performance. In line with this supposition, research has shown that detraining in the form of prolonged head-down bed rest reduces ventricular torsion and that supine exercise can mitigate this reduction and even improve the ventricular torsional motion (Dorfman et al., 2008).

**Left Ventricular Torsion in Disease and Aging**

The measurement of left ventricular torsion at rest may provide information regarding cardiovascular physiology beyond what is currently understood. Torsion has been shown to be reduced in those undergoing cardiac allograft rejection even with no significant reduction in stroke volume (Hansen et al., 1987) and to predict cardiac tissue changes after heart attack (Nucifora et al.). Another investigation examined left ventricular torsion in asymptomatic adults with no other signs of cardiovascular disease (clinical examination)(Lumens et al., 2006). Interestingly, older patients had significantly reduced ventricular torsion as compared to the younger cohort, suggesting twisting mechanics of the left ventricle may be more sensitive to cardiovascular disease risk factors than measures derived from clinical examinations (Lumens et al., 2006). Left ventricular torsion has also been shown to be increased in those with type I & II diabetes (Chung et al., 2006; Fonseca et al., 2004), possibly preceding more evident cardiac muscle fibre changes that are well documented in diabetics (Chung et al., 2006). Potential explanations for augmented ventricular torsion in those with cardiovascular disease include a change in muscle fibre orientation angle and a reduced elastic component which is less able to rebound after contraction and aid in chamber filling during the resting phase (Beyar et al., 1989; Tibayan et al., 2002). It has been suggested that although many torsional characteristics mirror changes in ventricular volumes, the untwisting rate during the ventricular relaxation phase provides new and detailed information regarding diastolic performance (Arts et al., 2009). Although many of the identified changes to ventricular torsion in various diseases lack an established pathological rationale, the existing information suggests that heart torsion may be more sensitive to cardiac pathology than traditional measures, or at least provide supplementary information on heart function.

**Conclusions**

Muscle fibre orientations found in the left ventricle allow for torsional/untwisting motion. By increasing ventricular filling and balancing muscle fibre shortening between the inner and outer muscle layers, ventricular torsion plays an important role ensuring efficient production of sufficient ejection pressures during contraction but more evidently during the relaxation phase of the heart cycle. Recent advances in imaging technology and increasing affordability of equipment has allowed for more detailed analyses of heart motion. The study of twisting forces occurring in the left ventricle over the cardiac cycle adds valuable information to the understanding of heart function during exercise, aging, and various disease states.

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The author qualifications are as follows: Aaron Phillips, MSc, CSEP CEP; Anita Cote, MSc, CSEP CEP, CSEP CPT-CC; Darren Warburton MSc, PhD, CSEP CEP, CSEP CPT-ME.

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| Figure 1.Muscle fibre orientation in various layers of muscle tissue in the ventricular wall. [(Streeter et al., 1969)]  |

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| Figure 2. Orientation of the left ventricle during the resting phase (left) and contraction phase (right) highlighting the rotational motion. [(Esch & Warburton, 2009)] |

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| Figure 3. Torsion is imperative at normalizing the shortening lengths of muscle fibres on the inner and outer ventricular wall. Notice in the bottom left that the inner wall muscle fibres shorten to a larger extent during non twisting reductions in ventricular diameter. Also notice in bottom right the rotational motion during contraction normalizes this imbalance. [(Lumens et al., 2006)] |

**References**

Akagawa, E., Murata, K., Tanaka, N., Yamada, H., Miura, T., Kunichika, H., et al. (2007). Augmentation of left ventricular apical endocardial rotation with inotropic stimulation contributes to increased left ventricular torsion and radial strain in normal subjects: quantitative assessment utilizing a novel automated tissue tracking technique. *Circ J, 71*(5):661-668.

Arts, T., Prinzen, F. W., & Delhaas, T. (2009). Potentials and limitations of ventricular torsion as indicator of cardiac function. *Conf Proc IEEE Eng Med Biol Soc, 2009*:181-184.

Beyar, R., Yin, F. C., Hausknecht, M., Weisfeldt, M. L., & Kass, D. A. (1989). Dependence of left ventricular twist-radial shortening relations on cardiac cycle phase. *Am J Physiol, 257*(4 Pt 2):H1119-1126.

Chung, J., Abraszewski, P., Yu, X., Liu, W., Krainik, A. J., Ashford, M., et al. (2006). Paradoxical increase in ventricular torsion and systolic torsion rate in type I diabetic patients under tight glycemic control. *J Am Coll Cardiol, 47*(2):384-390.

