CORRESPONDENCE

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Halothane and Ischemic Injury

To the Editor:—Recent publications[1,2] on anesthesia-related hepatotoxicity suggest that it may be, at least partially, hypoxic in nature. We would like to make two additional suggestions in this regard.

First, if the halothane-induced hepatic damage is hypoxia related, it may not be specific only for the liver. There are a number of publications confirming this. In both canine[3] and primate[4] studies, halothane has been shown to increase the extent of infarction after occlusion of the middle cerebral artery. Halothane also has been demonstrated to have adverse effect on spinal cord ischemic injury caused by temporary occlusion of the aorta in rabbits.[5] It has been shown that halothane increases the size of myocardial infarction induced by coronary artery ligation in rats.[6] In another study, it was reported that myocardial infarct size was significantly larger in halothane anesthetized dogs when compared with fentanyl-anesthetized animals.[7]

Second, since hepatic damage was caused not only by halothane but by other anesthetics as well, it is likely that the "hypoxic" factor contributing to the liver injury is a common factor for many anesthetic agents. Of course, hypoperfusion secondary to a decrease in cardiac output and the resultant hypotension can be one such factor. We would like to point out another effect that is present in the spectrum of actions of general anesthetics—their ability to deteriorate the oxidation-reduction status due to inhibition of the electron transport chain in mitochondria at the level of NADH dehydrogenase.[8,9] This effect may contribute to the development of ischemia and it is probably common for all general anesthetics. In the spectrum of actions of a general anesthetic, there are those that may be detrimental and those with beneficial influence on the outcome of tissue ischemia. The overall result depends on the constellation of various conditions. Inhibition of the electron transport chain is a potentially adverse contribution to the ischemia.

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Mean Airway Pressure and Hemodynamic Effects

To the Editor:—In their recent study,[1] Muneyuki and colleagues concluded that the hemodynamic effects of alternating lung ventilation and synchronous ventilation are the same. We believe it is important to point out that the conclusion is applicable only to the particular circumstances of their experiment, *i.e.*, normovolemic dogs. In healthy animals, cardiac output is determined by venous return to the heart. Institution of positive-pressure ventilation causes an increase in intrathoracic pressure, which tends to impede venous return. Normally, this effect