In reply:—This letter is a reply to comments by Dr. Blogg 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. Blogg'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 Nondepolarizing 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[1] 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 nondepolarizing 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).[2] 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.[2,3] 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[1]: "Both narcotics and antisialogogues appear to reduce intrinsic gastric muscle tone." The reference to antisialogogues is a paper by Professor Brock-Utne and his colleagues, who examined the effect of glycopyrrolate on the lower esophageal sphincter.[4] 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.[4]

It is unfortunate that Dr. Muravchick did not refer to the paper by Smith *et al.*[5] 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[6] 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 nondepolarizing muscle relaxant is unnecessary