Journal of the American College of Cardiology
© 2007 by the American College of Cardiology Foundation
Published by Elsevier Inc.

Vol. 50, No. 10, 2007
ISSN 0735-1097/07/$32.00

CORRESPONDENCE

Letters to the Editor

Effect on Obstruction on Longitudinal Left Ventricular Shortening in Hypertrophic Cardiomyopathy

Barac et al. (1) performed an elegant echocardiographic study to investigate the previously described midsystolic drop (MSD) in left ventricular (LV) cavity pressure that is frequently observed in patients with clinically relevant obstructive hypertrophic cardiomyopathy. In their intriguing paper, the authors conclude that "the MSD in ejection velocities and flow are caused by premature termination of LV longitudinal segmental shortening." This conclusion is based on longitudinal pulsed-wave tissue Doppler [imaging] (PW-TDI) data, which showed premature termination of longitudinal shortening in patients with LV outflow gradients of >60 mm Hg and normalization of the PW-TDI profiles after medical abolition of the gradient.

We have made similar observations in the past and firmly agree with the authors' conclusions about the significant impact of dynamic LV outflow tract obstruction on mechanical LV function. However, the PW-TDI technique may not be the most sensitive clinical tool to document the effects of dynamic outflow tract obstruction on longitudinal septal shortening. In our experience with high-frame-rate color-coded TDI, we did not observe complete premature termination of LV longitudinal shortening as described by Barac et al. (1), but rather a distinct pattern with an early systolic velocity spike followed by a sudden midsystolic septal deceleration and a second, late systolic, velocity peak (2,3). This pattern identified patients with a clinically significant resting gradient <30 mm Hg with a 93% sensitivity and 91% specificity and was always abolished by successful transcoronary ablation of septal hypertrophy.

***Ole-A. Breithardt, MD**
Berit Stolle, MD
Horst Kuhn, MD, FESC

*Medizinische Klinik 2
Universitätsklinikum Erlangen
Ulmenweg 18
DE-91054 Erlangen
Germany
E-mail: olebreithardt@gmx.de

doi:10.1016/j.jacc.2007.04.083

REFERENCES

1. Barac I, Upadya S, Pilchik R, et al. Effect of obstruction on longitudinal left ventricular shortening in hypertrophic cardiomyopathy. J Am Coll Cardiol 2007;49:1203–11.
2. Breithardt OA, Stolle B, Franke A, Janssens U, Hanrath P, Kuhn H. Mid-systolic septal deceleration—a new sign of left ventricular outflow tract obstruction obtained by color-coded tissue Doppler echocardiography. Z Kardiol 2003;92:1003–7.
3. Breithardt OA, Beer G, Stolle B, et al. Mid-systolic septal deceleration in hypertrophic cardiomyopathy: clinical value and insights into the pathophysiology of outflow tract obstruction by tissue Doppler echocardiography. Heart 2005;91:379–80.

Reply

We appreciate the comments of Drs. Breithardt, Stolle, and Kuhn on our paper concerning premature termination of systolic left ventricular contraction in hypertrophic cardiomyopathy (HCM) with severe obstruction (1). Their results are in a similar group of patients, acquired by color-coded tissue Doppler imaging (TDI). Although the data on timing of events recorded by TDI are not available in their reports (2,3), we agree that they have described the same phenomenon: an abnormal drop in left ventricular longitudinal shortening velocities in obstructive HCM. This abnormality was not observed in nonobstructed patients. The fact that it was abolished by reduction of the gradient either with pharmacologic intervention (1) or with alcohol ablation (2,3) confirms that the drop is caused by the acute imposition of afterload.

Breithardt et al. (2,3) observed a second late systolic velocity peak of TDI velocities after the midsystolic drop. We observed premature termination of contraction. We offer 2 possible explanations for the differences between our findings:

1. Our group included only patients with very severe left ventricular outflow track gradients: All of the patients had gradients of <60 mm Hg, with a mean gradient of 132 mm Hg. Dr. Breithardt's obstructed group included patients with gradients <30 mm Hg, with a mean gradient of 71 mm Hg (2). We believe that more severe obstruction among our patients produced the more pronounced abnormality: premature termination of septal longitudinal shortening. We also found these abnormalities in the lateral wall, which was not reported by Breithardt et al.
2. We used spectral TDI, in contrast to Breithardt et al., who used color-coded TDI. The differences between techniques have been described (4) and may have contributed to the differences observed. Comparisons in the same patients of peak spectral versus mean color-coded TDI tracings would be of interest, particularly with respect to the timing of contraction abnormalities.

Ivan Barac, MD
***Mark V. Sherrid, MD**

*St. Luke's-Roosevelt Hospital Center
Columbia University College of Physicians and Surgeons
1000 10th Avenue, 3B-30
New York, New York 10019
E-mail: msherrid@chpnet.org

doi:10.1016/j.jacc.2007.05.027