Anesthesiology
69:152–153, 1988

In Reply:—Dr. Gilbert has introduced some interesting questions regarding interpretation of our study,[1] and we appreciate the opportunity to respond. First, he has used our data to recalculate "coronary reserve," but using a different definition of this term. Whereas we defined coronary vascular reserve as the absolute difference ($ml^{-1} \cdot min^{-1}$) between peak and resting flow when measured at a specific pressure, he has used the ratio of peak to resting coronary blood flow as an alternative definition. Recalculating reserve using this definition, Dr. Gilbert notes that "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] We agree with these mathematics, but believe that use of the simple ratio of peak to resting flow provides only an incomplete description of this physiology, and has its own potential pitfalls.

In our study, coronary vascular reserve was defined as the absolute difference between baseline coronary blood flow and flow during maximal coronary vasodilation (adenosine-induced), when measured at the same coronary pressure (fig. 1). It can be seen from inspection of this figure that when "coronary reserve" is measured at higher coronary pressures, a higher number will be measured because maximal coronary flow is higher. For this reason, comparisons of absolute "coronary vascular reserve" are best made at the same blood pressure, as we did. If we had chosen a higher coronary end-diastolic pressure at which to make all of our reserve measurements, our numbers for absolute reserve, and peak-to-resting flow ratios, would be higher.

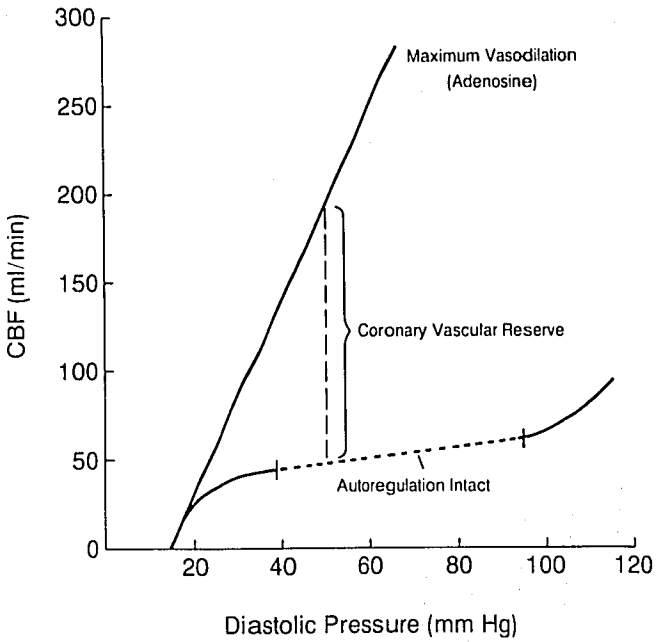

FIG. 1. Normal CBF autoregulation at a constant MVO_2. The horizontal portion (broken line) of the lower curve illustrates the pressure range where CBF remains nearly constant as coronary diastolic pressure changes. Coronary vascular reserve is measured by the distance between the lower curve and the straight maximum vasodilation line. Note that coronary vascular reserve will increase as pressure increases.

This clarifies the difference between our finding and those of Cohen, who did find a six- to seven-fold increase in peak circumflex coronary artery blood flow following adenosine injection in awake dogs. This is in contrast to a "coronary reserve ratio" of 3.9 calculated from our data at diastolic coronary pressure of 40 mmHg. Cohen did not report diastolic blood pressure in his study, but mean aortic pressure was slightly greater than 100 mmHg. If we calculate peak to resting flow ratios using data from our study at a diastolic coronary pressure of 60 mmHg (comparable to a mean coronary pressure of 100 in Cohen's study), then the coronary reserve ratio of our awake dogs is found to be 6.15, which is similar to that reported by Cohen.

