long as 22 days following anesthesia. Especially interesting is their patient who had halothane anesthesia for repair of an intracranial aneurysm. Though the operation was successful, the patient failed to regain consciousness for nine days. Consciousness was regained only as the plasma bromide levels started to decrease. While other factors may have contributed to the delayed recovery of this patient, the data reported by Tinker *et al.*, plus those reported by others, clearly indicate the potential for prolonged sedation following halothane anesthesia exists. The clinical significance of halothane may well lie more in this side-effect of halothane than in possible histotoxic responses to the metabolites of halothane.

Additional studies are needed to define more fully the risk of prolonged sedation following halothane anesthesia. In the meantime the prudent anesthesiologist would do well to bear in mind the data of Tinker *et al.* We may need to adjust our present concepts of the indications for and contraindications to halothane.—*N.M.G.*

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Theories of Anesthesia

CEREBRAL METABOLISM AND HALOTHANE Although many anesthetics have been demonstrated to interfere with mitochondrial respiration *in vitro*, there is a considerable body of data suggesting that normal clinical anesthesia is not associated with decreased cerebral energy stores. Because of major circulatory depression, higher than normal concentrations of halothane have not been studied *in vivo*. Extracorporeal circulation permitted the administration of high concentrations (to 9 per cent) of halothane to the intact dog. When 2.3 per cent halothane was exceeded, a dose-related diminution of cerebral oxygen uptake occurred. This did not depend upon whether the EEG was active or silent. Indeed, cerebral oxygen consumption continued to decline even after the EEG had become isoelectric. Although cerebral oxygen delivery remained adequate, a progressive decrease in brain ATP and phosphocreatine developed at concentrations exceeding 2.3 per cent. This was accompanied by increased brain lactate and an increase in the lactate/pyruvate ratio. These findings, indicative of anaerobic metabolism and diminished brain energy reserves, were reversed when halothane administration ceased. At the same time cerebral oxygen uptake was greater than normal. (*Michenfelder JD, Theye RA: In vivo toxic effects of halothane on canine cerebral metabolic pathways. Am J Physiol 229: 1050–1055, 1975.*) ABSTRACTER'S COMMENT: These data indicate similarity between *in vivo* and *in vitro* anesthetic effects. However, they cannot be extrapolated to all agents. The results differ significantly from those the authors observed previously when thiopental was examined (ANESTHESIOLOGY 41:231–236, 1974). With this drug, once the EEG became silent no further diminution of cerebral oxygen uptake was found. In addition, at no time was there evidence of decreased cerebral energy reserves.