CORRESPONDENCE

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Early Application of the Cross-suture Splint to Teeth Avulsed at Tracheal Intubation

To the Editor:—A 53-yr-old, 152-cm, 41-kg woman was scheduled for removal of a pheochromocytoma. The patient had no obvious dental caries or periodontal disease. After induction of anesthesia with 250 mg intravenous thiamylal and 10 mg vecuronium, the anesthetic resident ventilated the lungs with 5% sevoflurane for 3 min and attempted tracheal intubation. However, more than 30 s passed before the vocal cords were seen, and arterial blood pressure increased to 243/96 mmHg. The resident rushed the intubation, which led to complete avulsion of the maxillary incisors. We immediately consulted a dentist, who strongly recommended early stabilization of the teeth. The teeth were replaced into the socket in their original position, and a cross-suture splint was applied within 10 min. The patient was instructed to report to the dental clinic for subsequent observation and treatment.

On replantation, the durability of the teeth depends on which of three courses is followed: (1) Nearly normal function of the periodontal ligament is restored, in which the durability is almost the same as untraumatized teeth. (2) The root of the replanted tooth undergoes osseous replacement and eventually may cause loss of the tooth. (3) Root resorption occurs with a necrosed tooth pulp and early loss of the tooth.[1] The outcome depends largely on the first-aid treatment. Teeth are held to the alveolar bone by collagenous tissue that forms the periodontal ligament. It is imperative not to damage or dry the ligament if avulsed teeth are to be replanted successfully. The less time the tooth is out of its socket, the more successful the replantation will be. A 90% success rate occurs if the extraoral period of the teeth avulsed does not exceed 30 min.[1,2] Early dental consultation even before surgery is crucial to achieve satisfactory replantation.

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Vasomotor Effects of Isoflurane in the Coronary Circulation

To the Editor:—Park *et al.*[1] reported that isoflurane caused constriction of isolated coronary resistance vessels obtained from rat. This finding conflicts with observations obtained *in vivo* in several laboratories, including ours,[2–4] demonstrating that, when hemodynamic conditions are controlled, isoflurane causes significant increases in coronary blood flow (CBF). Because most of the resistance to CBF, by far, resides in the arteriolar segments,[5] these latter findings suggest that isoflurane is a dilator, rather than a constrictor, of coronary resistance vessels.

Park *et al.* acknowledge the increases in CBF caused by isoflurane in our *in vivo* studies and, in light of their *in vitro* findings, theorize that they are due to an opening of "nonnutritive" shunts. However, Park *et al.* provide no anatomic or functional evidence for these shunts, nor do they explain why isoflurane would cause opposite changes in vasomotor tone of the shunts and the coronary resistance vessels. Their use of the study of Gelman *et al.*[6] as support for their theory is puzzling, because Gelman *et al.* found that isoflurane anesthesia had no effect on shunting of 9-μ microspheres in the coronary circulation. These findings from Gelman *et al.* are consistent with those from our laboratory[2,7] and others[8] that indicate that coronary vasodilators, including isoflurane, do not increase the coronary shunting of microspheres.

We disagree with two points raised by Park *et al.* in their discussion. First, they state that our method of direct venous collection for assessing the amount of microsphere shunting in the coronary circulation[2] is inferior to the technique used by Gelman *et al.*, in

which tissue entrapment of two different-sized microspheres was compared. This criticism is without merit. The method of direct venous collection might be considered superior on two grounds: (1) It avoids the necessity to assume that the larger microspheres are completely trapped; and (2) It permits a determination of the size distribution of the microspheres that are shunted (and therefore of the arteriovenous pathways traversed). Second, Park *et al.* are not correct when they infer that shunted microspheres necessarily indicate flow that has passed through "nonnutritive" vessels. When a microsphere is shunted, it means only that the microsphere has passed through an arteriovenous vessel with a larger diameter than it. In the absence of more information, it is not possible to distinguish whether the shunted microsphere passed through a direct arteriovenous "nonnutritive" anastamosis or through a distended capillary.

In conclusion, when hemodynamic conditions are controlled *in vivo,* isoflurane consistently causes an increase in CBF, accompanied by a reduction in oxygen extraction.[2–4] These are classic signs of pharmacologic dilation of coronary resistance vessels. It remains to be determined whether the isoflurane-induced constriction of small coronary arteries demonstrated by Park *et al. in vitro* is a phenomenon unique to their preparation or whether it has more wide-ranging application.

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In Reply:—Although Crystal questions whether isoflurane-induced vasoconstriction of resistance coronary arteries is a preparation-dependent phenomenon without wide-ranging application, we have gathered multiples pieces of evidence to the contrary. First, this phenomenon has been observed in four different species—namely, rabbits,[1] rats,[2] pigs,[3] and human right atrial appendage arteries* (fig. 1). However, the effect of isoflurane depends on vessel size, and isoflurane dilates large epicardial arteries.[1,2] Second, isoflurane has been observed to attenuate β-adrenergic, cAMP-mediated vasodilation[4] in resistance coronary arteries. Third, in the same preparation, another anesthetic, halothane,[2] does not elicit constriction, but dilation, of resistance coronary arteries. We submit that the weight of evidence favors that the direct effect of isoflurane on small resistance coronary arteries (~100 μm) is constriction but depends on preexposure tone.[2]

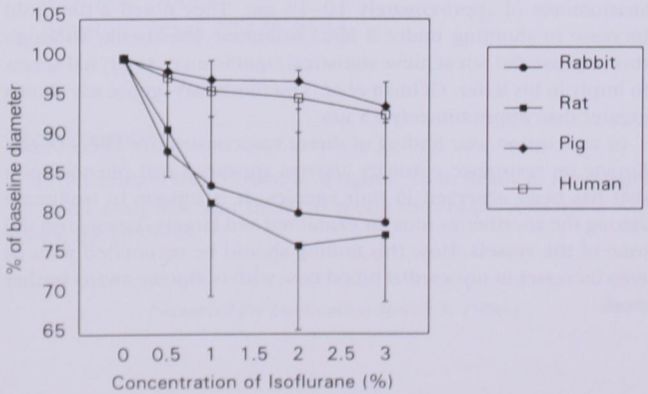

Fig. 1. Percent change from baseline diameter *versus* concentration of isoflurane for resistance coronary arteries from four species. Data are presented as mean ± SD. Isoflurane caused concentration-dependent constriction of resistance coronary arteries in all four species ($P < 0.05$ for each).

* Unpublished observation.