Another Source of Artifact in the Pulmonary Artery Pressure Waveform

To the Editor:—Mitchell et al.\(^1\) appear to have developed an excellent technique to remove respiratory artifact from pulmonary artery (PA) pressure waveforms. They also state that their algorithm "has the potential to easily incorporate an accurate digital readout for . . . (PA diastolic pressure)." However, neither their paper nor the accompanying editorial\(^2\) mentions another source of artifact that can erroneously depress the automatically determined PA diastolic pressure. I refer to the spike of noise caused when the tricuspid valve leaflets strike the catheter at the onset of ventricular systole.\(^3\) Since this spike is of short duration, it will contribute negligibly to the automatically determined mean pressure, but if it happens to have a large negative component, its nadir will be interpreted as the "diastolic" pressure by any minimum-seeking algorithm.

The magnitude of the resulting error will depend upon the dynamic response of the catheter/tubing/transducer system employed, patient factors, and the position of the catheter within the heart.

There is a suggestion of this phenomenon in the author's figure 1, and a more extreme example is shown in the accompanying figure 1, where the nadir of the noise spike appears to lie almost 3 mmHg below the "true" diastolic pressure. Bruner\(^4\) has suggested that high-frequency filtering might be used to eliminate this problem. Furthermore, the superposition theorem\(^5\) implies that, if a linear, high-frequency filter were developed to remove the noise spike, it could be applied in conjunction with the method of Mitchell et al. to produce a waveform relatively free of both of these annoying artifacts.

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REFERENCES

(In reply:—Dr. Bashein is correct in his assertion that inadequate dynamic response (fidelity) of the central vascular catheter-tubing-transducer hydraulic system may produce artifacts; low diastolic pressure values. A catheter system with poor fidelity is also likely to distort the contour of the systolic portion of the pressure waveform and, possibly, artificially increase the algorithmically determined systolic pressure. We did not feel that this problem, although significant, was within the scope of our algorithm.\(^1\)

The application of high-frequency filtering to reduce or eliminate the significance of this problem is fraught

Fig. 1. A human pulmonary artery pressure waveform. Vertical scale is 1 mmHg per division and horizontal scale is 40 msec per division.
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 arterioles.5 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. Priefe 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 coronaries was gradual (10–20 min), not sudden as observed by Dr. Priefe.5

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-