Left ventricular torsion and recoil: implications for exercise performance and cardiovascular disease

Ben T. Esch and Darren E. R. Warburton
Cardiovascular Physiology and Rehabilitation Laboratory, University of British Columbia, Vancouver, British Columbia, Canada

Submitted 6 February 2008; accepted in final form 5 November 2008

Esch BT, Warburton DE. Left ventricular torsion and recoil: implications for exercise performance and cardiovascular disease. J Appl Physiol 106: 362–369, 2009. First published November 6, 2008; doi:10.1152/japplphysiol.00144.2008.—In recent years, advancements in echocardiography assessment techniques have allowed for the quantification of left ventricular (LV) rotation. This information has provided new insight into LV function in health and disease. In this review, we discuss the importance of assessing LV circumferential rotation for understanding cardiac function in a wide range of populations. We provide a synopsis of LV rotational mechanics in the context of the various techniques currently available to assess LV rotation. We also highlight the factors that alter LV function at rest and during exercise. Finally, we discuss the influences of age, sex, and cardiac pathology on LV rotation. Collectively, this review highlights the importance of understanding LV rotation and its measurement in both health and disease.

diastolic suction; endurance athletes; ventricular recoil

RECENT TECHNOLOGICAL ADVANCES in echocardiography provide the ability to quantify left ventricular (LV) function across its three planes of motion: longitudinal, radial and circumferential (61). Tissue Doppler along with speckle-tracking imaging and analysis allow for the assessment of myocardial shortening (strain) and rates of shortening (strain rates) as well as of tissue velocities and tissue displacements (linear and angular) throughout the cardiac cycle. One of the most intriguing outcomes from these technological advancements is the ability to quantify LV rotation.

As will be discussed in this review, the importance of measuring LV rotation lies in its potential for early detection of pathology (25), the ability of torsion techniques to provide regional information, as well as the importance of LV rotation as an indicator of active diastolic relaxation (6). Torsion and diastolic recoil may be of particular importance when the cardiac cycle is abbreviated during exercise. Therefore, this review will consider the potential implications and uses for the study of LV circumferential rotation in health and disease.

LV TORSION AND RECOIL

Mechanics of torsion. Atrial-ventricular plane displacement has recently been shown to account for ~60% of stroke volume (8). The remaining contribution to stroke volume is from radial compression and LV circumferential twisting. LV twisting was first described by Harvey in 1628 (26). Looking from the ventricular apex, during systole the apex rotates counterclockwise, whereas the LV base rotates clockwise resulting in torsion (Fig. 1) (20, 40). Untwisting occurs in the opposite direction during early diastole, resulting in diastolic recoil. For the purposes of this review, twisting and untwisting are the rotational deformations (measured in degrees) for either the apex or base of the heart, and torsion and recoil are the net difference between the apex and base. Rotation is used as a blanket term to include both systolic and diastolic indexes of LV circumferential motion. LV torsion and recoil are a result of the dynamic interaction between obliquely oriented epicardial and endocardial fibers wound oppositely (56). Streeter et al. (56) elegantly showed the transition in myocardial fiber orientation in stages from the endocardium to the (relatively) perpendicular midwall, and to the opposite angles of the epicardium. The left-handed helix of the epicardium dominates rotational motion due to its longer lever arm from the center of the LV. The endocardial layer, with a right-handed helix, moves together with the epicardium, although providing some opposition to epicardial motion (75). Systolic twist acts to limit myocardial energy expenditure by creating high intraventricular systolic pressures with minimal muscle shortening, resulting in efficient LV contraction (3). The resultant recoil of the LV has important implications for diastolic filling. It should be duly noted that each systolic contraction has a significant impact on the following diastole (45). In fact, the elastic recoil that occurs during early diastole is thought to be a result of the vigorous contraction and compression of cardiac proteins such as titin (21, 31). The potential energy stored in the springlike titin is unleashed during diastole, aiding myocardial relaxation and diastolic filling (21, 31). A portion of the energy released during recoil may also be generated from the release of shear strains built up between the endocardial and epicardial layers (33, 48).

