
REPLY

We agree with Dr. Davila that the pathogenesis and medical treatment of chronic Chagas’ disease is complex and warrants further research. In our study, which included only a small number of patients with Chagas’ disease, there was no significant difference in sympathetic activation between Chagas and non-Chagas etiologies of heart failure (Fig. 2A of reference 1). This finding is in agreement with some (2,3), but not all (4) prior investigations. Of note, uniquely in our study, efferent muscle sympathetic nerve activity was measured directly utilizing microneurography, a sensitive and reproducible technique, rather than indirectly with plasma norepinephrine levels. Studies of the sympathetic activation in Chagas’ disease, which include patients with and without overt systolic dysfunction, may be able to distinguish between sympathetic dysregulation attributable to T. cruzi infection and neural pathways/autonomic ganglia and that attributable to the cardiac dysfunction present late in Chagas’ disease.

Although many hypotheses have been advanced to explain the sympathetic activation in heart failure, including baroreceptor dysfunction, overactivity of the arterial and/or muscle chemoreceptors, or abnormalities in the “heart failure milieu,” the exact mechanisms have not been established. It remains unknown whether the same mechanisms underlie the sympathetic activation in Chagas’ heart disease. Finally, the findings in our study lend support to the concept that, regardless of the etiology of left ventricular dysfunction and heart failure, a chronic exercise regimen is sympatholytic and beneficial. Studies enrolling larger numbers of patients with Chagas’ heart disease are underway in our laboratory in the hope of answering this question definitively.

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Assessment of Diastolic Function Using Myocardial Velocity Gradient

I read with interest an editorial comment by Dr. Shah (1) regarding a study on myocardial velocity gradient (MVG) and left ventricular dysfunction (2). I agree with his comment that no noninvasive measure of “pure” diastolic function has yet been achieved.

In his comment, Dr. Shah stated that diastolic MVG measurement was first reported by Fleming et al. (3) as being somewhat insensitive to preload or atrial filling pressures. To my knowledge, however, in 1994, MVG was introduced merely for the evaluation of myocardial contractility. Fleming et al. compared the velocity gradient with wall-thickness change to validate the new methodology (3). The application of MVG to assess diastolic dysfunction had to wait until the report by Palka et al. (4) differentiating hypertrophic cardiomyopathy from athletes’ hearts, in which, however, they did not assess the effects of preload alterations. Relative independence of MVG from preload alterations as compared to transmitral flow patterns was first reported by Shimizu et al. (5) in 1998. In addition, I would like to emphasize that a robust aspect of the tissue Doppler technique is the potential to assess regional, rather than global, diastolic function noninvasively, which should be validated and clinically applied in the near future.

Although the editorial comment by Dr. Shah raises an important issue in the assessment of diastolic function and thus is very informative, I am afraid his statement on the application of MVG measurements for the assessment of diastolic function seems to be inaccurate.
REFERENCES


REPLY

I thank Dr. Masaaki Uematsu for his thoughts related to my editorial comment dealing with hypertrophic cardiomyopathy and diastolic dysfunction (1).

Fleming et al. (2) first described myocardial velocity gradients detected by Doppler imaging and demonstrated Doppler velocity gradient waveforms in systole as well as in diastole. They compared these waveforms to rate of change of wall thickness both in systole and diastole and reported cyclic consistency. They showed that over 99% of systolic and 89% of early diastolic peaks in rate of change of wall thickness occurred concurrently with a peak in velocity gradient. Thus, their observations included Doppler velocity gradients during systolic thickening and diastolic thinning of the left ventricular posterior wall. They did not introduce this method “merely for the evaluation of myocardial contractility” even though they speculated on this potential.

As Dr. Uematsu, a second author on the contribution by Shimizu et al. (3), points out, their study showed a relative independence of Doppler myocardial velocity gradients to passive leg raising, which altered transmitral flow velocity profile. However, they also reported that volume-reducing therapy in congestive heart failure resulted in similar directional changes between velocity gradient and transmitral flow. This latter observation clearly indicates influence of preload on both parameters.

Dr. Uematsu considers that a robust aspect of Doppler velocity gradient is in assessment of regional rather than global diastolic function. This optimism ignores a caution expressed by Fleming et al. (2) that Doppler velocity gradient peaks may be influenced by the angle of the ultrasound beam relative to the muscle fibers. An oblique angle of the beam would yield a lower velocity gradient. The ventricular wall is composed of both circular and oblique fibers. This could have a profound effect on the noninvasive measurements of myocardial velocity gradients. Thus, in addition to physiologic and loading conditions mentioned in the editorial comment (1), the myocardial structure in terms of fiber orientation may add yet another confounding factor. We will do well to await further studies in validation and clinical application of this intriguing new Doppler technique.

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