In reply—This letter is a reply to comments by Dr. Bogg concerning a recent editorial I wrote regarding halothane hepatic injury. Explaining my intentions in employing such sesquipedalian terminology was not meant to be pure persiflage. The editorial was, in Dr. Bogg’s words, meant to “comment, stimulate, and provoke further interest” but not in a pedantic sense. Rather, the editorial was written with the basic philosophy of the classic Japanese haiku poem in that its meaning is entirely in the eye of the observer, and critical analysis is either impossible or infinite in scope. Our status of ignorance of the entity termed halothane hepatitis is such that we have been seriously misled by categorical statements made by well-meaning but data-poor anesthesiologists in the past. It was hoped the arcane phraseology used not only matched the scientific data available, but demanded inquiry and research for comprehension. If the problem of the toxic effect of halothane on the liver is given more inquiry and research in like fashion, it is conceivable we can state our answers to this perplexing problem in monosyllables in the future.

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Pretreatment with Nonpolarizing Muscle Relaxant Does Not Decrease Gastric Regurgitation Following Succinylcholine

To the Editor—I should like to dispute the conclusions reached by Drs. Muravchick, Burkett, and Gold in their recent paper on intragastric pressure changes produced by succinylcholine. The authors suggest that tendency to regurgitation of gastric contents into the esophagus is dependent upon the magnitude of increase in gastric pressure per se. This interpretation is witnessed by their final paragraph advocating routine pretreatment with a nonpolarizing muscle relaxant before administration of succinylcholine to patients with a full stomach.

However, it is now generally accepted that the major barrier to gastric esophageal reflux is the resting tone of the lower esophageal sphincter (LOS). In the healthy subject there is a reflex adaptive increase in LOS pressure to an increased intra-abdominal pressure (and intragastric pressure), thus preventing reflux. The tendency to reflux is therefore proportional to the barrier pressure (LOS pressure minus intragastric pressure).

Failure to differentiate between gastric pressure and barrier pressure is also suggested by an error appearing on page 183 of Dr. Muravchick’s paper: “Both narcotics and antialagologues appear to reduce intrinsic gastric muscle tone.” The reference to antialagologues is a paper by Professor Brock-Utne and his colleagues, who examined the effect of glycopyrrolate on the lower esophageal sphincter. These authors found that glycopyrrolate had no significant effect on gastric pressure, but there was a highly significant reduction in lower esophageal sphincter pressure and also barrier pressure.

It is unfortunate that Dr. Muravchick did not refer to the paper by Smith et al. These authors observed that following an induction dose of thiopentone (3 mg/kg body weight), there was a small but significant reduction in barrier pressure. At the height of muscle fasciculations produced by succinylcholine, although intragastric pressure was elevated, there was a correspondingly greater increase in LOS pressure so that the net effect on barrier pressure was a small increase. From these results it was concluded that there was no increased tendency to regurgitation in normal subjects at the height of fasciculations. It has also been noted in dogs that succinylcholine-induced fasciculations produce transient increases in both LOS and gastric pressures, but no change in barrier pressure. Our preliminary unpublished observations suggest that after induction of anesthesia with thiopentone followed by succinylcholine, barrier pressure is lowest during flaccid paralysis when the stage of fasciculations has passed.

This analysis leads to the conclusion that pretreatment with a nonpolarizing muscle relaxant is unnecessary.
when succinylcholine is used following induction of anesthesia. There is no evidence to support "the old concept."

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REFERENCES


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The Recording of Succinylcholine-induced Fasciculations

To the Editor.—The paper by Muravchick et al.1 attempts to relate the integrated electromyogram (IEMG) of fasciculating abdominal muscles following succinylcholine (SCh), to changes in intragastric pressure, but the method of EMG assessment seems open to comment.

Firstly, in our experience of over 800 cases,2-4 it has proved impossible to achieve reliable and consistent results using electronic integration of an input from surface electrodes. The main problem has been the marked baseline fluctuation of the EMG tracing, due particularly to movement associated with the fasciculations, as mentioned by the authors. We recorded IEMG values for resting tone (awake control) similar to those of Muravchick et al., but our mean IEMG values recorded during fasciculation activity were much greater, around 80 microvolts.

The use of integrals was abandoned in favor of planimetry of the raw filtered signal. This revealed, in virtually all cases, an almost complete disappearance of EMG activity (resting tone) within 15 s of thiopental induction, yet Muravchick et al. could only demonstrate a significant decrease in IEMG activity in three out of 48 patients. Their sampling period of 20 s following the completion of thiopental injection may have been too early to demonstrate significant IEMG depression, and it would be of interest to learn if the value of IEMG did indeed fall, between 20 and 40 s post-induction, in those cases where it was measured.

Recording from the biceps brachii muscle, we found that in the vast majority (96 per cent) of cases, fasciculation activity commenced between 8 and 15 s following injection of SCh, and in one case 40 s later. Furthermore, peak EMG activity did not usually develop for at least another 10 s, so that the first 20 s sampling period used by Muravchick et al. may have been too premature to allow for significant electrical activity to develop. Extrapolation from the figures for the 20-40 s sampling period, however, also fails to show a significant increase in IEMG amplitude.

We were able to demonstrate in 11 patients almost complete abolition of SCh-induced EMG fasciculation activity following pretreatment with a large dose of d-tubocurarine (10 mg), whereas only one of 11 patients in this series showed a significant fall in IEMG activity. Integration of the EMG signal thus would appear to produce misleading results and be of dubious advantage when handling SCh-induced depolarization.

Finally, the choice of recording sites may have been unfortunate. The surface electrodes presumably were recording from the rectus abdominis and possibly the external oblique muscles. We found these muscles to be remarkably "resistant" to SCh in that only two of 30 patients showed significant EMG fasciculation activity following SCh. This finding was attributed to these muscles being composed largely of red, slow-acting muscle-fibers, which were resistant to depolarization.4 These muscles were electrically silent, (or showed ECG only) following SCh, while surrounding muscle groups showed...