EDITORIAL COMMENT

Ventricular Torsion
An Aid to Ejection?*

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Ventricular torsion has long been recognized as a feature of normal mammalian cardiac function (1). During systole, the apex of the left ventricle rotates anticlockwise and the base clockwise, as viewed from the apex. This characteristic wringing motion is clearly visualized with magnetic resonance tissue tagging (2) or echocardiographic speckle tracking (3). It has been shown to be a sensitive marker of transplant rejection, successful ventricular reconstruction surgery, infarction, and diastolic dysfunction (1).

Torsion is an interesting measure of myocardial tissue performance since it can occur independently of pump function (although both are clearly driven by myocyte contraction). Torsion by definition is a shear deformation that does not in itself give rise to ejection or filling, unlike myofiber shortening or wall thickening. It therefore does not require a volume change to occur. Although torsion typically increases approximately linearly with ejection (relative to end-diastole), much of the end-systolic torsion is usually released during isovolumic relaxation (4). This is likely due to the mechanical release of stored elastic energy in the myocardial tissue. Torsion occurs due to the fibrous architecture of the mammalian heart, with a myocyte helix angle varying from $-60^\circ$ below equatorial on the epicardial surface to $80^\circ$ above equatorial on the endocardial surface. Endocardial and epicardial myocytes, therefore, act in torsional opposition, with the greater lever arm of the epicardial fibers typically winning out. This theory of mechanical action is borne out by mathematical models that can predict the magnitude of torsion due to normal fiber architecture (5) as well as situs inversus totalis (6).

There are several ways of quantifying torsion, leading to some confusion in the literature. Although commonly used, a simple difference in rotation between apex and base (often called twist) is not recommended, since this depends on the exact locations sampled and is difficult to reproduce in longitudinal studies. Twist per unit length of the ventricle is more robust but does not scale appropriately between hearts of different sizes (e.g., mice and humans have comparable ventricular torsion but quite different twist per length). A better measure is the torsional shear angle, which can be calculated using 2 short-axis slices as the twist multiplied by the mean radius and divided by the distance between slices (7). However, in order to understand torsion in relation to other strain components, a full 3-dimensional (3D) reconstruction of the strain tensor is required.

In this issue of iJACC, Ahmed et al. (8) investigate the role of torsion as a mechanism for increased ejection fraction in patients with resistant hypertension, despite reduced in-plane (circumferential and longitudinal) strain. The authors employ a state-of-the-art 3D strain analysis that enables calculation of in-plane strains, torsional shear angle, and 3D principal strain (the maximal contraction, which occurs in an oblique direction not aligned with any imaging plane). Torsion was found to be increased in the patient group, relative to the somewhat younger and leaner controls. This increased torsion led to maintained 3D principal shortening strains despite reduced in-plane strains in the patient group. The demonstration of a changed orientation of 3D principal shortening due to the increased torsion is indicative of the power of a 3D analysis.
technique. Wall thickening was also increased and the authors conclude that the increased torsion is linked to the increased wall thickening and increased ejection fraction in these patients. This is an interesting hypothesis that deserves further investigation. However, care must be taken in interpreting torsion as a mechanical substrate for ejection, since it does not in itself lead to volume change or wall thickening.

Previous studies using 3D cardiac magnetic resonance tagging have found increased torsion in the presence of reduced or maintained in-plane strain in a variety of hypertensive and hypertrophic pathologies, including mild hypertension late after repair of coarctation of the aorta (9), hypertrophic cardiomyopathy (2), and diabetic cardiomyopathy (10). This may be due to the increased concentric hypertrophy leading to an increased lever arm for the epicardial fibers. Another mechanism is that reduced endocardial shortening may also lead to increased torsion (11) since the forces opposing torsion are reduced. Mathematical models suggest that there is an optimal and load-independent torsion-to-shortening ratio (TSR) for the mammalian heart in which fiber shortening is approximately homogeneous from epicardium to endocardium (5). Increased TSR has been proposed as a marker of subendocardial tissue function (11,12). Torsion and TSR were also increased in healthy carriers of familial hypertrophic cardiomyopathy with normal wall thickness (13), perhaps indicating preclinical disease.

Concentric hypertrophy itself is known to lead to maintained or increased ejection fraction, even with reduced in-plane strain, due to the (near) incompressibility of myocardial tissue. For example, simple mathematical models show that increased concentric hypertrophy (as measured by the wall thickness to radius ratio) can lead to an increased ejection fraction, even in the presence of reduced circumferential strain, on the grounds of geometric constraints alone (14). However, the substantial wall thickening that is observed can only occur by the mechanism of transverse shear, in which cells slide over one another in the radial (transmural) direction (15). This shear is facilitated structurally by transmural myocardial laminae in the connective tissue, which can be observed by confocal and electron microscopy (15,16). The direction of maximum local shearing is aligned with the laminae orientation in the subendocardium, where the largest deformations occur.

When measured with respect to the canonical axes of the heart, 2 types of transverse shear can be found: one due to a difference in longitudinal motion between epicardium and endocardium (longitudinal-radial shear) and the other due to a difference in rotational motion between epicardium and endocardium (circumferential-radial shear). The latter typically occurs due to an increased rotation of the endocardium relative to the epicardium: at the apex the endocardium rotates more anticlockwise whereas at the base, the endocardium rotates more clockwise (2). This increased ventricular torsion at the endocardium relative to the epicardium, therefore, facilitates wall thickening by the mechanism of circumferential–radial transverse shear. However, there is currently no explanation as to the mechanism of this phenomenon. In fact, the increased endocardial torsion is mechanically paradoxical since endocardial fibers act in opposition to the epicardial fibers and would, in the absence of other effects, lead to a reduced endocardial torsion rather than the reverse. One possible mechanism suggested by Ubbink et al. (17) is the existence of transversely oriented myofibers, which can more effectively transmit epicardial forces to the endocardium. However, the significant transverse angles necessary for this have not been histologically confirmed. Another mechanism might be the connective tissue laminae themselves (15). Clearly, the mechanical mechanisms of wall thickening through transverse shear need further investigation.

The paper by Ahmed et al. (8) in the current issue of iJACC, therefore, raises important questions about the relationship between torsion and ejection that deserve further study. A combination of noninvasive imaging and mechanical models are required to determine why torsion is increased on the endocardium and the coupling mechanism that generates transverse shear and wall thickening. The relationships predicted by mechanical models between torsion and shortening need to be investigated experimentally in different pathologies. Although torsion is important, it is 1 component of the 3D strain tensor, and imaging the transverse shears will be necessary for future investigations of wall thickening and pump function. Higher-resolution, 3D noninvasive strain imaging methods, such as transmural speckle tracking (18) or 3D transmural displacement imaging with displacement-encoded stimulation echo cardiac magnetic resonance (19), will be very useful in these investigations.

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Key Words: hypertension ▪ left ventricular mechanics ▪ torsion ▪ transverse shear.