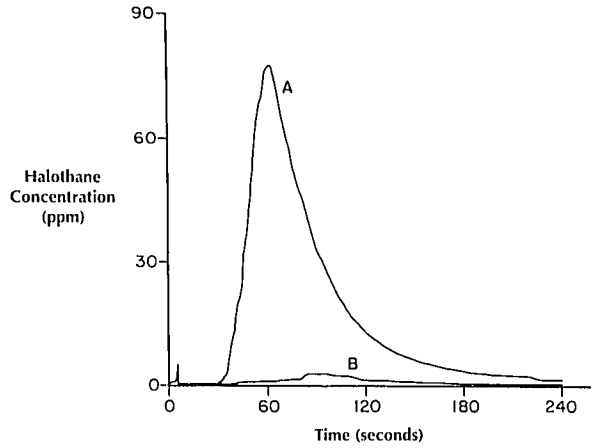

Fig. 2. Atmospheric concentration of halothane during and after the fill procedure. (A) Vaporizer filling without face tent scavenging. (B) Vaporizer filling with face tent scavenging.

fifth occasion, unintentional spillage occurred; nevertheless, the peak halothane concentration recorded was only 14 ppm.

This system has proved easy to use on Vapor 19.1 and Ohmeda Tec 3 and Ohmeda Tec 4 vaporizers. It does not interfere with the operation of either the vaporizer or the fill mechanism and does not restrict orientation of the bottle during the filling procedure. It can be used with both keyed and nonkeyed fill systems. The transparent nature of the face tent does not hinder visualization of the fluid level within the vaporizer reservoir sight glass. We believe this to be a simple solution to eliminate the usual peaks in atmospheric concentration associated with vaporizer filling and to usefully contribute to the maintenance of time-weighted levels close to those suggested in the NIOSH guidelines.† The face tent is of particular benefit when liquid agent is spilled during the fill procedure, because this normally would fall onto the anesthesia machine work surface and subsequently vaporize. Instead, both liquid and vapor are effectively scavenged.

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Exaggerated Anesthetic Requirements

To the Editor:—Recently, Antognini and Schwartz reported that the minimum alveolar concentration (MAC) of isoflurane was increased in goats in which the brain is preferentially anesthetized and suggested the importance of subcortical structures, such as the spinal cord, in generating "the purposeful movement in response to a painful stimulus" during general anesthesia.[1] This is an interesting study, and we agree with the authors in the importance of subcortical structures in the purposeful movement under general anesthesia. Nevertheless, we argue against their postulate of anesthesia of the supraspinal structures in producing the so called MAC state, *i.e.*, suppression of the purposeful movements. Citing our paper,[2] the authors suggested a possibility that isoflurane activated the supraspinal pain inhibition system and suppressed the spinal cord nociceptive neural mechanisms. This postulate is not acceptable because, contrary to nitrous oxide, which activates the spontaneous cell firing in brainstem reticular core, isoflurane does not activate but rather suppresses the brain stem reticular cell firing in a dose-related manner.[3] Their alternative postulate was that the preferential anesthesia of the supraspinal structures by a large dose of isoflurane induced a functional decerebration and subsequently "spinal shock," inducing a total suppression of the spinal reflexes. This postulate is also not acceptable. Their animals maintained a normal level of mean arterial blood pressure during the phase of preferential brain anesthesia, which contradicts the severe hypotension usually observed after brain death or transection at a high spinal cord level. Because considerable concentration of anesthetic is required to suppress spinal reflexes in spinal animals,[4,5] if they anesthetized brain "preferentially" leaving the spinal cord unanesthetized, spinal reflexes cannot be suppressed. Thus, the third and the most plausible mechanism we propose is that the high concentration of isoflurane, administered "preferentially" to the brain through bypass, distributed to the spinal cord through collateral circulation and directly suppressed the spinal cord.

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]