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Editorial Comment

Intraaortic Balloon Counterpulsation: Deciphering Its Effects on Coronary Flow*

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It has been nearly 30 years since Kantrowitz and colleagues (1) first described the use of intraaortic balloon counterpulsation for treatment of cardiogenic shock. Rapid inflation and deflation of a polyurethane sleeve positioned in the descending thoracic aorta, synchronized with the cardiac cycle, effectively augmented systemic arterial pressure and increased urinary output in two severely ill patients. Subsequent work has demonstrated that intraaortic balloon counterpulsation consistently increases peak aortic diastolic pressure and decreases peak left ventricular systolic pressure. Cardiac output increases ~15%, primarily as a result of reducing impedance to left ventricular ejection (2). More recently, intraaortic balloon counterpulsation has been used as an effective adjunct for patients undergoing open heart surgery (3) and coronary angioplasty (4). In each of these procedures, the benefit derived from intraaortic balloon counterpulsation is related to augmentation of a low cardiac output state resulting from depressed left ventricular function.

In 1973, Gold et al. (5) reported that intraaortic balloon counterpulsation could relieve ischemia in patients with unstable angina. Unlike prior uses of intraaortic balloon counterpulsation, benefit was achieved in the setting of normal ventricular function, that is, patients were not hypotensive or in congestive cardiac failure. Because intraaortic balloon counterpulsation increases peak aortic diastolic blood pressure, the intuitive explanation for the relief of rest angina was that the balloon pumping augmented coronary blood flow because anterograde left coronary flow occurs in diastole. However, investigations measuring coronary flow during intraaortic balloon counterpulsation have shown variable results. Several factors, including differences in the experimental model, manner and location of measuring coronary flow, effect of metabolic autoregulation and severity of coronary narrowing, have been identified as confounding factors that may explain these disparate findings.

In their report in this issue of the Journal, Kimura et al. (6) have attempted to set the story straight. These investigators went to great efforts to recognize and correct the potential deficiencies of prior investigations when designing their experimental model and protocol. The stenosis used was severe. Coronary flow was measured in both an epicardial, compliant artery as well as an intramural, distal artery that was subjected to external compressive forces (7). Both total and phasic coronary flow were measured with and without coronary stenosis. Two to one pumping was used to neutralize the effect of systemic unloading with consequential metabolic autoregulation.

These investigators found that in the absence of severe coronary narrowing, the direct (without the influence of ventricular unloading) effect of intraaortic balloon counterpulsation was to modestly increase coronary flow. However, the presence of severe stenosis negated this effect because intraaortic balloon counterpulsation failed to increase distal coronary pressure in diastole.

In their discussion, Kimura et al. acknowledge the important contribution of autoregulation on coronary flow. The reduction of left ventricular systolic pressure that results from intraaortic balloon counterpulsation decreases myocardial oxygen consumption, which in turn causes metabolically mediated vasoconstriction. The less the demand, the lower the flow.

The net result of intraaortic balloon counterpulsation on coronary flow results from the complex interplay of the extent to which the procedure transmits the increase in peak diastolic pressure across the stenosis, the magnitude of decrease in left ventricular systolic pressure and the severity of coronary narrowing. When narrowing is minimal, a minimal increase or no change in flow is observed because the mechanical increase in flow is offset by vasoconstriction arising from less demand. With severe coronary narrowing, the precise situation wherein an increase in flow would be most desirable, net flow declines or is unchanged. The augmented diastolic pressure wave is simply not transmitted across the stenosis, an observation confirmed in patients undergoing coronary angioplasty (8). If some degree of reserve exists and flow is not totally pressure dependent, then an actual decrease in flow may be observed with intraaortic balloon counterpulsation because its only influence is the unopposed decline in flow from reduced demand (9).

One potential clinical implication of these effects of intraaortic balloon counterpulsation relates to the technique of its use. Typically, emphasis is placed on adjusting the timing of balloon inflation and deflation to optimize peak aortic diastolic pressure. Although this is an important goal, equal attention should be directed to the magnitude of peak left ventricular systolic pressure reduction. In many instances, the extent to which this variable is affected will have the greatest influence on relieving myocardial ischemia.

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