The effective filling pressure of the LV is the difference between left atrial pressure and LV pressure, also known as the transmitial pressure gradient. During systole, LV pressures are high, and the mitral valve is closed. After systolic contraction, LV pressures begin to fall, the aortic valve closes, and isovolumetric relaxation commences, resulting in a further drop in LV pressure. Once left atrial pressure is greater than LV pressure the mitral valve opens and early diastolic filling occurs. The rate of blood flow into the LV during early diastole is directly proportional to the transmitial pressure gradient (12, 34). Early diastolic filling occurs until left atrial and LV pressures are approximately at equilibrium, and then, finally, atrial contrac-
During systole, the apex twists in a counterclockwise direction to aid with the ejection of blood. However, the rapid recoil (untwisting) of the myocardium, which primarily occurs during isovolumetric relaxation, has significant effects on both LV and transmitral pressures (44). Rapid untwisting during isovolumetric relaxation is crucial in the development of low LV pressures (44). Delayed or prolonged untwisting results in ineffective recoil, with little benefit to the transmitral pressure gradient (20, 44, 47). Rapid myocardial untwisting in early diastole has been shown to contribute to the occurrence of diastolic suction, whereby minimum LV pressure may become relatively negative (or increasing chamber dimension despite declining pressures) (64). A lower LV pressure (assuming a constant left atrial pressure) results in a greater transmitral filling gradient and a higher rate of flow (12, 34). Therefore, any perturbation that could affect the rate of untwisting either acutely (e.g., reduced end-systolic volume) or chronically (e.g., endurance training) could significantly impact transmitral filling and ultimately stroke volume.

Diastolic filling rates, transmitral pressures, and LV rotation during exercise. An elevated heart rate during exercise results in reduced diastolic filling time (23). Consequently, diastolic function (and diastolic filling rate) must be augmented during exercise to attain the same LVEDV in a shorter amount of time. Endurance-trained athletes have larger increases in LVEDV and stroke volume compared with untrained individuals, and, therefore, they must have a more dramatic increase in diastolic filling and function during exercise (23, 70). Doppler echocardiography has been used to show an increase in the early diastolic peak filling velocity with exercise and a greater increase in endurance athletes compared with normal individuals (13, 59). These findings are all indicative of enhanced filling rates in endurance athletes during exercise. Numerous mechanisms could contribute to greater LV filling and EDV in endurance athletes including greater blood volumes (14, 68), increased LV compliance (38), reduced diastolic ventricular interactions (17), as well as potentially greater increases in the transmitral pressure gradient and LV rotation with exercise.

Increased early diastolic filling velocities during exercise suggest an increase in the transmitral pressure gradient. Numerous researchers have shown the transmitral pressure gradient (or the intraventricular pressure gradient measured from apex to base) to be increased during exercise (9, 44, 52). In the animal model, Cheng et al. (9) demonstrated an increase in the transmitral pressure gradient as a result of a reduced minimal LV pressure without a significant change in left atrial pressure. Because of the reductions in minimal LV pressure, there was a downward shift in the diastolic portion of the pressure-volume relationship during exercise compared with rest. The net result was an increased LVEDV and stroke volume despite a shorter diastolic filling time (9). Similar increases in the transmitral pressure gradient as a result of reductions in minimal LV pressure have been shown in humans during dobutamine infusion (65).

Recently, Notomi et al. (44) have demonstrated that ventricular torsion and recoil increase during submaximal exercise in

---

Fig. 1. The left ventricle (LV) on the left is shown in diastole; the LV on the right depicts (viewed from the apex) the counterclockwise rotation of the apex and the clockwise rotation at the base during systole. Note: the vertical lines on the LV do not represent fiber orientation, but are present to illustrate the direction of torsion.

