increase in end-diastolic pressure. Using simultaneous Doppler echocardiographic and apexcardiographic recordings, we have previously reported (3,4) that in patients with coronary and hypertensive heart disease, a decreased peak A flow velocity is most frequently associated with a significantly elevated apexcardiographic A wave height. According to our data, atrial systolic function, which is given by the "generation of pressure" and assessed by the relative height of the apexcardiographic A wave, is not decreased but significantly increased in such patients. Consequently, the decrease in flow through the mitral valve during atrial contraction in this clinical setting does not reflect a diminished power of atrial contraction; rather, it is the result of elevated ventricular filling pressures only. Thus, "true atrial function" can be evaluated only by an additional assessment of the power of "pressure generation" and the resulting "atrial kick" by the use of apexcardiographic recordings. These alternative fundamental pathophysiologic aspects, which have been extensively analyzed by many investigators using various techniques, were entirely neglected by Manning et al. By excluding these important data from the interpretation and discussion of their findings, the authors arrived at false conclusions about the nature and definition of atrial function itself.

A combined Doppler echocardiographic and apexcardiographic A wave index would probably help greatly in providing a clinically useful evaluation of "true atrial function." Such an index could be, for example, the ratio of the relative A wave to total height of apexcardiogram and the peak A flow velocity; the former provides information about the force of "pressure generation" and the latter about the "change in flow" during atrial contraction.

We hope that our previous work using both Doppler (flow) and apexcardiographic (pressure) A waves will stimulate the development of such combined indexes for accurately evaluating the "true atrial function and performance," which can only be assessed when both parts of the equation are given.

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References

1. Manning WJ, Silverman DI, Katz SE, Douglas PS. Atrial ejection force: a noninvasive assessment of atrial systolic function. J Am Coll Cardiol 1993;22:221–5.
2. Choong CY, Herrmann HC, Weyman AE, Fifer MA. Preload dependence of Doppler-derived indexes of left ventricular diastolic function in humans. J Am Coll Cardiol 1987;10:800–8.
3. Manolas J, Melanidis Y, Steriotis J. Comparison between Doppler and apexcardiographic indices of left ventricular diastolic function using simultaneous tracings in coronary artery disease [abstract]. J Mol Coll Cardiol 1991;23 Suppl V:77.
4. Manolas J, Melanidis Y, Steriotis J. Comparison of simultaneous Doppler echo and apexcardiogram in detecting left ventricular diastolic dysfunction in patients with systemic hypertension. Acta Cardiologica 1992;47:231–6.

Reply

In our report (1) we define atrial ejection force as "that force exerted by the left atrium in propelling blood into the left ventricle during atrial systole . . . and should not be misinterpreted as an assessment of 'total' atrial force." Total atrial force would be the vector sum of all forces acting within the atrium. Utilizing echocardiographic variables, atrial ejection force is proportional to peak A velocity squared.

We agree that peak A velocity is frequently elevated among patients with heart disease. Because peak A velocity may be increased among patients with coronary and hypertensive heart disease, one would then have expected atrial ejection force to be increased in the study group (compared with control subjects), yet it was significantly depressed after cardioversion and continued for at least 1 week after cardioversion. With each patient used as their own control, atrial ejection force significantly increased during the succeeding period of observation. To explain our findings on the basis of changes in filling pressure alone, one would have to hypothesize that left atrial filling pressure increased during the month after cardioversion. It is more likely that filling pressures declined (2). Thus, through the use of longitudinal data, we are comfortable in affirming the validity of atrial ejection force as an index of atrial systolic function. We are unaware of serial apexcardiographic data among patients undergoing cardioversion and cannot be certain how this variable would change. Because the height of the apexcardiographic A wave is more closely related to ventricular stiffness, end-diastolic pressure and the volume of atrial systolic flow, concordance between it and atrial ejection force may be limited.

We fully appreciate that "transmitral Doppler data alone do not fully reflect changes in ventricular compliance and . . . a less compliant ventricle might present greater resistance to transmitral inflow and result in a depressed peak A wave velocity" (1). Better models are indeed needed, but because of the complexity of left ventricular diastolic and left atrial systolic function, one must carefully identify which components of cardiac performance are being assessed.

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References

1. Manning WJ, Silverman DI, Katz SE, Douglas PS. Atrial ejection force: a noninvasive assessment of atrial systolic function. J Am Coll Cardiol 1993;22:221–5.
2. White CW, Kerber RE, Weiss HR, Marcus ML. The effects of atrial fibrillation on atrial pressure-volume and flow relationships. Circ Res 1982;51:205–15.

Heparin and Aspirin in Unstable Angina: Insufficient Sample Size May Lead to Erroneous Conclusions

In their article, Holdright et al. (1) address the interesting question whether, in patients with unstable angina, heparin combined with aspirin is more effective in preventing transient myocardial ischemia than aspirin alone. The authors attack the current standard of practice in the United States, which is to use both aspirin and heparin (2). The authors, therefore, have the burden of proof.

Holdright et al. conclude that "combined therapy with heparin and aspirin compared with aspirin alone makes no difference in the development of [transient myocardial ischemia]." Strikingly, their data shown in Table 2 (1) suggest just the opposite. The number of patients with at least one episode of transient myocardial ischemia was 25% less in the heparin plus aspirin group than in the aspirin alone group, 18% vs. 24% of patients, respectively. Even more strikingly, this pattern was consistent in every single variable presented by the authors. The total number of episodes in the heparin plus aspirin group was less by 35%, the median duration of episodes shorter by 16%, the total duration of