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LETTERS TO THE EDITOR

Color M-Mode Doppler Flow Propagation Velocity in Cardiac Tamponade

We read with interest the article by Garcia et al. (1) describing color M-mode Doppler flow propagation velocity (Vp) as a preload insensitive index of left ventricular (LV) relaxation. When the conditions prevailing in Garcia's study are present (i.e., during cardiac surgery and following pericardiotomy), the evidence that reducing preload does not change Vp and, by inference, tau, is convincing. In contrast to these findings, we have noted a pronounced respiratory variation of Vp in the setting of cardiac tamponade. Figure 1 shows the color M-mode flow propagation into the LV before (A) and immediately after (B) pericardiocentesis in a case of pericardial effusion with cardiac tamponade. Prior to pericardiocentesis, the Vp slope varies with respiration, with values ranging from 70 cm/s at end-inspiration to 100 cm/s at end-expiration.

After pericardiocentesis, this variation disappears, and Vp is constant at 60 cm/s. The increased flow propagation prior to pericardiocentesis is likely due to the accelerated LV relaxation that has been demonstrated in cardiac tamponade (2). Assuming that Garcia's data are correct, we have to consider other mechanisms than variability of preload to account for the increased respiratory variation of Vp in tamponade. Transmural ventricular diastolic pressure does not change with respiration; pericardial and ventricular diastolic pressures change equally in response to

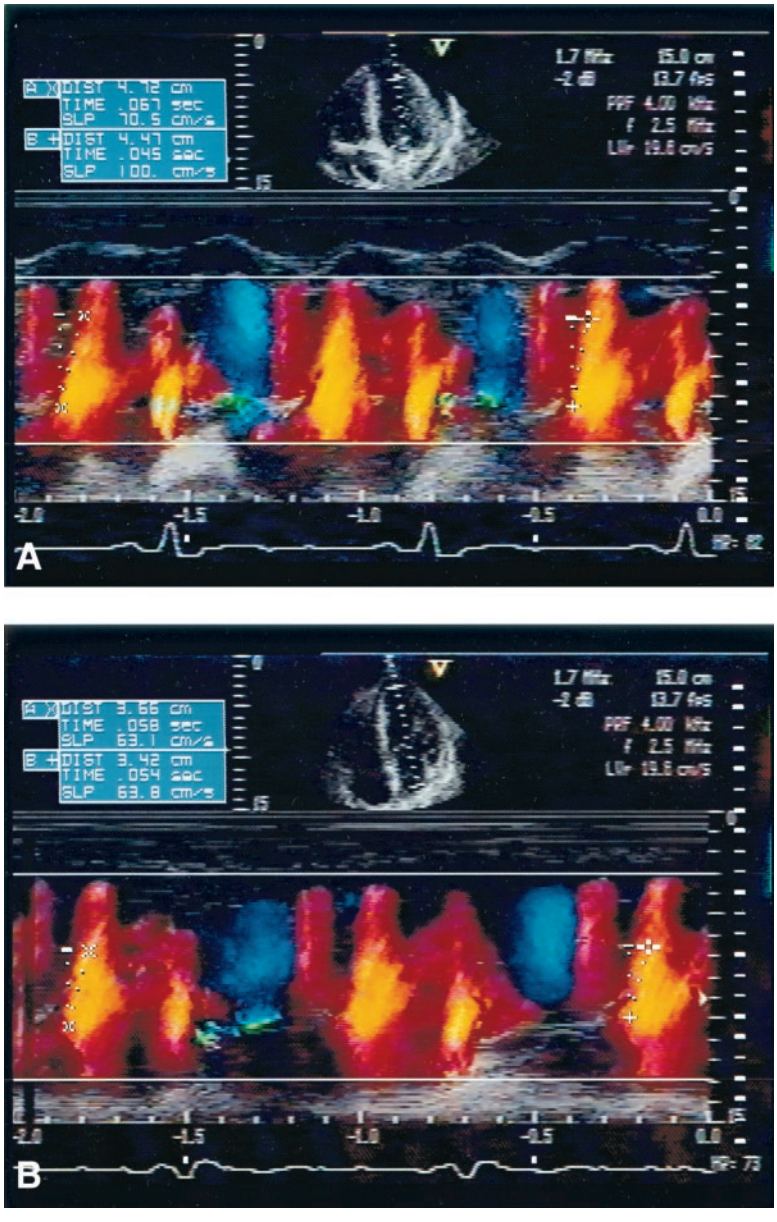

Figure 1. Color M-mode Doppler flow propagation into the LV before **(A)** and immediately after **(B)** pericardiocentesis in a patient with cardiac tamponade.