Fig. 2. Top left: LV in diastole. Bottom left: LV ejection without torsion. Note the much greater degree of endocardial shortening. Top right: LV torsion without ejection. Note the endocardial expansion with epicardial shortening. Bottom right: LV ejection and torsion together allow for optimal shortening of both the endocardium (endo) and epicardium (epi). +, Myocardial fiber shortening; −, myocardial lengthening; more − means greater shortening. [From Lumens et al (39).]
healthy individuals. Interestingly, these authors demonstrated a sequential time course between peak diastolic untwisting rates, peak intraventricular pressure gradient (an indicator of diastolic suction) (42), and peak early diastolic filling velocity (Fig. 3). The sequential nature of their findings suggests the important contribution of each phase on the following step; that is, untwisting aids in generating the pressure gradient, and the pressure gradient is essential for peak diastolic filling velocity. Notomi and colleagues also reported that during exercise the greatest relative increase in proportional contribution to stroke volume is from LV circumferential motion. It should be noted that the participants in the study by Notomi et al. were exercising at a relatively low intensity (heart rate of ~112 beats/min) for a short period of time. Others have also shown increases in torsion and recoil with low intensity acute exercise, or after exercise (41, 63). Stuber et al. (57) found no differences in resting LV torsion and recoil between healthy normal individuals and competitive rowers; however, no exercise data were obtained. In contrast, Nottin et al. (48) found apical rotation and LV torsion to be reduced in elite cyclists (48). They suggest that their findings may be related to differences in heart rates between athletes and nonathletes or to structural adaptations of the myocardium. Interestingly, Nottin et al. found differences between endocardial and epicardial torsion in their control group but no such transmural differences in the athletes. They also suggested that a lower torsion at rest in the athletes may allow for a greater torsion reserve during exercise; however, exercise torsion was not measured in their investigation. Interestingly, Rovner et al. (52) demonstrated that the presence of higher intraventricular pressure gradients (i.e., greater LV suction) was directly related to maximal oxygen consumption (Fig. 4). There has yet to be a systematic assessment of LV torsion and recoil during incremental or steady state prolonged exercise, and there have been no systematic comparisons between endurance-trained athletes, sedentary individuals, and cardiac patients. A partial explanation for the lack of rotational data during exercise is due to the technical difficulty of obtaining high quality ultrasound images during high ventilations and heart rates. However, the work of Cheng and colleagues (9) as well as Notomi et al. (44), suggest that LV torsion and recoil may play a significant role in the augmentation of diastolic function seen during exercise.

MEASUREMENT OF VENTRICULAR TORSION

Quantification of LV torsion and recoil has been conducted primarily with three methods: implanted myocardial markers, magnetic resonance image (MRI) tissue tagging, and echocardiography (tissue Doppler and speckle tracking). Implanted radiopaque metallic markers or sonomicrometers in the myocardium can be established during clinically indicated cardiac surgery, but the invasive nature of this procedure makes them rare, if not unnecessary, in humans (considering the newer noninvasive technologies).

MRI is the current gold-standard for measuring LV rotation (46). To track the degrees of rotation throughout the cardiac cycle, the myocardium is “tagged” using a unique pulse sequence to manipulate the longitudinal magnetization of the myocardium (49, 76). Analyzing the movement of tissue tags provides quantification of the dynamic behavior of the myocardium. The technique of tagged MRI has been used in numerous investigations to assess torsion and recoil in a wide range of populations (20, 40, 46, 49). The assessment of LV torsion and recoil with MRI requires the acquisition of cross-sectional (short axis) images at both the LV apex and base (level of the mitral valve). The major advantages of MRI-derived torsion measurements are the spatial resolution and precision of the tissue tagging. High-quality MRI images with tissue tagging can also allow for separate assessments of the endocardium and epicardium (39). Conversely, the temporal resolution is relatively slow compared with echocardiographic methods. As with echocardiography, MRI techniques are still limited by a two-dimensional image. The spatial constraints of the magnet as well as the accessibility and affordability of MRI make it less practical than echocardiography for many researchers.

