A Clinician's Guide to Tissue Doppler Imaging

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Tissue Doppler imaging (TDI) is a relatively new echocardiographic technique that uses Doppler principles to measure the velocity of myocardial motion. We describe the principles behind and the clinical utility of TDI.

Principles of TDI

Doppler echocardiography relies on detection of the shift in frequency of ultrasound signals reflected from moving objects. With this principle, conventional Doppler techniques assess the velocity of blood flow by measuring high-frequency, low-amplitude signals from small, fast-moving blood cells. In TDI, the same Doppler principles are used to quantify the higher-amplitude, lower-velocity signals of myocardial tissue motion.

There are important limitations to TD interrogation. As with all Doppler techniques, TDI measures only the vector of motion that is parallel to the direction of the ultrasound beam. In addition, TDI measures absolute tissue velocity and is unable to discriminate passive motion (related to translation or tethering) from active motion (fiber shortening or lengthening). The emerging technology of Doppler strain imaging provides a means to differentiate true contractility from passive myocardial motion by looking at relative changes in tissue velocity.

TDI can be performed in pulsed-wave and color modes. Pulsed-wave TDI is used to measure peak myocardial velocities and is particularly well suited to the measurement of long-axis ventricular motion because the longitudinally oriented endocardial fibers are most parallel to the ultrasound beam in the apical views. Because the apex remains relatively stationary throughout the cardiac cycle, mitral annular motion is a good surrogate measure of overall longitudinal left ventricular (LV) contraction and relaxation.[1]

To measure longitudinal myocardial velocities, the sample volume is placed in the ventricular myocardium immediately adjacent to the mitral annulus. The cardiac cycle is represented by 3 waveforms (Figure): (1) Sa, systolic myocardial velocity above the baseline as the annulus descends toward the apex; (2) Ea, early diastolic myocardial relaxation velocity below the baseline as the annulus ascends away from the apex; and (3) Aa, myocardial velocity associated with atrial contraction. The lower-case "a" for annulus or "m" for myocardial (Ea or Em) and the superscripted prime symbol (E') are used to differentiate tissue Doppler velocities from conventional mitral inflow. Pulsed-wave TDI has high temporal resolution but does not permit simultaneous analysis of multiple myocardial segments.

With color TDI, a color-coded representation of myocardial velocities is superimposed on gray-scale 2-dimensional or M-mode images to indicate the direction and velocity of myocardial motion. Color TDI mode has the advantage of increased spatial resolution and the ability to evaluate multiple structures and segments in a single view.

Clinical Applications of TDI

Assessment of LV Systolic Function

Systolic myocardial velocity (Sa) at the lateral mitral annulus is a measure of longitudinal systolic function and is correlated with measurements of LV ejection fraction[2] and peak *dP/dt*.[3] A reduction in Sa velocity can be detected within 15 seconds of the onset of ischemia,[4] and regional reductions in Sa are correlated with regional wall-motion abnormalities. Incorporation of TDI measures of systolic function in exercise testing to assess for ischemia, viability, and contractile reserve has been suggested[5] because peak Sa velocity normally increases with dobutamine infusion and exercise[6] and de-

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The online-only Data Supplement, which contains a movie, is available at http://circ.ahajournals.org/cgi/content/full/113/10/e396/DC1.

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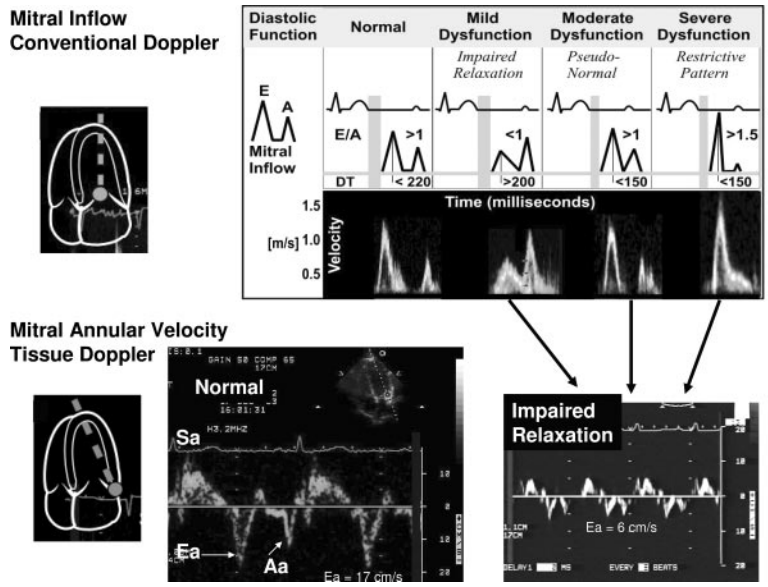

The upper panel illustrates conventional Doppler interrogation of mitral inflow. The normal mitral valve inflow pattern is characterized by E>A and an E-wave deceleration time of 150 to 220 ms. Impaired LV relaxation or decreased LV compliance is associated with a reversal of E and A waves and prolongation of E-wave deceleration to >220 ms. Pseudonormalization of the E-A ratio can occur when increased left atrial pressure results in an increased driving pressure and a consequent increased E-wave velocity across the mitral valve into a noncompliant LV. With severe diastolic dysfunction, the mitral valve inflow pattern can become restrictive, reflecting rapid equilibration of elevated left atrial and LV diastolic pressures in the noncompliant LV. The lower panel illustrates the 3 basic waveforms of tissue Doppler interrogation: Sa (systolic myocardial motion), Ea (early diastolic motion), and Aa (atrial contraction). Other abbreviations are as defined in text.

creases with ischemia.[7] The technical difficulties of timely acquisition of both 2-dimensional and TDI data during exercise represent the major limitations to routine integration in stress testing.

