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.

CHARLES W. BUFFINGTON, M.D.
Professor of Anesthesiology
Department of Anesthesiology and Critical Care Medicine
University of Pittsburgh School of Medicine
Desoto at O'Hara Streets, Room 2405
Pittsburgh, Pennsylvania 15213

REFERENCES

1. Cheng DCH, Moyers JR, Knutson RM, Gomez MN, Tinker JH: Dose-response relationship of isoflurane and halothane versus coronary perfusion pressures. Effects on flow redistribution in a collateralized chronic swine model. ANESTHESIOLOGY 76:113–122, 1992

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

flow than is mean arterial pressure. As a result, the myocardial blood flows were compared at the same CPP over different levels as regulated by inhaled anesthetics. Adenosine served as the positive control in our model. We did not control heart rate in this study by pacing, because we set out to define myocardial blood flow under the clinical concentration of isoflurane and halothane at different CPP. We intended to study blood flows with the inhaled anesthetics as closely as possible to the clinical conditions. If it is an intrinsic property of the agent to vary heart rate with regulated CPP, the resulting myocardial blood flow will have more clinical significance in our clinical practice than if heart rate was artificially changed.

DAVY CHENG, M.D., M.Sc., F.R.C.P.C.
Assistant Professor of Anesthesia
University of Toronto
The Toronto Hospital
Toronto Western Division
399 Bathurst Street
Toronto, Ontario, Canada M5T 2S8

JOHN MOYERS, M.D.
Professor of Anesthesia

JOHN TINKER, M.D.
Professor and Head

Department of Anesthesia
University of Iowa College of Medicine
Iowa City, Iowa 52242

REFERENCES

1. Cheng DCH, Moyers JR, Knutson RM, Gomez MN, Tinker JH: Dose–response relationship of isoflurane and halothane *versus* coronary perfusion pressures: Effects on flow redistribution in a collateralized chronic swine model. ANESTHESIOLOGY 76:113–122, 1992
2. Millard RW: Induction of functional coronary collaterals in the swine heart. Basic Res Cardiol 76:468–473, 1981
3. Sjoquist PO, Duker G, Almgren O: Distribution of the collateral blood flow at the lateral border of the ischemic myocardium after acute coronary occlusion in the pig and dog. Basic Res Cardiol 79:164–175, 1984
4. Becker LC: Conditions for vasodilator-induced coronary steal in experimental myocardial ischemia. Circulation 57:1103–1110, 1978
5. Patterson RE, Kirk ES: Coronary steal mechanisms in dog with one-vessel occlusion and other arteries normal. Circulation 67:1009–1015, 1983
6. Gross GJ, Warltier DC: Coronary steal in four models of single or multiple vessel obstruction in dogs. Am J Cardiol 48:84–92, 1981
7. Buffington CW, Romson JL, Levine A, Duttlinger NC, Huang AH: Isoflurane induces coronary steal in a canine model of chronic coronary occlusion. ANESTHESIOLOGY 66:280–292, 1987.
8. Slogoff S, Keats AS, Dear WE, Abadia A, Lawyer JT, Moulds JP, Williams TM: Steal-prone coronary anatomy and myocardial ischemia associated with four primary anesthetic agents in humans. Anesth Analg 72:22–27, 1991
9. Leung JM, Goehner P, O'Kelly BF, Hollenberg M, Pineda N, Cason BA, Mangano DT. The SPI Research group: Isoflurane anesthesia and myocardial ischemia: Comparative risk versus sufentanil anesthesia in patients undergoing coronary artery bypass graft surgery. ANESTHESIOLOGY 74:838–847, 1991
10. Cason BA, Verrier ED, London MJ, Mangano DT, Hickey RF: Effects of isoflurane and halothane on coronary vascular resistance and collateral myocardial blood flow: Their capacity to induce coronary steal. ANESTHESIOLOGY 67:665–675, 1987
11. Hartman JC, Kampine JP, Schmeling WT, Warltier DC: Steal-prone coronary circulation in chronically instrumented dogs: Isoflurane *versus* adenosine. ANESTHESIOLOGY 74:744–756, 1991

(Accepted for publication April 13, 1992.)

Lingual Nerve Injury

To the Editor:—The case reported by Silva and colleagues[1] poses many unanswered questions concerning lingual nerve injuries associated with airway manipulation. Although the authors astutely recommend that pharyngoscopy be performed to determine the presence or absence of gross pathology as a cause of the injury, we should not be surprised to observe either an absence of pathology or findings that may confuse the diagnostic picture altogether.

For instance, I recently worked with a patient who was found to have hypesthesia of the left side of the tongue approximately 12 h after a 4-h anesthetic for elective abdominal surgery including tracheal intubation. Ventilation *via* mask was performed uneventfully, and oral laryngoscopy and tracheal intubation were accomplished atraumatically and without difficulty and without cricoid pressure. Of interest, the patient's endotracheal tube was consistently kept on the *right* side of the lips, oral cavity, and pharynx, with postextubation pharyngoscopy revealing an edematous and erythematous area on the *right* side of the base of the tongue immediately adjacent to the tonsillar pillar. Nerve

conduction studies performed on the tongue (facilitated by the hypesthesia) were consistent with a left lingual nerve ischemic lesion proximal to the tongue itself. The patient discussed by Silva *et al.* apparently demonstrated swelling of the left posterior aspect of the tongue only, despite having a bilateral deficit. As in that case, our patient eventually progressed to complete recovery as well.

It seems probable that, as in the case of our incomplete knowledge associated with ulnar nerve injuries,[2] the etiology of lingual nerve injuries is truly multifactorial, as suggested by Silva and colleagues, and goes beyond simple compression, impingement, or stretching. However, also similar to ulnar nerve injuries, it is imperative that we continue to avoid (and document) those factors—or, more likely, combination of factors—that we know may predispose to nerve injury, such as improper positioning, prolonged compression, low arterial inflow, and poor venous outflow.

Finally, because lingual nerve injuries are so rarely reported, prospective study investigating predisposing factors should involve more