Noninvasive Assessment of Myocardial Composition and Function in the Hypertrophied Heart

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Myocardial hypertrophy is both a useful physiologic adaptive process and an abnormal response to a variety of stimuli. Hypertrophy as an adaptive response to excessive loading conditions is an important compensatory mechanism that tends to minimize abnormalities in myocardial stress related to the inciting load. However, recent studies have indicated that hypertrophied muscle differs from normal muscle in many respects, including its structure, mechanical properties, vascularity, biochemistry, and electrophysiology.

Current noninvasive imaging methods permit the clinical differentiation of several etiologies of left ventricular hypertrophy, including pressure or volume overload, infiltration, and hypertrophic cardiomyopathy. This differentiation is usually accurately accomplished with echocardiography or other tomographic imaging methods, such as computed tomography or nuclear magnetic resonance imaging. Unfortunately, some diagnostic ambiguities occur that may not be easily resolved with current diagnostic methods. For example, amyloid cardiomyopathy presents the picture of concentric left (and right) ventricular hypertrophy. Although many patients with amyloid heart disease display an unusual, "speckled" myocardial appearance on standard echocardiograms, this phenomenon is not always noted, and the amyloid-infiltrated left ventricle may thus be confused with pressure-overload hypertrophy. Hypertrophic cardiomyopathy may resemble infiltrative cardiomyopathy on echocardiograms; conversely, amyloid cardiomyopathy may be mis-

taken for hypertrophic cardiomyopathy. Finally, the elderly patient with left ventricular hypertrophy related to hypertension may present an echocardiographic appearance difficult to distinguish from concentric hypertrophic cardiomyopathy. These diagnostic problems emphasize the continuing need for more specific methods of differentiating among the several etiologies of left ventricular hypertrophy.

One potential method of defining the structural basis of cardiac hypertrophy is that of ultrasound tissue characterization. Echocardiographers have frequently noted anomalies of regional tissue appearance on the ultrasound image in several disorders associated with hypertrophy. Unusual patterns of echo reflection can be seen in regions of myocardium with abnormal myofibrillar architecture such as hypertrophic cardiomyopathy or with infiltration of abnormal material such as amyloid. Although occasionally useful clinically, visual assessment of standard echocardiographic images is prone to the variability associated with all subjective observations. Further, the several image-processing manipulations performed during the recording of an echocardiogram (altering gain, time-gain compensation, and gray scale mapping functions) may have dramatic and confounding effects on image appearance. Thus, attempts have been made to develop objective methods of evaluating myocardial acoustic properties toward the goal of quantitative ultrasound tissue characterization. Substantial previous investigation has established that the amount of ultrasound energy returning to the echocardiographic transducer from the myocardium (referred to as ultrasound "backscatter") is reproducibly altered by acute and chronic ischemic injury as well as by experimental cardiomyopathies. Similarly, as alluded to above, an abnormal two-dimensional spatial pattern or "texture" of echo reflections from the myocardium has been associated with particular histopathologic abnormalities. Computerized methods of analyzing echocardiographic tissue texture have recently been shown capable of discriminating among hypertensive left ventricular hypertrophy, amyloid cardiomyopathy, hypertrophic cardiomyopathy, and normal myocardium.

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fact that these latter observations were made by analyzing standard, clinical echocardiograms recorded on videotape suggested that sufficient information may be contained in the ultrasound reflections from hypertrophied myocardium to begin to distinguish particular etiologies of hypertrophy in the clinical setting. Unfortunately, methods reported up to this time have had significant drawbacks, particularly the need for complex, off-line assessment of echocardiographic amplitude data with an additional computing system and frequently requiring substantial analysis time.

In this issue of Circulation, Masuyama and coworkers present intriguing evidence to support the notion that a novel echocardiographic imaging system yielding real-time images and measurements of integrated ultrasound backscatter may help to characterize pressure-overload hypertrophy and hypertrophic cardiomyopathy. In reviewing these interesting and provocative observations, I will briefly consider the mechanisms potentially responsible for the findings, the strengths and limitations of the particular method reported, and the place of these methods in the overall investigative field of cardiac tissue characterization.

