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. 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 the flow-limiting segment. Thus, the model is limited to supply blood flow to the collateral vessels. 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.

In Reply—Buffington suggested that our model is not sensitive to the steal phenomenon. As pointed out in our discussion section, although the chronic swine model used was a single occlusion of the left anterior descending coronary artery (LAD) without stenosis of the left circumflex artery, an angiographic study and preliminary work in our laboratory demonstrated the presence of well-established collateral vessels supplying the myocardial distal to the LAD occlusion. Further evidence of collateralization occurred in every 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. The "coronary steal-prone anatomy" as initially termed by Becker, 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 showed that the latter stenosis is not absolutely necessary for steal to occur. 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 animals studied the pressure distal to the stenosis was unknown or impossible to measure. An earlier study, which compared the effects of inhaled anesthetics on myocardial blood flow, was confounded by the use of a concomitant basal intravenous anesthetic (o-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 anesthetics in clinical concentrations, and the CPP was tightly regulated by the inhalational agent.

The proper comparison for the 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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REFERENCES
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