**Fig. 3.** Mean LV torsional velocity at rest (A) and during submaximal exercise (B) in healthy individuals. The data are presented over time as a percentage of systolic duration, where 0% is end diastole (onset of QRS interval) and 100% is aortic valve closure. The vertical lines signify valve timing (MC, mitral valve closure; AO, aortic valve opening; AC, aortic valve opening) as well as aortic (EJ, peak aortic ejection velocity) and mitral (PK-E, peak early mitral filling velocity) peak flow velocities. At rest and during exercise, peak IVPG was found to occur between MO and PK-E. Generally, positive values are indicative of systolic twisting and negative values are indicative of untwisting in diastole. [Adapted from Notomi et al. (44).]
Recently, developments have been made to allow for the assessment of torsion and recoil using two-dimensional and Doppler echocardiography (for a review see Ref. 60). Numerous recent publications have demonstrated the abilities of slightly different methods of analysis to arrive at a similar outcome (1, 43, 46). Myocardial tissue velocities can be tracked at the base and apex in the short-axis plane allowing for the determination of rotational velocities (46). The tissue Doppler technique of assessing torsion has been shown to correlate quite well with MRI [r = 0.84 (46)]; however, as with all Doppler recordings there is concern about angle dependency. Two-dimensional ultrasound speckle tracking has been proposed as a less angle and velocity dependent measure of LV torsion (29, 43). Structures smaller than the wavelength of the ultrasound beam cause constructive and destructive interference that results in speckle patterns within the tissue (43). Speckle motion is highly related to myocardial motion, particularly when small displacements are involved (as in circumferential rotation) (29, 43). The speckles are tracked throughout the cardiac cycle to quantify the rotational displacement of the myocardium (43). After manually tracing the endocardial border, computer software is used (for example, EchoPAC, GE Healthcare) to automatically select “speckles” and then track them frame by frame using the sum of absolute difference algorithm (37). As with MRI, short-axis, two-dimensional images are required at the apex and base (mitral valve level) (60). Circumferential motion (in degrees) and rates of rotation (degrees/second) are provided by the software for both a mean and immediately following peak exercise. [From Rovner et al. (52).]

Factors affecting LV rotation during acute exercise and exercise training. Given that the previously discussed factors have been shown to affect LV rotation and minimum LV pressure generation, let us consider the exercising human model. Heart rate, venous return, contractility, and sympathetic stimulation all increase with the onset of exercise. Conversely, because of an elevation in cardiac output, systolic blood pressure (a commonly used surrogate for ventricular afterload) increases throughout exercise. As previously discussed, an elevation in afterload is detrimental to LV torsion. However, the adverse effects of an elevated afterload are likely offset by an increase in all of the other factors that affect torsion. Therefore, it is not surprising that numerous investigators have shown torsion to increase with exercise (41, 44, 63).

**FACTORS THAT AFFECT LV ROTATION**

Numerous factors have been implicated in varying LV rotation. Alterations in preload, afterload, contractility, heart rate, and sympathetic activation have been shown to alter rotation. Dong et al. (15) manipulated preload, afterload, and contractile state (via dobutamine infusion) independently in an isolated canine heart preparation (15). The directly proportional relationship between torsion and LVEDV and the inversely proportional relationship between torsion and end-systolic volume in the study by Dong et al. exhibit the volume dependency of LV torsion. In fact, many others have shown a low end-systolic volume to be the strongest predictor of increases in untwisting rate and diastolic suction (45, 64, 67). In the same investigation, Dong et al. fixed LVEDV and end-systolic volume during dobutamine infusion. Dobutamine resulted in an elevated peak systolic pressure and an increase in LV torsion and recoil, demonstrating the force dependent nature of LV rotation. Although Cheng et al. (9) did not measure torsion directly; they demonstrated that generation of a lower minimal LV pressure was affected by both heart rate and sympathetic stimulation (9). Conversely, Wang et al. (67) found no relationship between heart rate and peak untwisting rates in heart failure patients.

