EDITORIAL COMMENT

What Are We Actually Measuring by Doppler Tissue Imaging?*

Karl Isaaz, MD, FESC, FACC
Saint Etienne, France

In current Doppler blood flow studies, the low Doppler shift frequencies of high energy generated by the wall motion are purposely filtered out. Since these low Doppler shift frequencies are produced by the contracting myocardium, they are expected to contain potential information for the assessment of myocardial properties. Early descriptions of directional pulsed Doppler tissue imaging (DTI) had already suggested the potential of this original technique for evaluating both systolic and diastolic myocardial function (1–6). However, pulsed DTI suffers from the following limitations: 1) the need for manually performed mapping; 2) a limited spatial resolution with the impossibility of differentiating subendocardial from subepicardial myocardial velocities; 3) the impossibility of simultaneous recording of different ventricular wall segments; and 4) although pulsed Doppler signals can be obtained with all available commercialized ultrasound machines simply by using a high-frequency transducer, a low wall filter and low gain setting (1–6), important differences exist between the various equipment regarding the quality of the obtained signals.

Color DTI of the myocardium allows us to superimpose wall motion velocity on the two-dimensional echocardiographic imaging by velocity color coding or to track serial changes in ventricular wall motion velocity over time with the use of M-mode color DTI. Two-dimensional color DTI of the myocardium 1) allows a rapid visual qualitative assessment of the dynamics of the wall, 2) provides a good spatial resolution that permits differentiation of the velocity profiles between subendocardial and subepicardial layers, and 3) allows us to analyze simultaneously various myocardium regions, but it is limited by its poor temporal resolution. M-mode color-coded tissue imaging is characterized by a high spatio-temporal resolution, but sampling is performed only on a single line. Both two-dimensional and M-mode color DTI require specific modification of the current ultrasound machines.

Advantages of DTI over conventional ultrasound techniques. As Doppler signals generated by walls motion are of high energy, they are little affected by tissues interfaces between the site of interest and the transducer, so that it remains possible to examine subjects with pulsed DTI despite poor two-dimensional echocardiography imaging windows. The dynamics of any structure in motion are better described by the following three variables: velocity, acceleration and displacement (6), which remain difficult to be measured by conventional M-mode and two-dimensional echocardiography imaging techniques but are more easily quantified by pulsed DTI (6). The high temporal resolution of pulsed DTI allows us also to quantitate systolic and diastolic hemodynamic events during isovolumic periods (5,6). More recently, other investigators have used with success M-mode color DTI for analyzing these brief periods (10,11). Myocardial motion middiastolic small velocity waves can also be recorded by pulsed DTI, and their analysis might provide new insight into diastolic mechanical properties of the myocardium (6). Finally, DTI allows us to sample regional and segmental myocardial areas and to perform quantitative analysis of the intramyocardial velocity profiles across the walls.

Assessment of ventricular function along the meridional direction. The overall function of the left ventricle (LV) depends on a normal contraction of the longitudinally and circumferentially orientated myocardial fibers (12). Quantitative of the LV function in the longitudinal axis may be clinically relevant since the contraction in this direction is mainly due to subendocardial fibers (12). In particular, one can expect that in case of ischemia which specifically alters subendocardial layers, first abnormalities of wall motion will appear in the longitudinal axis. As the apex of the heart remains remarkably stationary (12), long axis changes are reflected in movements of the base of the heart. The first detailed data on the analysis of longitudinal dynamics of the heart with the use of pulsed DTI by locating the sample volume at the lateral margin and at the common septal margin of the tricuspid and mitral anuli were reported 11 years ago (5,6). By analyzing the relationship between the motion of the base and mitral flow, it has been shown: 1) that peak early diastolic velocity of base wave precedes slightly the peak velocity of early diastolic transmitral wave, which supports the concept of myocardial elastic recoil during early diastole and suction effect, and 2) that Doppler...
What are we actually measuring by DTI? From a fundamental standpoint, systolic pump function in patients with heart failure is characterized by a reduction in the effect of beta-adrenergic receptor stimulation on contractility related to a diminished beta-adrenergic receptor density (14,15). As it leads to production of cyclic adenosine monophosphate, which plays a role in the regulation of myocardial relaxation, beta-adrenergic-receptor stimulation also hastens relaxation through beta-adrenergic receptors (16–19). Doppler tissue imaging is now recognized as a validated technique for assessing both systolic and diastolic myocardial function. In particular, it is of importance to evaluate relaxation, in a clinical setting, since it plays a major role in the pathophysiology of many heart diseases and is often more severely impaired than contraction in the ischemic heart. Early work had already reported, in ischemic heart disease and aortic stenosis, abnormal DTI diastolic wave patterns of myocardial areas despite preserved systolic function characterized by a decreased DTI early diastolic velocity and an increase in the DTI late-to-early diastolic waves velocity ratio (3,4).

