Detailed information can now be obtained on the microstructural architecture of the heart. This microstructure has a significant impact on cardiac performance. This lecture will give course participants an overview of our current understanding of the relationships between cardiac microstructure and cardiac function. Recent clinical applications will be reviewed and areas of productive future research will be highlighted.

Since William Harvey speculated that the fibrous architecture of the heart must have a substantial influence on its mechanical operation (1), there has been a great deal of research on function-structure relationships in cardiac mechanics (2). In particular, left ventricular torsion is an interesting index of cardiac performance which provides important information on myocardial mechanics over and above standard pump function indices. Mathematical models, which solve the Newtonian equations of motion in a continuum stress balance, have shown that the normal distribution of myofiber orientation in healthy subjects equilibrates fiber contraction and fiber stress across the wall, so that myocytes contract uniformly from epicardium to endocardium (3,4). Torsion is markedly different in conditions where the myofiber architecture is not normal. For example, in situ versus totalis, the apical and epicardial basal fiber orientation is normal but the deeper basal fibers have an inverted fiber orientation. Torsion is consequently normal at the apex but changes sign towards the base (5).

Mathematical models have shown that a specific relationship between torsion and ejection is required to balance forces and maintain a uniform fiber shortening across the wall (6). This leads to a constant ratio between shortening and torsion during systole across mammalian species, known as the torsion-to-shortening ratio (TSR) (7) which is theoretically independent of contractility, afterload and preload. Changes in this ratio therefore indicate transmural differences in fiber contraction. TSR has been shown to be substantially increased in patients with aortic stenosis (7) with smaller increases seen in normal aging (8). Recently, Russel et al. (9) found increased torsion and TSR in HCM mutation carriers with normal wall thickness, perhaps indicating preclinical subendocardial dysfunction.

The substantial wall thickening which occurs during systole cannot be solely due to myocyte thickening, so substantial shear deformations must act to facilitate wall thickening, and therefore pump function. These transverse shears are mechanically facilitated by myocardial laminae and maximum local shearing is aligned with the laminae orientation in the subendocardium (10-12). One source of transverse shear is due to a difference in rotational motion between epicardium and endocardium (circumferential-radial shear). MRI tagging studies have shown that the endocardium rotates more anticlockwise at the apex while at the base the endocardium rotates more clockwise than the epicardium (13). This increase in torsion towards the endocardium gives rise to a characteristic circumferential-radial shear pattern which varies longitudinally from positive in the apex to negative in the base, passing through zero in the middle (13). This is paradoxical since subendocardial fibers mechanically reduce torsion at the subendocardium, not increase it. One possible mechanism may be the existence of transverse myocardial fibers which can more effectively transmit epicardial forces to the endocardium (14). Although there is some evidence of transverse angle from DTMRI (15), whether this is sufficient to account for the observed transverse shear is not yet known.

Recently, computational physiological models of cardiac function have been used to estimate biophysical parameters underlying cardiac function (16). These modeling methods combine deformation and structural imaging into a mechanical analysis, solving the force balance equations using finite element analysis. By customizing the
model parameters to a patient’s images, insights can be obtained into the underlying physiological causes of cardiovascular disease. 

In summary, the relationships between myocardial microstructure and cardiac function are a fruitful area of study and show promise in the enhancement of patient treatment.

1. Harvey W. Exercitatio anatomica de motu cordis et sanguinis in animalibus. 1628.