Dong, S. J., Hees, P. S., Huang, W. M., Buffer, S. A., Jr., Weiss, J. L., & Shapiro, E. P. (1999). Independent effects of preload, afterload, and contractility on left ventricular torsion. *Am J Physiol, 277*(3 Pt 2):H1053-1060.

Dorfman, T. A., Rosen, B. D., Perhonen, M. A., Tillery, T., McColl, R., Peshock, R. M., et al. (2008). Diastolic suction is impaired by bed rest: MRI tagging studies of diastolic untwisting. *J Appl Physiol, 104*(4):1037-1044.

Esch, B. T., Scott, J. M., Haykowsky, M. J., Paterson, I., Warburton, D. E., Cheng-Baron, J., et al. (2010). Changes in ventricular twist and untwisting with orthostatic stress: endurance athletes versus normally active individuals. *J Appl Physiol, 108*(5):1259-1266.

Esch, B. T., Scott, J. M., Warburton, D. E., Thompson, R., Taylor, D., Cheng Baron, J., et al. (2009). Left ventricular torsion and untwisting during exercise in heart transplant recipients. *J Physiol, 587*(Pt 10):2375-2386.

Esch, B. T., & Warburton, D. E. (2009). Left ventricular torsion and recoil: implications for exercise performance and cardiovascular disease. *J Appl Physiol, 106*(2):362-369.

Fonseca, C. G., Dissanayake, A. M., Doughty, R. N., Whalley, G. A., Gamble, G. D., Cowan, B. R., et al. (2004). Three-dimensional assessment of left ventricular systolic strain in patients with type 2 diabetes mellitus, diastolic dysfunction, and normal ejection fraction. *Am J Cardiol, 94*(11):1391-1395.

Fuchs, E., Muller, M. F., Oswald, H., Thony, H., Mohacsi, P., & Hess, O. M. (2004). Cardiac rotation and relaxation in patients with chronic heart failure. *Eur J Heart Fail, 6*(6):715-722.

Gledhill, N., Cox, D., & Jamnik, R. (1994). Endurance athletes' stroke volume does not plateau: major advantage is diastolic function. *Med Sci Sports Exerc, 26*(9):1116-1121.

Han, W., Xie, M. X., Wang, X. F., Lu, Q., Wang, J., Zhang, L., et al. (2008). Assessment of left ventricular torsion in patients with anterior wall myocardial infarction before and after revascularization using speckle tracking imaging. *Chin Med J (Engl), 121*(16):1543-1548.

Hansen, D. E., Daughters, G. T., 2nd, Alderman, E. L., Stinson, E. B., Baldwin, J. C., & Miller, D. C. (1987). Effect of acute human cardiac allograft rejection on left ventricular systolic torsion and diastolic recoil measured by intramyocardial markers. *Circulation, 76*(5):998-1008.

Helle-Valle, T., Crosby, J., Edvardsen, T., Lyseggen, E., Amundsen, B. H., Smith, H. J., et al. (2005). New noninvasive method for assessment of left ventricular rotation: speckle tracking echocardiography. *Circulation, 112*(20):3149-3156.

Katona, P. G., McLean, M., Dighton, D. H., & Guz, A. (1982). Sympathetic and parasympathetic cardiac control in athletes and nonathletes at rest. *J Appl Physiol, 52*(6):1652-1657.

Levine, H. J. (1997). Rest heart rate and life expectancy. *J Am Coll Cardiol, 30*(4):1104-1106.

Lumens, J., Delhaas, T., Arts, T., Cowan, B. R., & Young, A. A. (2006). Impaired subendocardial contractile myofiber function in asymptomatic aged humans, as detected using MRI. *Am J Physiol Heart Circ Physiol, 291*(4):H1573-1579.

Mundal, R., Kjeldsen, S. E., Sandvik, L., Erikssen, G., Thaulow, E., & Erikssen, J. (1996). Exercise blood pressure predicts mortality from myocardial infarction. *Hypertension, 27*(3 Pt 1):324-329.

Neilan, T. G., Ton-Nu, T. T., Jassal, D. S., Popovic, Z. B., Douglas, P. S., Halpern, E. F., et al. (2006). Myocardial adaptation to short-term high-intensity exercise in highly trained athletes. *J Am Soc Echocardiogr, 19*(10):1280-1285.

