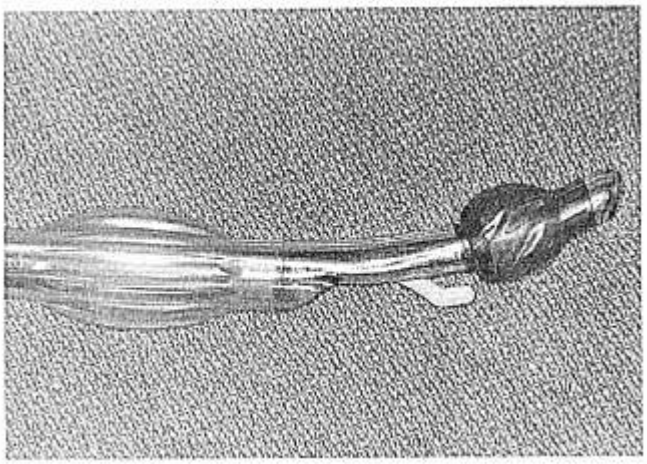

FIG. 1. Tip of a 37 Fr Broncho-Cath® with carinal hook.

cally be easier to treat; since the hook prevents excess caudal movement, if the tube becomes dislodged, it should merely be advanced until gentle resistance to movement is felt, indicating that the carinal hook has been reseated. I found these tubes a bit more difficult to pass than the Broncho-Cath®, but not excessively so. Of course, a bronchoscope can still be used to confirm proper tube position. However, if a bronchoscope is not available, I believe this tube may offer an increased margin of safety when blindly positioning (or repositioning) a left-sided double-lumen tube.

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In Reply:—Dr. Alfery suggests that the addition of a carinal hook to a left-sided double-lumen tube may protect against left upper lobe obstruction and may provide a solution to the problem of not having an appropriately sized fiberoptic bronchoscope for positioning double-lumen tubes. I have three difficulties with this line of reasoning. First, I believe that hospitals (however large) in which double lumen tubes are used should have a fiberoptic bronchoscope that fits down the lumens of double-lumen tubes. Second, the carinal hook is set approximately 8 mm proximal to the cephalad surface of the endobronchial balloon, thereby allowing an 8 mm deeper insertion into the left mainstem bronchus compared to having the cephalad surface of the left cuff just below the tracheal carina; this may increase the incidence of left upper lobe obstruction. Third, and as Dr. Alfery hinted, double-lumen tubes with carinal hooks are harder to insert. In summary, the best chance of not causing left upper lobe obstruction is to see the cephalad surface of the blue left cuff just below the tracheal carina with a fiberoptic bronchoscope.

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Is Coronary Vascular Reserve Really Not Affected by Volatile Anesthetics?

To the Editor:—Hickey *et al.*[1] have recently described coronary blood flow autoregulation and coronary vascular reserve in dogs. The advantage of the study is, as the authors point out, that chronic instrumentation allowed determination of physiologic pressure/flow relationship in the coronary vasculature in normal, awake animals, which was then compared with recordings obtained during halothane, enflurane, and isoflurane anesthesia. However, several questions and a few reservations are raised with respect to their conclusions. The authors measured coronary reserve as the absolute increase in left circumflex coronary artery (LCCA) blood flow during adenosine infusion at a diastolic LCCA pressure of 40 mmHg. Using their mean values for baseline and peak flow during maximum coronary vasodilation (their tables 2, 3), I have calculated the ratio

of peak flow to baseline flow (coronary reserve) to be 3.9 awake, 8.3 during halothane, 5.3 during enflurane, and 3.9 during isoflurane anesthesia. The awake and isoflurane values are substantially lower and the halothane value higher than described both in awake dogs and during halothane and isoflurane anesthesia in swine.[2,3] Cohen[2] found six- to seven-fold increases in peak circumflex coronary artery blood flow following adenosine injection in awake, chronically instrumented mongrel and greyhound dogs. We have found the ratio of peak hyperemic to baseline autoregulated LAD-blood flow velocity to be 5.5 ± 3.2 (SD) at 1 MAC isoflurane and 3.6 ± 1.5 (SD) during 1 MAC halothane anesthesia. Why did coronary reserve almost double from control during 1 MAC halothane, and why were control and isoflurane values so low? I would suggest several explanations. First, Hickey *et al.*[1] measured coronary reserve at a fixed LCCA diastolic pressure of 40 mmHg which they reached by adjusting LCCA-pressure with a hydraulic occluder placed distal to an electromagnetic flow probe on the LCCA. Since the "spontaneous" mean LCCA diastolic blood pressure was 83 ± 7 mmHg in awake dogs and 62–65 ± 7–11 mmHg during anesthesia according to their table 1, the LCCA-occluder must have been used to produce an average pressure gradient of between 43 mmHg (awake) and 22–26 mmHg (during anesthesia) to diminish diastolic LCCA pressure to 40 mmHg, since adenosine caused no change in systemic blood pressure or heart rate. If so, I suspect that maximum coronary (LCCA) blood flow was mechanically hampered by the occluder, and that adenosine-induced maximum LCCA blood flow was lower than the potential, unhindered maximum flow. This, in turn, would give a lower coronary reserve both awake and during anesthesia. In other words, how much was the occluder inflated in the different situations, and could this be a "functional" LCCA-stenosis?

Second, since heart rates were significantly lower during halothane anesthesia compared both to awake values and to values during equipotent isoflurane and enflurane anesthesia, coronary vascular reserve would be *expected* to be higher during halothane anesthesia compared to the other conditions. The most marked difference was between halothane and isoflurane, with mean heart rates during 1 MAC isoflurane 25% higher than during 1 MAC halothane. As the authors correctly point out in the Discussion, both increased contractility and increased heart rate have been shown to reduce coronary vascular reserve.[4] Thus, comparable baseline may not have been present to justify the conclusion that "coronary vascular reserve was not different in the awake and anesthetized dog."[1] The significant difference in heart rates may, in part, explain the significantly lower coronary blood flow during 1 MAC halothane. Actually, mean LCCA blood flow was 48% lower at 40 mmHg LCCA diastolic pressure and 49% lower at 60 mmHg diastolic pressure during halothane anesthesia compared to isoflurane in their study. Thus, there were significant differences affecting important determinants of residual capacity for coronary dilation both in awake (heart rates) and in enflurane- and isoflurane-exposed animals (heart rates and LCCA baseline blood flow). The authors[5] have previously shown that if tachycardia and diastolic pressure changes are avoided, the effects of halothane and isoflurane on coronary vascular conductance are small. In this study, they controlled diastolic coronary pressure, but different heart rates make final conclusions on coronary reserve (and, thus, minimal coronary conductance) difficult to draw.

Finally, the authors raise the question if myocardial cells are capable of increasing their oxygen extraction in situations with "excess flow" and stress. We have indications that this may, in fact, be a real phenomena. During 1 and 1.5 MAC halothane anesthesia, we found a significant increase in myocardial oxygen extraction from 55 ± 6.6% at 0.5 MAC to 72.1 ± 5.9% at 1.5 MAC, while extraction was unchanged during isoflurane anesthesia.* The "extraction reserve" may play a significant role when pharmacological effects significantly diminish vascular reactivity in the coronary bed.

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