**Factors affecting LV torsion during acute exercise and exercise training.** Given the previously discussed factors have been shown to affect LV rotation and minimum LV pressure generation, let us consider the exercising human model. Heart rate, venous return, contractility, and sympathetic stimulation all increase with the onset of exercise. Conversely, because of an elevation in cardiac output, systolic blood pressure (a commonly used surrogate for ventricular afterload) increases throughout exercise. As previously discussed, an elevation in afterload is detrimental to LV torsion. However, the adverse effects of an elevated afterload are likely offset by an increase in all of the other factors that affect torsion. Therefore, it is not surprising that numerous investigators have shown torsion to increase with exercise (41, 44, 63).
Examining the factors that affect LV rotation, and knowing that these factors generally increase during acute exercise, the question then becomes: Does chronic endurance training result in changes to these factors, ultimately increasing LV rotation during exercise? It has been established that endurance exercise training results in an increased blood volume (11, 14, 71). Increased blood volume is one of the major adaptations to endurance training and it has a direct impact on LVEDV and stroke volume (69). Taken independently, because of higher LVEDV, endurance athletes would be expected to have greater LV rotation compared with untrained individuals during exercise (15). Using systolic blood pressure as a surrogate for afterload, Gledhill et al. (23) demonstrated that at matched heart rates, endurance athletes have a significantly reduced systolic blood pressure. Based on differences in afterload alone, endurance-trained athletes would exhibit higher degrees of LV torsion and recoil during exercise. Thus training-induced changes in total blood volume and/or afterload may account for higher LV torsion and recoil during exercise.

Small increases in contractility have been shown to occur with exercise training (53, 70), which could influence the force dependency of torsion in endurance-trained athletes. Altered LV torsion and recoil as a result of chronic exercise training may also be due to changes in heart rate. Intrinsic heart rate has been shown to be reduced in endurance athletes (35), and lower resting heart rates have been shown to be related to reduced resting torsion in athletes (48). As well, peak heart rates have been shown to be reduced in trained individuals (73). Despite potentially having lower resting and peak heart rates, the greater heart rate reserve found in athletes may prove to be beneficial for increasing torsion with exercise.

Concentric hypertrophy caused by pressure overload has been shown to have detrimental consequences for LV torsion (57). Resting LV torsion and recoil were found to be reduced in elite cyclist who exhibited an increase in end-diastolic diameter, wall thickness, and LV mass (48). It is possible that structural adaptations to the endocardium or epicardium may have resulted in these changes. However, the effects of training induced LV eccentric hypertrophy on LV torsion and recoil during exercise have yet to be determined.

Interestingly, Dorfman and colleagues (16) have shown that cardiac deconditioning induced by 18 days of head-down tilt resulted in reduced LV untwisting. However, individuals undergoing head-down tilt were able to increase LV untwisting rates by exercising in the supine position (16), emphasizing the important influence of exercise training on LV recoil.

Factors affecting LV torsion during prolonged exercise. To date, no studies have examined the effects of sustained (1 h or more) exercise on LV rotation. However, numerous investigations have shown alterations in parameters that affect LV rotation or could be a consequence of reductions in LV rotation. It has been clearly established that reductions in ejection fraction and contractility can occur following prolonged exercise (generally longer than 4 h) (55, 72) and both reductions in contractility and higher end-systolic volumes have been related to reductions in LV torsion and recoil (15, 64). As discussed above, sympathetic stimulation can directly influence LV rotation, and the cardiac response to sympathetic activation has been shown to be blunted after prolonged strenuous exercise (55, 72). In addition, indexes of diastolic function such as Doppler tissue velocity and early diastolic filling velocity are often reduced following prolonged exercise, which may also indicate reductions in the early transmitral filling gradient (22, 55, 72). Whether these factors contribute to reduced LV rotation or are a consequence of it, it seems highly plausible that LV torsion and recoil are altered following prolonged exercise. This remains an area that requires further research to confirm these hypotheses.

