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Correspondence

Bibliography on Shock Hazards

To the Editor:—In May 1972, we completed a new bibliography on Electrical Shock Hazards in the Hospital Environment. This is available on application to interested persons. The information has been set up on our computer and will be brought up to date each summer. The computer printout has been lithographed and is available in the form of a 35-page booklet containing more than 1,000 references. Where funds are available a voluntary contribution of $2.00 is asked to cover the cost of printing and mailing. Requests should be sent to me at the address below.

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Subarachnoid Injections for Intractable Pain

To the Editor:—To confirm the clinical implications of the study, "Progressive Changes in the Concentration of Ethyl Alcohol in the Human and Canine Subarachnoid Spaces," by Drs. Matsuki, Kato and Ichyanagi (ANESTHESIOLOGY 36:617–621, 1972), I wish to report my experience with approximately 100 subarachnoid injections of alcohol for relief of intractable pain. The patients were turned to the supine position 15 to 20 minutes after injection with no untoward sequelae, such as motor paralysis or excessive sensory loss. In fact only one complication, prolonged leg and bladder paresis, occurred, in a patient in whom numerous blocks had been performed; this complication clearly was not related to the change in position.

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Brain Anesthetic Concentration—A Misleading Concept?

To the Editor:—Dr. Wolfson and his colleagues [1] suggest that the brain anesthetic concentration at a particular endpoint may serve as a basis for comparison of different agents. However, such an idea may be misleading, since the concentration in whole brain may differ greatly from the concentration at the anesthetic site of action.

The brain is a multiphasic system (about 78 per cent water, 12 per cent lipids, and 8 per cent protein),[2] and anesthetics probably act in hydrophobic subcellular areas [3, 4] such as a particular part of a membrane or of a protein molecule. Solution of anesthetic agent in other phases in the brain, such as water, is incidental to, rather than essential for, anesthesia. Thus, the overall brain concentration will vary with the proportion of essential to incidental phases if the relative affinity of the anesthetic is different for each. For example, the rat brain halothane concentration midway between the concentration permitting and that preventing movement in response to tail clamp was 27 mg/100 g ("BAC").[1] Assuming a

brain–gas partition coefficient of 6.0[5] and a brain specific gravity of 1.07, 27 mg/100 g will be produced by an alveolar partial pressure of 0.0063 atmospheres (0.63 vol per cent) —a figure which is not far from other data for the anesthetic potency of halothane.[6] However, if the site of anesthetic action has hydrophobic solvent properties similar to those of olive oil[3,4] (olive oil–gas partition coefficient 224[5] and specific gravity 0.9), then at a partial pressure of 0.0063 atmospheres the concentration at the site of action should be 1.2 g/100 g—a value greatly different from the "BAC" of 27 mg/100 g. Such considerations suggest that the brain anesthetic concentration might deviate considerably from the concentration at the site of action. Furthermore, the deviations would be different for other anesthetics.

To avoid such difficulties, anesthetizing partial pressures have been measured in the alveolar gas phase.[3] At equilibrium such partial pressures will equal those existing at the site of action, regardless of the concentration differences. In the above example, the halothane partial pressure of 0.0063 atmospheres would apply to all sites in the brain. The experiments reported by Wolfson *et al.* are an interesting demonstration of the sharp cut-off nature of the quantal response. However, for comparisons between agents, the brain anesthetic concentration may be a misleading concept.

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To the Editor:—In considering this technique initially, we did wonder about the possible dilutional effect of brain water on our final concentration. We, too, looked at olive oil–gas partition coefficients and compared these with water–gas partition coefficients. For halothane these are 244 and 0.74, respectively, a ratio of 330:1. A brain lipid-to-water content ratio of 1:6.5 (12 per cent: 78 per cent as quoted by Dr. Halsey) would lead to a dilutional effect or error of approximately 2 per cent. Similar calculations for methoxyflurane would yield a possible error of 3 per cent. On this basis, we considered it reasonable to start our investigation.

Dr. Halsey suggests that our figure of 27 mg/100 g brain represents a concentration of 1.2 g/100 g brain at the site of action and that this site consists of only 12 per cent of brain volume. From this it may be calculated that even in the total absence of halothane in the rest of the brain (patently an oversimplification) the concentration measured should be 144 mg/100 g. This, in turn, would represent an alveolar concentration of 3.4 per cent, which is far removed from any published figures for MAC for halothane in animal or man. However, the use of olive oil partition coefficients in the study of anesthetic agents is based on tradition and availability rather than the suggestion that brain lipids have the same composition as olive oil. Indeed, it has been pointed out that anesthetic potency is more closely related to solubility in naturally occurring lipids (lecithin and cephalin) than to that in olive oil.[1]

Although recent work favors the lipid rather than the clathrate theory of anesthesia, it has certainly not been proven that lipid solubility is the only factor involved, and I think it is perhaps premature to refer to the presence of anesthetic agent in any part of the brain as "incidental" to the state of anesthesia. Indeed, in one of the publications quoted by Dr. Halsey,[2] the last sentence reads ". . . attempts to