Dole *et al.* have shown that peak hyperemic coronary artery blood flow (and, thus, the ratio of peak to resting flow used by Dr. Gilbert) varies directly with coronary perfusion pressure.[4] A similar point is made in Hoffman's recent review article.[5] While the ratio of peak to resting coronary flow is an acceptable definition of "coronary reserve," this index is most meaningful only when reserve is measured at similar coronary pressures. To measure coronary reserve at similar coronary inflow pressure, Gilbert *et al.*[3] used an aortic cinch to restore mean aortic pressure in halothane-anesthetized swine to equivalent levels obtained during iso-MAC anesthesia with isoflurane. We achieved the same goal by using a coronary occluder as a stenosis, and measuring absolute coronary vascular reserve with the coronary diastolic pressure set to the same level, 40 mmHg, in all animals. Our approach has the advantage of not inducing simultaneous and, possibly, confounding changes in left ventricular end-diastolic pressure, wall stress, and myocardial oxygen consumption, which may affect coronary vascular reserve, however it is measured.

One further concern we have regarding the use of peak flow to resting flow ratios after 15-occlusions to quantify coronary vascular reserve is that, if myocardial oxygen demand is significantly reduced (as during deep halothane anesthesia), a 15-s occlusion may no longer

represent a maximal stimulus, and may not be completely comparable to the same duration occlusion applied during a state of higher myocardial oxygen consumption. For this reason, we chose to measure coronary reserve pharmacologically, using adenosine, which ensured that we could attain maximal coronary vasodilation in each case.

In summary, because of the literature indicating that coronary vascular reserve is dependent on the pressure at which it is measured, we felt it necessary to make our comparisons of reserve at the same resting coronary pressure in all experimental conditions. Using a diastolic coronary pressure of 40 mmHg allowed us to obtain reserve measurements in all animals under all anesthetic conditions. Use of this diastolic pressure was, furthermore, appropriate, as this pressure was within the autoregulatory range in these dogs. When absolute numbers for coronary vascular reserve were used, the differences between awake and anesthetized animals were small and not statistically significant, despite lower heart rates in the halothane-anesthetized animals. A difference can be found if ratios of peak and resting CBF are used instead of absolute data. Although not a settled issue, we would favor use of absolute data whenever possible, particularly when "reserve" is changed by alterations of baseline coronary blood flow, rather than by diminished peak coronary flow.

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(Accepted for publication April 1, 1988.)

Anesthesiology
69:153–154, 1988

Management of an Intravascular Epidural Catheter

To the Editor:—When an epidural catheter is inadvertently introduced into an epidural vein, the recommended management is to remove the catheter from the epidural space and reinsert it *via* an adjacent interspace.[1] The obese or uncooperative patient, for example, presents a problem where the primary insertion of the needle or catheter may be difficult, only to enter an epidural vein. The use of our technique on ten obstetric patients has salvaged the initial insertion of the epidural catheter.

Over the course of time, ten patients in whom an intravascular insertion of an epidural catheter occured were treated in the following way. Using an 18-gauge Hustead needle and a loss-of-resistance technique with saline and a glass syringe, 2–3 ml of saline is injected upon entrance into the epidural space.[2] The catheter is introduced through the needle approximately 5 cm into the epidural space. The needle is removed. If blood is easily aspirated from the catheter, preservative-free saline is injected to clear the catheter. The catheter is then withdrawn a small distance, approximately ½ cm. This process is repeated until blood can no longer be aspirated. If the markings on the catheter relative to the skin indicate the catheter is still in the epidural space, a routine test dose (3 ml of xylocaine 2% with 1:200,000 epinephrine) is administered to confirm that the catheter is indeed extravascular.[3,4] We recognize that blood aspirated from an epidural catheter may come from an injured vein and that the catheter may not actually be intravascular.

This technique has proved useful in patients in whom insertion of the epidural catheter has been difficult. In none of the patients has the test dose confirmed an intravascular injection, and all subsequent injections were followed by adequate epidural analgesia. We feel this technique offers an alternative to repeating insertion of the epidural catheter at a second interspace, and may be advantageous in the patient in whom the insertion of the first catheter was technically difficult.