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Coronary Steal Models

To the Editor:—Cheng and colleagues have investigated coronary steal in a swine model and concluded that neither isoflurane nor halothane causes intercoronary or transmural redistribution of myocardial blood flow.[1] However, the model used by Cheng *et al.* is not sensitive to a steal phenomenon. The model included an occlusion of the left anterior descending artery but no stenosis of the left circumflex artery, which is most likely to supply blood flow to the collateral-dependent area. Although a single-occlusion model has demonstrated intercoronary steal with the administration of very powerful coronary dilating drugs, it is unlikely that a steal phenomenon would be caused in this model by a less powerful dilator such as isoflurane.

The authors have correctly used adenosine as a positive control to test the sensitivity of their model to a steal phenomenon. However, their interpretation of the data obtained during adenosine infusion is flawed. Steal occurs when flow is increased to one area of myocardium *at the expense* of flow to another area. Their data with adenosine fail to demonstrate steal because flow to a compromised zone did not decrease. Lower flow ratios (endocardial:epicardial and collateral:normal) are the result of increased flow to the zone in the denominator. Thus, even the powerful coronary dilator adenosine did not produce steal in their model.

Finally, and of greater concern, the issue of steal cannot be tested with the authors' experimental design, because no control measurements were made in the absence of inhaled anesthetics or adenosine. The proper comparison for a diagnosis of steal is between the flows observed at the same mean arterial pressure and heart rate in the presence and the absence of the vasodilator. These control measurements were not made. Perhaps *both* isoflurane and halothane disturbed the distribution of flow. The results neither support nor refute the hypothesis that isoflurane causes coronary steal: the data are simply uninterpretable.

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In Reply:—Buffington suggested that our model is not sensitive to the steal phenomenon. As pointed out in our Discussion section,[1] although the chronic swine model used was a single occlusion of the left anterior descending coronary artery (LAD) with no stenosis of the left circumflex artery, an angiographic study[2] and preliminary work in our laboratory demonstrated the presence of well-established collateral vessels supplying the myocardium distal to the LAD occlusion. Further evidence of collateralization occurred in each study animal because no myocardial infarct could be demonstrated in any heart after LAD occlusion. In contrast to the canine heart, where collateral vessels develop only in a narrow subepicardial layer, collateral vessels in the human and porcine hearts develop predominantly in the subendocardium with a histologic structure of abnormally thin-wall arteries.[3] The "coronary steal-prone anatomy" as initially termed by Becker,[4] comprises a total occlusion of a major coronary branch with collateral flow distal to the occlusion and proximal stenosis of a vessel supplying the collateral circulation.

However, studies show that the latter stenosis is not absolutely necessary for steal to occur.[5,6] It is the decrease in perfusion pressure distal to the stenosis, *i.e.*, at the origin of collateral vessels, that is responsible for the coronary steal phenomenon. In most of the animal studies the pressure distal to the stenosis was unknown or impossible to measure. An earlier study,[7] which compared the effects of inhaled anesthetics on myocardial blood flow, was confounded by the use of a concomitant basal intravenous anesthetic (α-chloralose) and by the fact that the coronary perfusion pressures (CPP) were considerably different when isoflurane and halothane groups were compared. We studied the effects of isoflurane and halothane as the sole anesthetic in clinical concentrations, and the CPP was tightly regulated by the inhalational agent only. This decrease in CPP mimics the decrease in CPP distal to a proximal left circumflex artery stenosis and to the origin of collateral vessels that supply distal to an occluded LAD.

Our results demonstrate that in this swine model of chronic coronary artery occlusion, the decreases in absolute flow to the collateral-dependent (CD) zones by the inhalational anesthetics were not the result of either intercoronary or transmural redistribution of coronary blood flow. Therefore, the data suggest that our model is not insensitive to isoflurane steal, but rather that isoflurane does not cause coronary steal when it is used as the sole anesthetic in clinical concentrations. This has recently received support by increasing evidence in clinical[8,9] and chronic multivessel canine[10,11] models.

With regard to the positive control with adenosine, we have shown in table 2 of our study[1] that the regional myocardial blood flow was significantly less in CD than in control zones of normal perfusion (CNT), particularly in endocardial (ENDO) regions over the range of CPP studied. In addition, CD.ENDO blood flow was significantly less in 30-mmHg CPP when compared with baseline 55-mmHg CPP. Therefore, flow to the compromised area (CD.ENDO) decreased with adenosine, and the significantly decreased CD/CNT.ENDO (fig. 1 of our study) and ENDO/EPI.CD (EPI = epicardial) flow ratios (fig. 4) were the result of coronary steal.

Our study was designed to investigate the possibility of redistribution of regional myocardial blood flow by isoflurane or halothane as the sole anesthetic after producing the specific CPPs in random order. We believe it would be questionable from the ethical point of view to perform control measurements without an inhaled anesthetic, as Buffington seems to suggest, unless a confounding intravenous agent were used. Furthermore, we think CPP is a better determinant of coronary blood