changes in intrathoracic pressure. Leftward shift of the ventricular septum during inspiration though may impair filling of the LV and, consequently, diminish Vp.

Finally, we would like to solicit the authors' comments, and we wonder whether a respiratory variation in Vp may be a marker for hemodynamic compromise due to pericardial effusion.

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REPLY

The recent implementation of new Doppler echocardiographic methods for the assessment of diastolic function has improved our understanding of this complex entity. Standard indices of transmitral flow are hampered by their dependency on loading conditions and left ventricular (LV) relaxation and have therefore been unable to differentiate a patient with normal (normal relaxation and preload) versus pseudonormal (impaired relaxation and increased preload) LV filling (1). More recently, the velocity of flow propagation into the LV (Vp) has been shown to provide an estimate of LV relaxation (2,3). Takatsuji et al. (4) studied a large group of patients with normal relaxation, delayed relaxation and pseudonormal pulsed Doppler patterns of LV filling confirmed by hemodynamic findings. While pulsed Doppler indices showed the typical "U-shaped" distribution from normal to delayed relaxation in pseudonormal patients, color M-mode Doppler Vp was equally low between the last two groups. Furthermore, their study also showed a strong negative correlation between τ and Vp, despite a wide variability in LV filling pressures among the three groups of patients, suggesting that Vp was less influenced by preload. In a study published in the January 2000 issue of the *Journal* (5), we demonstrated in controlled experimental settings that Vp was not affected by preload reductions in dogs undergoing caval occlusion and humans during partial bypass.

The letter of Togni et al., describing the changes in color M-mode flow propagation velocity (Vp observed in a patient with cardiac tamponade, is of significant interest. The authors demonstrate 1) significant respiratory variability of Vp during cardiac tamponade, increasing during inspiration and 2) a significant decrease in Vp after pericardiocentesis. A possible explanation for the respiratory variability observed may be periodic misalignment between the direction of flow and the M-mode cursor, changing the Doppler angle of incidence. This is likely to occur in the presence of a large pericardial effusion, when increased inspiratory venous return to the right heart can result in lateral translation of the LV. We agree with the authors, who conclude that the overall higher Vp during tamponade is likely due to a catecholamine-driven increase in LV relaxation. The fact that Vp decreased after pericardiocentesis, when venous return to the LV should increase, further supports that Vp is a preload-insensitive index.

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Apolipoprotein E Genotype and Coronary Heart Disease

We have read with interest the article by Frikke-Schmidt et al. (1), which concludes that male carriers of the Apo E epsilon43 and epsilon44 genotypes are particularly susceptible to ischemic heart disease.

We studied 220 men younger than 50 years of age (mean age 43 $\pm$ 5 years; range 26 to 50 years) and diagnosed with coronary artery disease (CAD). The polymorphisms of the apolipoprotein E (Apo E) were determined and compared to a control group of 200 healthy individuals matched with patients for age and ethnicity and residents in the same region (Asturias, northern Spain). We analyzed the principal cardiovascular risk factors, and during hospitalization and after fasting for 12 h a lipid profile study was carried out.

The Apo E genotype frequencies are summarized in Table 1. In our population, the Apo E gene and genotype frequencies were similar between patients and controls. Also, Apo E gene and genotype frequencies did not differ between patients with or without diabetes, or with or without hypertension. In addition, average biochemical values did not differ between the genotypes of each of the four polymorphisms.

Compared to other Caucasian populations, we found a lower frequency of the Apo Eϵ4 allele. These data are in agreement with