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Title : EFFECTS OF AFTERLOAD ON VENTRICULAR PERFORMANCE

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INTRODUCTION. Previous studies in man have indicated that in the normal heart, increases in afterload are well tolerated; whereas, in the diseased heart, stroke volume and stroke work tend to decrease.[1] Recently, however, several investigators have challenged this concept for patients undergoing anesthesia and surgery.[2] These authors reported that left ventricles with good function are sensitive to increases in afterload during anesthesia and surgery. In contrast, ventricles with poor function were significantly less sensitive to changes in afterload. Because of the clinical implications of these results, we studied the relationship between ventricular performance and afterload over periods of acute hemodynamic change and over a 24-hr perioperative period.

METHODS. We received informed consent and approval from the Committee on Human Research to perform two studies (one chronic and one acute) on 37 patients admitted for coronary artery surgery. Preoperative ejection fractions ranged from 0.26 to 0.82. Anesthesia consisted of morphine sulfate (1.5 to 3 mg/kg iv) and diazepam (0.25 to 0.50 mg/kg iv). Ventilation (with 100% oxygen) was controlled. Chronic Study: In 22 patients, hemodynamic variables were measured 1) prior to cardiopulmonary bypass; 2) 1/4 and 1 hour after bypass in the operating room; and 3) at 2, 4, 8, 18, and 24 hr after bypass in the intensive care unit. At each of these times, alterations in hemodynamics were induced by changing the patient's body position from horizontal to 30° Trendelenburg and then to 30° reverse-Trendelenburg. The following relationships were analyzed: systemic vascular resistance (SVR) vs. 1) cardiac index (CI), 2) stroke volume index (SVI), and 3) left-ventricular stroke work index (LVSWI); and pulmonary vascular resistance (PVR) vs. 1) CI, 2) SVI, and 3) right-ventricular stroke work index (RVSWI). Acute Study: In 15 patients, hemodynamic measurements were made over a 30-min period after bypass. Hemodynamics were acutely altered by volume loading with 500, 1000, 1250, and 1500 ml of whole blood. Hemodynamic variables, as well as left ventricular ejection fraction (EF) and end-diastolic and end-systolic volumes (EDV, ESV), were measured using radionuclide angiocardiography. Left-ventricular wall tension (T) was estimated using the radionuclide measurements. We analyzed the effect of increasing wall tension on EF, ESV, and other indices of ejection for each patient.

RESULTS. Chronic Study: In Figure 1, results for the 11 patients with good preoperative ventricular function (EF > 0.50, no dyssynergy) are compared with those for the 11 patients with poor function (EF < 0.40 or with dyssynergy). The inverse correlation between CI and SVR or PVR is significantly greater for the group with poor preoperative ventricular function. These results were true for the individual measurement periods as well. The correlation of SVI and SVR was stronger for the group having poor function (-0.70 to -0.91) than for the group having good function (-0.57 to -0.67), as was that for SVI and PVR (-0.46 to -0.67 vs. -0.18 to -0.36). Acute Study: In each of the 15 patients, as T increased, EF decreased (Fig. 2). Other indices of ejection (ESV, systolic blood pressure ÷ ESV) also demonstrated similar deterioration of function with increasing T. These results were true for all 15 patients, but were significantly more pronounced in the group with poor ventricular function.

DISCUSSION. The results of this study support the hypothesis of Ross and Braunwald as applied to the intraoperative setting: changes in afterload have a more marked effect on diseased ventricles than on normal ventricles. In patients with poor preoperative ventricular function, both ventricular ejection and output are significantly affected by acute (30 min) or chronic (24 hr) changes in afterload during the perioperative period. Thus, in contrast to other recent studies,[2] our results suggest that reduction in afterload may significantly improve the performance of diseased ventricles when either acute or chronic perioperative dysfunction occurs.

REFERENCES

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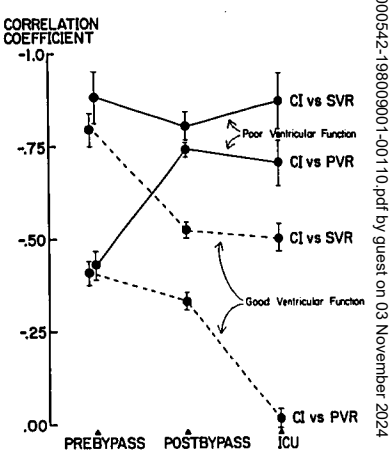

Fig. 1. Relationship between CI and vascular resistance.

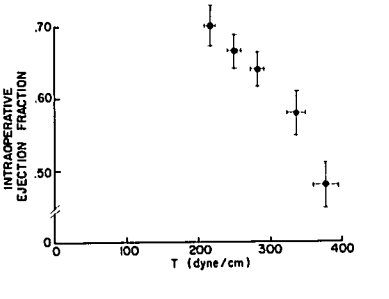

Fig. 2. Relationship between EF and wall tension.