Nikolic, S. D., Feneley, M. P., Pajaro, O. E., Rankin, J. S., & Yellin, E. L. (1995). Origin of regional pressure gradients in the left ventricle during early diastole. *Am J Physiol, 268*(2 Pt 2):H550-557.

Notomi, Y., Lysyansky, P., Setser, R. M., Shiota, T., Popovic, Z. B., Martin-Miklovic, M. G., et al. (2005). Measurement of ventricular torsion by two-dimensional ultrasound speckle tracking imaging. *J Am Coll Cardiol, 45*(12):2034-2041.

Notomi, Y., Martin-Miklovic, M. G., Oryszak, S. J., Shiota, T., Deserranno, D., Popovic, Z. B., et al. (2006). Enhanced ventricular untwisting during exercise: a mechanistic manifestation of elastic recoil described by Doppler tissue imaging. *Circulation, 113*(21):2524-2533.

Notomi, Y., Setser, R. M., Shiota, T., Martin-Miklovic, M. G., Weaver, J. A., Popovic, Z. B., et al. (2005). Assessment of left ventricular torsional deformation by Doppler tissue imaging: validation study with tagged magnetic resonance imaging. *Circulation, 111*(9):1141-1147.

Notomi, Y., Srinath, G., Shiota, T., Martin-Miklovic, M. G., Beachler, L., Howell, K., et al. (2006). Maturational and adaptive modulation of left ventricular torsional biomechanics: Doppler tissue imaging observation from infancy to adulthood. *Circulation, 113*(21):2534-2541.

Nottin, S., Doucende, G., Schuster-Beck, I., Dauzat, M., & Obert, P. (2008). Alteration in left ventricular normal and shear strains evaluated by 2D-strain echocardiography in the athlete's heart. *J Physiol, 586*(Pt 19):4721-4733.

Nucifora, G., Marsan, N. A., Bertini, M., Delgado, V., Siebelink, H. M., van Werkhoven, J. M., et al. Reduced left ventricular torsion early after myocardial infarction is related to left ventricular remodeling. *Circ Cardiovasc Imaging, 3*(4):433-442.

Osman, N. F., Kerwin, W. S., McVeigh, E. R., & Prince, J. L. (1999). Cardiac motion tracking using CINE harmonic phase (HARP) magnetic resonance imaging. *Magn Reson Med, 42*(6):1048-1060.

Park, S. J., Miyazaki, C., Bruce, C. J., Ommen, S., Miller, F. A., & Oh, J. K. (2008). Left ventricular torsion by two-dimensional speckle tracking echocardiography in patients with diastolic dysfunction and normal ejection fraction. *J Am Soc Echocardiogr, 21*(10):1129-1137.

Streeter, D. D., Jr., Spotnitz, H. M., Patel, D. P., Ross, J., Jr., & Sonnenblick, E. H. (1969). Fiber orientation in the canine left ventricle during diastole and systole. *Circ Res, 24*(3):339-347.

Tanaka, H., Oishi, Y., Mizuguchi, Y., Miyoshi, H., Ishimoto, T., Nagase, N., et al. (2008). Contribution of the pericardium to left ventricular torsion and regional myocardial function in patients with total absence of the left pericardium. *J Am Soc Echocardiogr, 21*(3):268-274.

Tibayan, F. A., Lai, D. T., Timek, T. A., Dagum, P., Liang, D., Daughters, G. T., et al. (2002). Alterations in left ventricular torsion in tachycardia-induced dilated cardiomyopathy. *J Thorac Cardiovasc Surg, 124*(1):43-49.

Tischler, M., & Niggel, J. (2003). Left ventricular systolic torsion and exercise in normal hearts. *J Am Soc Echocardiogr, 16*(6):670-674.

Warburton, D. E., Gledhill, N., Jamnik, V. K., Krip, B., & Card, N. (1999). Induced hypervolemia, cardiac function, VO2max, and performance of elite cyclists. *Med Sci Sports Exerc, 31*(6):800-808.

Whyte, G. P., George, K., Shave, R., Middleton, N., & Nevill, A. M. (2008). Training induced changes in maximum heart rate. *Int J Sports Med, 29*(2):129-133.

Zerhouni, E. A., Parish, D. M., Rogers, W. J., Yang, A., & Shapiro, E. P. (1988). Human heart: tagging with MR imaging--a method for noninvasive assessment of myocardial motion. *Radiology, 169*(1):59-63.