fusion pressure than used with halothane. Drs. Priebe and Foex show clearly that, with a critical coronary stenosis, isoflurane-induced *hypotension* has detrimental effects on coronary flow.[5]

We strongly disagree with Dr. Becker that isoflurane is contraindicated in patients with ischemic heart disease. Allowing coronary perfusion pressure to drop using isoflurane seems unwise, since isoflurane-induced hypotension appears to be an integral factor in these models of redistribution of coronary flow. Appropriate use of isoflurane should avoid hypotension in patients with coronary artery disease. A balanced anesthetic technique using a narcotic base with supplemental low-dose isoflurane (less than 0.5%) can achieve this goal. Higher levels of isoflurane may be added to control blood pressure if the patient is hyperdynamic. In fact, under those circumstances, isoflurane may even be protective.[7–9]

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In Reply:—I quite agree with most of Dr. Bollen's sentiments and would offer only two observations:

First, we were also surprised to find that adenosine failed to decrease the inner:outer flow ratios in both normal and collateral-dependent myocardium under conditions where steal occurred between these zones. Perhaps this finding can be explained by adenosine's preferential dilation of subendocardial vessels when this compound is administered in sub-maximal doses.[1]

Second, Dr. Bollen comments that coronary pressure was lower during isoflurane than during halothane under conditions that produced steal. Indeed, this fall in coronary pressure resulted from arteriolar dilation by isoflurane, and is a critical component of the hemodynamics of steal.[2] Total coronary *flow*, not coronary pressure, was the independent variable in this experiment, which asked: "How do these agents affect the *distribution* of flow under identical conditions of blood pressure, heart rate, and total coronary flow?"

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