THE EFFECTS OF AGE AND SEX ON LV TORSION

Torsion across the life span. Important functional changes occur in the myocardium from infancy to late adulthood. Some of these alterations are directly related to LV rotation. Notomi and colleagues (47) assessed LV torsion and twisting velocities in individuals from 9 mo to 49 yr and found that with advancing age there was an increase in LV torsion and untwisting velocity. However, when torsion was normalized for LV length, there was a decline in torsion and untwisting velocity during childhood (3−10 yr old) with an increase to above infant levels in middle aged adults (35−49 yr old) (47). Interestingly, Notomi et al. found that in infancy, both basal and apical rotation were counterclockwise during systole, but throughout childhood and adolescents basal systolic rotation gradually became more clockwise resulting in ventricular torsion. As well, the timing of LV untwisting in infancy was less effective than in adults since the majority of untwisting in infants occurs in conjunction with or after mitral valve opening. A delay that prevents a larger contribution of LV untwisting to reduced transmitral gradients (47). Several other investigations examining older individuals have shown LV torsion to be maintained or increased compared with younger adults (5, 28, 50). However, others have shown intraventricular pressure gradients to be reduced in the elderly (51). Indeed, Burns et al. (5) found LV torsion to be elevated in older males at rest, yet the augmentation (or reserve) of torsion during exercise was shown to be significantly less in the older cohort. Lumens et al. (39) found reduced endocardial circumferential shortening in elderly individuals. They suggested that reduced endocardial function would result in less opposition to the dominant epicardium ultimately causing elevated rotation. It has been proposed that endocardial function is more likely to reduce with age due to the subendocardium’s greater susceptibility to fibrosis and/or subclinical reductions in perfusion (7, 66). The finding of reduced subendocardial function and increased torsion in older individuals occurred despite no abnormalities in ejection fraction or LV mass, indicating measurements of torsion may be important for the early detection of cardiovascular dysfunction (39). Further details examining the effects of exercise on LV torsion in individuals of all ages warrants further investigation.

Sex differences. Numerous investigations examining LV rotation have included both male and female participants (29, 44, 47, 50). However, because the data were pooled, any potential sex differences remain unknown. Differences in sympathetic activity, systolic function, LV chamber compliance and blood volume between women and men may result in some disparities in LV rotation (18, 27, 32, 54, 74). If the assessment of torsion is to become an important clinical and research tool, normative data in a wide range of populations, including females, must be identified.
**LV ROTATION AND CARDIAC DYSFUNCTION**

The importance of quantifying LV rotation lies in its potential for guiding clinical treatment. Measurements of peak torsion, the time of peak torsion, as well as the rate and time of peak untwisting provide further insight into pathology beyond traditional clinical measures such as ejection fraction and Doppler indexes of diastolic function. Numerous explanations for alterations in LV torsion in cardiac patients have been proposed, including 1) remodeling of the myocardium, causing less mechanically advantageous orientation for contraction; and 2) degradation of the extracellular matrix, which aids in the storing of potential energy during systolic contraction to be released during early diastole (4, 62). It is also possible that alterations in regional myocardial blood flow (e.g., the endocardium) or electrical activation may result in altered patterns of LV rotation in cardiac patients. Therefore, the ability to easily quantify and track changes in LV rotation may provide further insights into cardiac structure and function beyond traditional measures. As an example, Hansen et al. (25) have shown that despite no change in ejection fraction, reductions in torsion and torsion rate were found in patients undergoing acute cardiac allograft rejection (25). As the patients recovered, torsion returned to prerejection levels. In addition, LV torsion has been shown to be increased in diabetics individuals (Type 1 and 2) (10, 19), and it is suggested that this may be a precursor to the myocardial alterations found in diabetic cardiomyopathy (10).

