with difficulty. The frequency content of the artifact is similar to the frequency content of the pressure wave; thus, any filter one would apply must recognize, or be able to assess, the overlap of frequencies and their magnitude and phase. If this cannot be accomplished, then reducing the magnitude of the artifact is likely to reduce the magnitude of the pressure pulse. However, if a filter were developed which would selectively remove artifact caused by poor catheter fidelity, it could be applied in conjunction with our processing algorithm just as Dr. Bashein suggests.

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Isoflurane and the Coronary Circulation

To the Editor:—We read with interest the papers and accompanying editorial regarding isoflurane and the coronary circulation in the March issue of Anesthesiology.1–5

Dr. Sill’s paper concludes that isoflurane must be a dilator of coronary arteries.6 Yet, at 0.75% concentration, isoflurane did not appear to cause significantly different flow compared to control. It was only above 0.75% isoflurane that significant increases in coronary flow were seen. In a clinical situation, isoflurane is added to a base of narcotic in concentrations of 1/2 MAC or less. In such situations, based on Dr. Sill’s results, isoflurane should cause no significant coronary dilation.

The immediate increase in coronary flow upon adding isoflurane observed by Dr. Priebé is of interest.5 Was this caused by the addition of isoflurane? We would not expect the end-tidal isoflurane concentration within 30 s of exposure to be greater than 0.75% (the concentration below which Dr. Sill observed no significant increase in flow). In our own experiments with isolated pig coronary segments, the vascular relaxant effect of isoflurane or halothane on pre-constricted coronary segments was gradual (10–20 min), not sudden as observed by Dr. Priebé.6

Based on the observation that isoflurane and adenosine, but not halothane, decreased collateral to normal coronary flow ratios in the presence of decreased coronary flow, Dr. Buffington concluded that isoflurane induced direct coronary dilation which produced coronary steal.4 It does appear that isoflurane does decrease coronary vascular resistance to a greater extent than does halothane, probably in part by direct effect on coronary vessels. However, we caution against equating isoflurane with adenosine in regard to effect and mechanism of action on large and small coronary vessels.

There are major differences in Dr. Buffington’s study between isoflurane and adenosine in their effect on transmural distribution of myocardial blood flow. Adenosine and isoflurane caused equivalent decreases in coronary vascular resistance. Isoflurane decreased the inner:outer flow rates in both normal and collateral zones, while adenosine and halothane did not. Although, in other laboratories, adenosine has been shown to cause transmural redistribution, why not in this experiment? These differences make it difficult for us to equate the vascular smooth muscle effects of isoflurane with those of adenosine. It is possible that redistribution of transmural flow by isoflurane is caused by other myocardial or central effects of isoflurane, independent of direct effects on vascular smooth muscle.

Dr. Buffington suggests that a ceiling exists to coronary dilation caused by isoflurane. This is based on his observation that coronary vascular resistance in protocols 1 and 2 were similar, while end-tidal isoflurane concentration was 0.94% in protocol 1 and 1.46% in protocol 2. The data of Dr. Sill, which show no significant increase diameter at 0.75% isoflurane, but with progressive increases in coronary diameter at 1.5% and 2.25%, argue against this.

Dr. Buffington’s paper does, indeed, show a coronary steal occurring with isoflurane under very special controlled conditions where total coronary flow is reduced 16–20% below the control value and at much lower per-
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.

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.

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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.

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. 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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REFERENCES

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