Editorial Comment

Improved Cardiac Performance Secondary to Dobutamine: The Role of Ventricular-Vascular Coupling*

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The present study. In this issue of the Journal, Binkley et al. (1) report the results of hemodynamic studies from 10 patients with idiopathic dilated cardiomyopathy before and after intravenous infusion of dobutamine. By using high fidelity catheters to determine pressure and flow simultaneously, the authors defined the hydraulic load facing the left ventricle in terms of the impedance spectrum of the systemic circulation. They found that in addition to increasing left ventricular contractility, dobutamine produced direct effects on the circulatory bed, including a decrease in characteristic impedance of the aorta, a decrease in arterial wave reflection and an increase in power transfer from the left ventricle to the aorta. Thus, in addition to enhancing left ventricular contractile performance, dobutamine appears to improve ventricular-vascular coupling in patients with heart failure.

The results of this study (1) are important for several reasons. First, they remind us that to consider the left ventricle in isolated terms is simplistic and that to neglect the behavior of the vasculature is to miss half the story. Although most clinicians know that dobutamine is a positive inotropic agent, some may not be aware that it has direct vasodilatory properties. These likely result from peripheral beta-agonist effects (2) and, as shown by Binkley et al. (1), play an important role in the clinical setting of congestive heart failure. The use of state-of-the-art hemodynamic analysis provides insights into the separate actions of dobutamine on both components of the cardiovascular system, some of which might otherwise go unnoticed.

Second, although the indexes used in this study to evaluate the vascular system are not intuitive, they are important enough to merit careful consideration by students of clinical physiology. The hydraulic load facing the left ventricle is more than resistance alone. To understand how the pulsatile output of the heart is converted to steady state flow in the capillary beds, it is necessary to understand the meaning of vascular impedance. To understand the nature of the aortic pulse wave contour, it is necessary to consider that pressure and flow waves are reflected and that these reflected waves add to or subtract from the instantaneous pressure in the aorta (3). These considerations are particularly important when examining age-related changes in arterial pressure (4) and in left ventricular mass (5), which may to a large extent be due to alterations in vascular dynamics.

Finally, it is worth emphasizing that 2 of the 10 patients studied by Binkley et al. (1) had very different responses to dobutamine. The inotropic index (maximal rate of rise in left ventricular pressure, dP/dt), in these two patients was increased, but characteristic impedance was also increased rather than decreased, and the augmentation of stroke volume as compared with that in the other eight patients was greatly attenuated. These differences have two implications. First, they underscore the importance of vascular response in modulating the effects of contractile stimulation. Second, they illustrate that each patient must be evaluated individually; the assumption that dobutamine will improve ventricular-vascular coupling in every patient may be wrong in 20% (8 of 10) of patients. If a diminutive increase in stroke volume occurs in patients with heart failure after dobutamine infusion, additional vasodilators may be needed to reduce afterload and to further improve left ventricular performance.

Evaluating the net cardiovascular effect of an inotropic intervention. Not all data support the hypothesis that improved ventricular-vascular coupling is a general response to increases in myocardial performance. First, the two patients in this study with a suboptimal increase in stroke volume had increased, not decreased characteristic impedance. Second, recent data from our laboratory (6) suggest that inotropic stimulation secondary to increased heart rate differs from that resulting from dobutamine. In that study, although the slope of the end-systolic pressure-volume relation was increased by tachycardia, the effective arterial elastance (7) was also increased, and thus, the stroke work from any given left ventricular end-diastolic volume was decreased. Dobutamine, as in the study by Binkley et al. (1), increased contractility, decreased effective arterial elastance, and substantially improved cardiac performance. Thus, not all forms of contractile stimulation are accompanied by improved ventricular-vascular coupling. Only by separating the response of the heart from that of the vasculature can the net effect be fully appreciated.
cardiovascular effect of a positive inotropic intervention be properly interpreted.

Conclusions. Further studies along the lines of investigation of this report (1) are necessary to fully define the influence of drugs on the heart and vasculature. This more complete definition will improve our understanding of heart failure, guide approaches to treatment and enable us to develop better forms of therapy for reducing symptoms in patients with congestive heart failure.

References

6. Freeman GL, Colston JT, O'Rourke RA. The role of ventriculo-vascular coupling in cardiac response to increased contractility (abstr). Circulation 1989;80 suppl II:11-667.