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Inotropic Effect of Amrinone

To the Editor:—The article by Rooney *et al.*[1] is a worthwhile contribution to our understanding and appreciation both of the character and of the limitation of amrinone as an inotropic agent in surgery and anesthesia. Over a decade since becoming clinically available, amrinone, unlike dobutamine, has gained little popularity in surgical as well as other anesthetized patients who need urgent hemodynamic support. Without the indirect effect from afterload reduction, amrinone can be considered only a mild or weak inotropic agent, as shown by the isovolumetric peak left ventricular pressure increase of 12.8% after 500 μM. For patients without heart failure, the afterload reduction effect of amrinone may not be beneficial.

Recently, in a different preparation, we studied the direct effect of infusion of amrinone on isolated rabbit myocardial septum.[2] With concentrations ranging from 1 to 1,000 μg/ml, the results showed that at concentrations greater than 10 μg/ml, amrinone caused slight (5–11%) increases in peak developed tension and maximal acceleration (dT/dt). In contrast to Rooney *et al.*'s study, we did not observe a dose-dependent increase of contractility. Our conclusion is that amrinone is not a strong inotropic agent and cannot be treated the same as dobutamine or dopamine.

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In Reply:—We appreciate the interest of Lee and Virtusio regarding our work describing the isolated cardiac effects of amrinone.[1] Their particular concern is whether amrinone is really much of a positive inotropic agent. We agree that it has only a mild positive inotropic effect *in vitro*. Perhaps it does not produce much of a tachycardia because it is also not a potent direct positive chronotropic agent. We also observed that amrinone did not produce coronary vasodilation above the metabolic demand in the isolated heart. Its major effect *in vivo* probably is peripheral venodilation and arteriolar vasodilatation.[2] With administration of amrinone, the increase in cardiac output without a change in myocardial oxygen consumption in patients with congestive heart failure likely reflects a reduction in afterload and enddiastolic ventricular volume more than it does a direct positive inotropic effect.[3] A reduction in ventricular filling pressure by amrinone may decrease wall tension in patients with dilated ventricles and so counteract any increase in myocardial oxygen consumption due to a direct mild inotropic effect. Higher loading doses than originally recommended, however, may result in a greater inotropic effect.[4]

Differences in inotropy *in vivo* and *in vitro* may be due to species differences or to adrenergic stimulation. Amrinone may be more effective in the presence of catecholamines, and afterload reduction may stimulate an adrenergic response. Whether amrinone or traditional drugs such as digitalis, epinephrine, isoproterenol, dopamine, or dobutamine are better choices for inotropic support, alone or with peripheral vasodilators, after cardiopulmonary bypass has been debated recently.[4,*] The combined use of amrinone and other inotropic agents also has been discussed. It appears that milrinone and other newer phosphodiesterase III inhibitors† under development have greater positive inotropic effects that may approach the effects of the natural and synthetic catecholamines.

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