Recently, Oki et al. (20) have reported a close relationship between pulsed DTI wave early diastolic velocity and the rate of LV relaxation expressed by the invasively calculated Tau constant.

However, until now, the link between the fundamental concepts of contraction-relaxation and the clinical observations on DTI had been lacking. The study published in this issue of the Journal by Shan et al. (21) is unique and original since it demonstrates for the first time that systolic (Sm) and early diastolic (Em) velocities measured by DTI are directly dependent on myocardial structure characterized both by the percent of interstitial fibrosis and the myocardial beat-adrenergic receptor density. The article by Shan et al. (21) provides, therefore, new insight into our understanding of the assessment of both systolic and, diastolic function by pulsed DTI. However, some points are still unclear. It remains difficult, in the study of Shan et al. (21), to differentiate an independent effect of myocardial structure on Em from an interrelationship between Sm and Em. First, we have to know whether any significant relation between Sm and Em can be demonstrated using regression analysis both at rest and with low-dose dobutamine. This would allow us to assess the concept of stored elastic energy in the muscle during contraction which is released during relaxation resulting in an elastic recoil of the ventricle. Second, if any significant relationship exists between absolute systolic and diastolic velocities, is this relation different between normal and dysfunctional segments? It would have been interesting to compare, in the article by Shan et al. (21), normal and dysfunctional segments regarding the relation between not only absolute values but also between relative changes in Sm and Em in response to various dobutamine infusion rates. Indeed, in their study, the authors (21) do not mention whether dobutamine-induced relative or absolute changes in both Sm and Em are greater in normal segments than in dysfunctional segments. Parker et al. (18) have demonstrated that the magnitude of relaxation acceleration induced by beta-adrenergic receptor stimulation is similar both in patients with congestive heart failure and in normal subjects, whereas substantial attenuation in the beta-adrenergic positive inotropic response is found in those patients with heart failure.

The method of the study by Shan et al. (21) does not allow to differentiation of beta1- and beta2-adrenergic receptors. It has been shown that affinity and agonist potency of dobutamine are similar at both beta1, and beta2-adrenergic receptors (19). So it is not possible to determine, from the data of the authors, whether DTI velocities are most related to beta1- or to beta2-adrenergic receptors. This might be important to know as the distribution of beta1/beta2 myocardial receptors may vary depending on the severity of cardiac dysfunction due to downregulation of beta1-adrenergic receptors.

In addition to baseline beta-adrenergic receptors density and interstitial fibrosis, other important parameters like ischemia or hypoxia may alter systolic and diastolic myocardial function. The level of baseline myocardial ischemia or hypoxia in each of the 20 abnormal myocardial segments has not been quantitated by Shan et al. (21) in their study. Derumeaux et al. (22) have shown in an experimental study that changes in systolic and diastolic pulsed DTI velocities can be detected within 5 s of coronary occlusion. These authors have also demonstrated that the amplitude of DTI systolic velocity decrease depends on the severity of ischemia during coronary stenosis/or occlusion with a significant relationship between regional myocardial blood flow and velocities (22).

Conclusions. The important study by Shan et al. (21) represents the first step toward a better understanding of what we are actually measuring with DTI based on fundamental concepts rather than on clinical observations. To progress, further studies are needed in which invasively derived dimension-pressure data of myocardium regional normal and abnormal segments are recorded with both DTI and more detailed structural and functional analysis of biopsy specimens. As transfer under digital format to a microcomputer of two-dimensional and M-mode color-coded DTI allows us to perform quantitative analysis of the intramyocardial velocity profiles across the walls, this latter technique could be used in the future to study, in more detail, the relationship between DTI and intramyocardial distribution of adrenergic receptors and interstitial fibrosis.
Reprint requests and correspondence: Pr. Karl Isaaz, Service de Cardiologie, Hôpital Nord, Centre Hospitalo-Universitaire de Saint Etienne, Saint Etienne 42055 CEDEX 2, France. E-mail: isaaz@univ-st-etienne.fr.

REFERENCES