Assessment of Diastolic Function

Traditional echocardiographic assessment of LV diastolic function relied on Doppler patterns of mitral inflow. Reflecting the pressure gradient between the left atrium and LV, transmitral velocities are directly related to left atrial pressure (preload) and independently and inversely related to ventricular relaxation. Because mitral inflow patterns are highly sensitive to preload and can change dramatically as diastolic dysfunction progresses, the use of mitral valve inflow patterns to assess diastolic function remains limited.

TDI assessment of diastolic function is less load dependent than that provided by standard Doppler techniques. Ea reflects the velocity of early myocardial relaxation as the mitral annulus ascends during early rapid LV filling. Peak Ea velocity can be measured from any aspect of the mitral annulus from the apical views, with the lateral annulus most commonly used. Because of intrinsic differences in myocardial fiber orientation, septal Ea velocities are slightly lower than lateral Ea velocities.

Validated against invasive hemodynamic measures, TDI can be correlated with τ, the time constant of isovolumic relaxation.[8] Lateral Ea velocities can be 20 cm/s or higher in children and healthy young adults, but these values decline with age. In adults >30 years old, a lateral Ea velocity >12 cm/s is associated with normal LV diastolic function.[9] Reductions in lateral Ea velocity to ≤8 cm/s in middle-aged to older adults indicate impaired LV relaxation and can assist in differentiating a normal from a pseudonormal mitral inflow pattern. Unlike conventional mitral inflow patterns, Ea is resistant to changes in filling pressure, although preload dependence is more pronounced in structurally normal hearts.

Novel Applications of TDI

A number of emerging applications for TDI are under active investigation.

Estimation of LV Filling Pressures

Simultaneous cardiac catheterization and echocardiographic studies have shown that LV filling pressures are correlated with the ratio of the mitral inflow E wave to the tissue Doppler Ea wave (E/Ea).[10,11] This relation is based on Ea velocities that "correct" E-wave velocities for the impact of relaxation. The E/Ea ratio can be used to estimate LV filling pressures as follows: E/lateral Ea>10 or E/septal Ea>15 is correlated with an elevated LV end-diastolic pressure, and E/Ea<8 is correlated with a normal LV end-diastolic pressure.

Differentiation Between Constrictive and Restrictive Physiology

Both constrictive pericarditis and restrictive cardiomyopathy are associated with abnormal LV filling. With constrictive physiology, pericardial constraint impedes normal filling. In the absence of myocardial disease, Ea velocities typically remain normal. In contrast, the intrinsic myocardial abnormalities characteristic of restrictive cardiomyopathy result in impaired relaxation and reduced Ea velocities.

Early Diagnosis of Genetic Disease

Although unexplained LV hypertrophy is typically required to diagnose hypertrophic cardiomyopathy (HCM), the degree of hypertrophy and age of onset are highly variable. Abnormalities of diastolic function, as reflected by a reduction of Ea velocities, are present in individuals who have inherited a sarcomere gene mutation before the development of LV hypertrophy.[12,13]

Reduced Ea velocities have been similarly demonstrated in patients in the early stages of Fabry disease.[14]

Differentiation of Athlete's Heart From HCM

Approximately 2% of elite athletes may have an abnormal degree of LV hypertrophy.[15] Discriminating physiological hypertrophy due to intense athletic conditioning from pathological hypertrophy can be challenging. Athletes typically have highly compliant ventricles with brisk Ea velocities, in contrast to the reduced Ea velocities in individuals with HCM.[16]

Assessment of Cardiac Dyssynchrony

Identifying patients who will benefit from cardiac resynchronization therapy, which can improve heart failure morbidity and mortality rates, has been challenging. TDI can be used to assess the relative timing of peak systolic contraction in multiple myocardial regions.[17] The standard deviation of the time to peak contraction represents a measure of overall ventricular synchrony and may help identify potential responders to cardiac resynchronization therapy.

Assessment of Right Ventricular Function

The complexity of right ventricular anatomy and geometry challenges accurate assessment of right ventricular systolic function, an important prognostic indicator in patients with heart failure and in postinfarction patients.[18] Reduced tricuspid annular velocities with TDI have been documented in a variety of disease settings, including postinferior myocardial infarction, chronic pulmonary hypertension, and chronic heart failure.[19]

Disclosures

None.

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