Reductions in peak and peak rates of torsion and recoil have been shown to be reduced in heart failure patients with a reduced ejection fraction (20). In dilated cardiomyopathy, alterations of the timing of diastolic untwist have been shown to occur, whereby less rotation occurs in the isovolumetric phase of diastole, resulting in less effective untwisting (62). Conversely, Wang et al. (67) have shown that heart failure with preserved ejection fraction have normal LV twist and untwist. The differences observed between patients with systolic and diastolic heart failure may lie in differences in chamber geometry or in the inability of patients with systolic heart failure to reduce their end-systolic volume (48).

Patients with systolic heart failure and nonuniform electromechanical contraction are candidates for cardiac resynchronization therapy (CRT) (24). Recent investigations have shown the ability of speckle-tracking derived strain in different regions (circumferentially) to aid in the placement of pacing leads, that is, target the area of greatest delay in contraction (58). Investigators have shown that the degree to which circumferential shortening is synchronized can be used to predict which patients will respond to CRT (30), whereas others have shown no change in LV torsion following CRT despite improvements in ejection fraction (77). There is no doubt that the multidimensional (longitudinal, radial, and circumferential) and regional (around the circumference of a two-dimensional image) information provided by tissue Doppler and speckle tracking analysis is of great benefit for the advancement of CRT treatment and outcomes. More research is required in this emerging area of research to help identify the predictive and evaluative powers of assessing LV rotation in patients undergoing CRT.

Individuals with chronic aortic stenosis have been shown to display elevated peak LV torsion compared with healthy controls (57). Numerous investigations have demonstrated increased LV torsion in the face of chronically increased afterload, which has been attributed largely to wall thickening and altered fiber orientation (40, 57). However, those with aortic stenosis exhibit delayed diastolic untwisting, where a greater proportion of untwisting occurs after mitral valve opening, indicating less effective diastolic recoil and reduced early diastolic filling (40, 57).

The above discussion provides a few examples of how measures of LV rotation can be used to provide additional insights into myocardial structure and function which go beyond basic traditional imaging measures. The further potential of LV rotational analysis may be seen during physiological stress as the heart may adequately compensate for dysfunction at rest, yet be limited and exposed during exercise. As an example, Notomi et al. (44) found elevated resting torsion in patients with hypertrophic cardiomyopathy compared with healthy individuals, yet the patients were unable to augment torsion and untwisting with exercise. Therefore, the importance of incorporating measures of LV rotation into standard stress-echocardiography may provide clinicians with further insight into cardiac dysfunction.

**SCALING OF LV ROTATION**

One important issue in the assessment of LV rotation, particularly when comparing different populations, is the normalization of data. Be it athletes, children, or cardiac patients, heart size will have an impact on LV rotation. Normalizing torsion to LV length has been proposed as a method for comparing different groups (47). As well, others have shown torsion to be related to the ratio of LV wall thickness to LV radius, a measure which could potentially be incorporated into a normalization of torsion allowing comparisons across groups (39).

**CONCLUSIONS**

LV rotation has important implications for diastolic function both at rest and during exercise. The timing and magnitude of rotation have direct influences on the development of transmural pressure gradients and ultimately LV filling. Recently advances in technology have made the quantification of LV rotation more available as both a research and clinical tool. The importance of assessing LV torsion lies in the ability to quantify ventricular function more accurately spatially (over different planes of motion and regions of the heart) and temporally (e.g., for mechanical synchrony or for examining myocardial motion compared with valve events). Measuring LV rotation may become an important clinical tool as it appears to be more sensitive to dysfunction than some of the traditional echocardiography measures.

**GRANTS**

B. T. Esch was supported by the Natural Sciences and Engineering Research Council of Canada (NSERC) and the Michael Smith Foundation for Health Research (MSFHR) scholarships. D. E. R. Warburton’s laboratory is currently supported by the Canada Foundation for Innovation, the British Columbia Knowledge Development Fund, and the NSERC. D. E. R. Warburton is currently a MSFHR Clinical Scholar and a Canadian Institutes of Health Research New Investigator. The authors also thank GE Healthcare and Dr. Jack Taunton for their continued support of the Cardiovascular Physiology and Rehabilitation Laboratory.
REFERENCES


LV ROTATION AND EXERCISE


