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Lipolysis by Halothane Questioned

To the Editor:—The article, "Studies of the Dual Effects of Halothane on the Lipolysis of Human Fat Cells," by Drs. J. Bennis and U. Smith (ANESTHESIOLOGY 45:379–384, 1976) falls short of its title and lacks adequate documentation to make the results interpretable for the reader. The data presented to document "the dual effects of halothane on lipolysis" are, at best, questionable, since they were obtained under different experimental conditions.

Fragments of fatty tissue, and not fat cells, were used (although the method for obtaining the latter is available).[1] More importantly, one does not know when halothane "via the gas phase above the medium" was added. Was the experiment performed when equilibrium was achieved? When was it achieved, if known? Was the rate of lipolysis linear during the two-hour incubation? The reader cannot compare the concentrations of halothane added directly to the medium with the concentrations of halothane in the experiments when it was added via the gas phase.

The results listed in Table 2 of the article are even more disturbing. Halothane, 10^{-6} M, produces an increase of lipolysis of 27.7 per cent above control with a standard error of the mean of 34.8 per cent. This result is claimed to be significant as determined by the t test for paired data, but the reader cannot confirm this. The data certainly could not be significantly different when analyzed by unpaired Student's t test. The results showing the effects of propranolol and practolol on lipolysis at different concentrations of halothane are not convincing. In fact, the standard error of the mean in the series with propranolol was as high as ±30.7 per cent of the control, and the maximum change from control in the presence of propranolol and halothane was 12 per cent. It goes without saying that, even if β-adrenergic antagonists would decrease the effect of halothane significantly, one would be very daring in making such a conclusion based on one single dose of the antagonist tested. I find the conclusion, the "lipolytic effect of halothane is exerted via direct β-adrenergic stimulation," not substantiated by the data presented.

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Magnesium Deficiency Causing Persistent Hypokalemia

To the Editor:—I wish to comment on the case of primary aldosteronism with severe total-body depletion of potassium reported by Gangat and colleagues (ANESTHESIOLOGY 45:542–544, 1976). Upon induction of anesthesia the patient had tonic muscular contractions of the upper torso and arms, and possible diagnoses of myotonia dystrophica, malignant hyperpyrexia, electrolyte imbalance, and cerebrovascular accident were considered. Subsequently a marked total-body deficit of potassium was determined to be present; it took nine days of intensive therapy to correct the deficit.

I wish to emphasize that primary aldosteronism may cause profound urinary excretion of magnesium as well as potassium. Once established, hypomagnesemia may make correction of the potassium deficiency more difficult and protracted unless magnesium is also administered.[1] Anesthetists should be aware of the relationship of magnesium to potassium and consider measurement of magnesium levels when they encounter unexplained hypokalemia.

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