EDITORIALS

Does Increased Work of the Right Ventricle Diminish Left Ventricular Function?

The anatomic contiguity of the right and left ventricles suggests that alterations in the mechanical properties, configuration, or contractile performance of one chamber might influence the function of the other. By virtue of the anatomic arrangements of muscular fibers, right ventricular ejection is dependent on both longitudinal and circumferential shortening of its free wall against a convex interventricular septum, whereas left ventricular ejection relies on a fairly uniform contraction of all portions of the cylinder (Fig 1A).

The structural relationship between these chambers is such that marked changes in the size of the left ventricle will not ordinarily result in any fundamental changes in the bellows mechanism of right ventricular ejection. In contrast, the potential influence of increases in either right ventricular volume or in pressure-work on left ventricular systolic pump function, as well as diastolic compliance, is more apparent (Fig 1B). These anatomic considerations have led to experimental and clinical studies of left ventricular performance in the presence of an increased tension of the right ventricular wall.

Overfilling of the normal right ventricle in isolated cardiac preparations results in a greater elevation of left ventricular end-diastolic pressure for a given level of left ventricular filling. Kelly et al described similar changes in the diastolic pressure-volume curve of the left ventricle in dogs with chronic right ventricular pressure and volume overload; however, this abnormal left ventricular compliance was normalized when the hypertrophied right ventricle was filled only to a normal end-diastolic pressure, thus demonstrating that the changes in left ventricular compliance were not secondary to any intrinsic alterations in ventricular distensibility.

Kelly et al also demonstrated a modest decrease in both peak velocity of the contractile elements and peak change in pressure over time (dP/dt) of the left ventricle in these animals with chronic right ventricular failure. Whether this systolic impairment was related to altered geometry or to biochemical changes, such as marked biventricular depletion of levels of catecholamines, was not resolved. That short-term changes in configuration have little effect on left ventricular function is supported by a study in dogs in which the main pulmonary artery was partially obstructed with a balloon. The ensuing right ventricular dilation caused a decrease in the septal-lateral wall diameter of the left ventricle, yet the left ventricular ejection fraction (LVEF) remained normal. Further studies would be required to resolve these apparent differences regard-
ing the effects of short-term vs long-term right ventricular failure on the left ventricle.

Congenital cardiac lesions provide a convenient clinical setting in which we can examine whether left ventricular performance is diminished in the face of gross increases in right ventricular volume or pressure-work, or both. Pulmonic stenosis with an intact interventricular septum exemplifies increased right ventricular pressure-work, and in the absence of right heart failure and right-to-left shunting at the atrial level, the LVEF is normal. The LVEF is also normal with an increased volume load of the right ventricle, as occurs in an atrial septal defect. Here the left ventricular end-diastolic volume is modestly reduced, yet the left ventricular end-diastolic pressure is not diminished, which suggests that a volume load of the right ventricle does alter left ventricular compliance. In transposition of the great vessels, the LVEF remains normal despite the arterial desaturation and some decrease in right ventricular ejection. Although in these subjects, hypoxemia does not appear to compromise left ventricular performance, a very mild reduction in LVEF can occur in patients with tetralogy of Fallot. This reduced LVEF could also derive from the decreased left ventricular volume which is secondary to diminished pulmonary blood flow. It is apparent from these clinical studies that in the absence of right heart failure, the LVEF remains virtually normal in the presence of severe pressure or volume loads (or both) of the right ventricle.

In view of these experimental and clinical data, one would not anticipate that abnormalities in the pressure-work and ejection fraction of the right ventricle, which occur in chronic obstructive pulmonary disease, would result in a significant abnormality of left ventricular performance. Indeed, in this issue (see page 556), Kline and associates have presented additional data indicating that indices of left ventricular ejection, as measured by echocardiographic and radionuclide angiographic techniques, are generally normal in such patients. The study of Kline et al assumes added significance in that the patients were selected on the basis of suspected left ventricular failure. Comparable data, based on a similar radionuclide technique, were obtained by Steele and his co-workers in 92 patients with acute respiratory failure and in 28 patients with stable chronic pulmonary disease. The LVEF was normal in the majority of those subjects in whom there was no clinical or subsequent evidence at autopsy of coronary arterial disease. These studies, utilizing noninvasive methods, corroborate earlier inquiries into left ventricular function in patients with obstructive pulmonary disease.

Increases in right ventricular work do not significantly diminish left ventricular performance in patients with either congenital cardiac lesions or chronic obstructive pulmonary disease. When left ventricular dysfunction is demonstrable in such patients, one should search for another disease process that directly affects this chamber.

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