Masuyama and coworkers evaluated the acoustic properties of normal and hypertrophic myocardium by measuring relative ultrasound backscatter throughout the heart cycle. These investigators noted that the amplitude of cardiac cycle-dependent (cyclic) backscatter variation was significantly smaller in the ventricular septum of both pressure-overload hypertrophy and hypertrophic cardiomyopathy than in the septum of normal subjects. The authors attribute these decrements in cyclic backscatter variation to two mechanisms: abnormalities of myocardial contractile function and myocardial fibrosis. In fact, previous observations have suggested that myocardial contractile function is an important determinant of the amplitude of cyclic backscatter variation. However, Masuyama et al observed a nonlinear relation between wall thickening and the amplitude of cyclic backscatter variation, a phenomenon previously noted by others. These data suggest that factors other than contractile function may be contributing to alterations in cyclic backscatter variation. A decrement in septal thickening in hypertrophic cardiomyopathy is a well-described phenomenon that may serve as one mechanism of decreased cyclic backscatter variation. However, wall thickening of the septum in pressure-overload hypertrophy should be normal or nearly so; thus, an additional mechanism must be implicated to explain a decrease in cyclic backscatter variation in pressure-overload hypertrophy.

Myocardial fibrosis is suggested by Masuyama et al to explain acoustic abnormalities in the septum of pressure-overloaded hearts. Previous observations concerning the deposition of collagen in pressure-overload hypertrophy have varied. Some authors have found an increase in regional fibrosis, assessed histologically or biochemically, while others have found an increase in total myocardial collagen but not an increase in the concentration of collagen in hypertensive left ventricular hypertrophy. Further, the degree of fibrosis may differ greatly between hypertensive hypertrophy and hypertrophic cardiomyopathy. Because histologic data concerning the presence and degree of fibrosis in the patients studied by Masuyama et al were not available, the contribution of collagen deposition to alterations in ultrasound backscatter data remains conjectural. Further, fibrosis in pressure-overload left ventricular hypertrophy would not be expected to vary greatly in different regions of the myocardium, whereas there was a substantial difference in the degree of cyclic backscatter variation in ventricular septum versus posterior wall in the present study. Further research is indicated to evaluate mechanisms of backscatter variability and its alteration by the hypertrophic process.

The key strength of the system reported by Masuyama et al appears to be its online capability. Thus, data on regional backscatter variation are available for qualitative interpretation at the time of examination, and assessment of the quantitative characteristics of backscatter variation may be accomplished rapidly after the examination. Two important limitations of the technique, however, should be recognized. First, in the present study, the estimation of regional myocardial backscatter was performed with an M-mode echocardiographic method, thus providing a spatially limited sample of myocardium. Given the dramatic regional differences in backscatter variation noted by these authors, sampling-related variability of data must be considered an important problem. A second limitation of this method is the wide range of cyclic backscatter variation noted in ventricular septum in normals and, particularly, in the hypertrophic groups. This may be due in part to the complex fiber architecture of the septum and inherent transmural variations in contractile function. Similar wide variability of septal cyclic backscatter amplitude has been noted by others in normal myocardium and may make the interpretation of data in a given patient extremely difficult.

The observations made by Masuyama et al are timely in that they build on substantial previous investigative work suggesting that the measurement of ultrasound backscatter, either averaged through the heart cycle or sampled at many points during contraction and relaxation, is a robust although technically demanding method of identifying abnormalities in myocardial composition or physiologic state. This study is also of importance because of its validation in a clinical setting of the value of this method of backscatter estimation. Unfortunately, the finding of blunted cyclic backscatter variation must be considered somewhat nonspecific, being found in hypertrophic cardiomyopathy and pressure-overload hypertrophy in the present study as well as...
in acute and chronic myocardial infarction and dilated cardiomyopathy.30 Thus, the lack of normal cyclic variation may indicate that myocardium is abnormal but not the specific nature of the abnormality.

The interesting observations made by Masuyama et al give further impetus to consideration of the use of ultrasound tissue characterization methods both to distinguish normal from abnormal myocardium and to begin to dissect the specific nature of myocardial abnormalities. Further research aimed at determining the mechanisms of backscatter variation and at evaluating acoustic features of myocardium that may differentiate among the several etiologies of decreased cyclic backscatter variation will be important for further development of this promising area of cardiac diagnosis.

References


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