As is shown in their figure 1, there is a connection between the vertebral artery and rete mirabile, and further, the anterior and posterior spinal arteries may be connected with the vertebral arteries. To rule out this possibility (or our postulate), it is essential to determine the end-expiratory anesthetic concentration in the lung ventilation during bypass. If it were measured, the discussion of communication from the cerebral to noncerebral circulation would not be required.

The authors used the expression "subcortical structures such as the spinal cord." This comment gives an impression that the authors excluded the important structures between the cerebral cortex and the spinal cord, *i.e.*, the medulla, pons, midbrain, diencephalon, and cerebellum. It is well known that the brainstem reticular core strongly modulates the spinal reflexes.[6] It is thus inappropriate for the authors not to define whether the brainstem structures were included or not in the "subcortical structures."

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In Reply:—I appreciate the interest in our article expressed by Adachi *et al.* They have doubts regarding our speculative mechanisms and offer evidence (including some of their own work) against these mechanisms.

First, they previously determined that isoflurane depresses neurons in the midbrain reticular formation (MRF), whereas nitrous oxide appears to activate a supraspinal inhibitory system.[1,2] However, other investigators have found that inhalational anesthetics (halothane in particular, isoflurane to a lesser extent) may activate inhibitory responses in MRF neurons, especially at higher concentrations (*e.g.*, 2–3%).[3] Species differences may explain these discrepant results. Nonetheless, I believe such evidence clearly supports a possible mechanism of enhanced inhibition. Indeed, enhanced inhibition is a proposed mechanism of general anesthesia.[4]

Second, they have questions regarding our postulate of a "spinal shock-like" state. Our animals' condition might have been similar, but not *identical,* to spinal shock. Our animals were clearly not in spinal shock, as they point out. I remind Adachi *et al.* what constitutes a negative response in a minimum alveolar concentration (MAC) study: lack of gross, purposeful movement in response to a painful stimulus. When stimulated, however, our animals often withdrew the stimulated extremity or stiffened, and these were arbitrarily defined as negative responses. Suppression of gross, purposeful movement is just one point on a spectrum. For example, when an animal progresses from intact neurologic function to brain death (or deep anesthesia), they will first respond to painful stimuli with gross, purposeful movement, followed by nonpurposeful movement, and finally, with no movement at all. In this spectrum of neurologic dysfunction (or anesthesia), it is the intermediate stage that most often described the animals that did not "move." Brain death and spinal shock represent conditions that are more powerful suppressors of spinal cord function than those found in our study, so I agree that the term spinal shock is a bit confusing. I disagree with their belief that spinal reflexes are not influenced by the anesthetized brain. Although the "machinery" is present in the spinal cord to generate purposeful movement in response to pain, the brain can still influence anesthetic requirements.[5]

Adachi *et al.* offer their own mechanism, namely, isoflurane-rich blood flowing from the rete mirabile to the spinal cord. Although our figure shows a connection (basilar artery) between the rete and the vertebral arteries, anatomically it is small. Also, the head pressure during bypass was about 35 mmHg lower than the systemic blood pressure, and therefore, blood flow from the rete to the spinal cord seems unlikely. As regards the end-tidal isoflurane concentration during bypass, it was 0.2%, as stated in the article.

I agree that the statement "subcortical structures, such as the spinal cord" is incomplete. Prior investigators (as cited in our article) have shown that the goat cerebral circulation can be isolated to the level of the caudal medulla. In fact, we subsequently showed that our preparation excludes the systemic circulation down to the level of the caudal medulla and high spinal cord.[6]

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A Potential Complication with the Lightwand

To the Editor:—The lighted stylet is part of the armementarium available for blind intubation of the trachea. We report a potential complication of this instrument that is an otherwise useful adjunct for intubation.

A 22-yr-old male was scheduled for elective septoplasty and rhinoplasty under general anesthesia. A single-use lighted stylet (Xomed-Treace, Jacksonville, FL) was removed from the sterile package and prepared according to instructions for insertion into an 8.0 oral RAE endotracheal tube (Mallinckrodt, Glens Falls, NY). Before inserting the stylet through the tube, a clear piece of plastic tubing 2–3 mm in length was noted to be on the stylet over the bulb, protruding slightly past the bulb and adherent to the stylet. The endotracheal tube then was placed over the stylet. However, the tube extended several centimeters past the light source, and 2–3 cm were cut from the endotracheal tube to facilitate placement with the stylet into the trachea. While the tube was being cut, the small piece of plastic tubing was noted to be lodged at the preformed bend of the oral RAE tube. A second tube was prepared and the lighted stylet, without the plastic piece, was successfully used for tracheal intubation of this patient.

The instructions accompanying the lighted stylet were consulted with no mention found of the need to remove this plastic before use.

A company representative stated that this plastic recently had been added to protect the bulb from damage during shipment and should be removed before use. In view of the difficulty with detecting this plastic piece and the confusion that this is an integral part of the stylet, this poses a distinct hazard for unrecognized dislodgement into the tracheobronchial tree and difficulty with detection and recovery due to size and radiolucency. If protection is essential, then a red, occlusive and perhaps bulky covering that prevents use, a tag that notifies the user to remove the cover before use, or some other form of product label is in order, particularly to notify the occasional user who may be unfamiliar with the device.

We report this potential complication to warn other users and to encourage packaging modifications so that a useful device will remain available to us.

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