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*In reply:* The epinephrine solutions used in this study were as follows: 1) E (1:200,000 epinephrine in normal saline solution) prepared by diluting 1:1000 epinephrine with normal saline—*pH* = 6.1; and 2) LE (1:200,000 epinephrine with 0.5% lidocaine) prepared by diluting a commercially available 1% lidocaine containing 1:100,000 epinephrine (Astra, Fujisawa) with normal saline—*pH* = 4.9.

Thus, the differences of these solution could partly be responsible for the variation in epinephrine uptake as in-

dicated by Sosis and Temple. Further study is needed to identify the respective roles of *pH* and lidocaine on absorption of epinephrine.

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### Concerning Assay of Serum Cholinesterase with Succinylcholine

*To the Editor:*—The recent publication of a method for measuring serum cholinesterase using succinylcholine as the substrate<sup>1</sup> requires comment. Although the authors claim that this is “a new enzymatic method for assaying cholinesterase activity,” we had published an assay using the same principle<sup>2</sup> more than a year earlier. Our procedure reliably identifies individuals at risk of suxamethonium apnea and is suitable for use as a preoperative screen.

There are a number of serious problems in the analytical procedure published by Wakid *et al.* In particular, they use a reagent blank in which water rather than serum is added. This is unsatisfactory as it does not allow for the nonspecific catalysis hydrolysis of succinylcholine by substances other than cholinesterase.<sup>3</sup> Clearly, their reagent blank would be better replaced by a serum blank in which cholinesterase is inhibited by physostigmine.<sup>2</sup> Their assay measures hydrolysis of succinylcholine in the presence of phenol and aminoantipyrine; however, our

experimental work endeavoring to develop a kinetic procedure showed that these are both inhibitors of cholinesterase. Even though their phenol and aminoantipyrine concentrations are slightly lower than our lowest concentration in table 1, there will still be significant interference. These problems are overcome by performing the assay in two stages, *viz.*: 1) enzymatic hydrolysis of succinylcholine; 2) inhibition of cholinesterase by physostigmine and determination of the choline produced.<sup>2</sup>

Although Wakid *et al.*<sup>1</sup> have assayed cholinesterase in the presence of these inhibitors, they paradoxically obtain average enzyme activities comparable with those by our two-stage method. This is probably explained by their invalid calibration system. Their assay was apparently standardized when “serum was replaced by water, and succinylcholine by choline chloride.” Their choice of choline chloride was unwise as it is extremely deliquescent and therefore cannot be used; the nondeliquescent choline iodide should have been used.<sup>4</sup> Further, their calibration system cannot apply to a kinetic assay; it is appropriate only to an end-point assay. This is because the rate of the reaction of the standard depends on the initial high choline concentration, whereas, in the serum samples choline is continually being generated. A valid standard reflecting the kinetic analysis would be to use a serum sample pre-calibrated by our two-stage method.<sup>2</sup>

Fortuitously, an increase in “cholinesterase activity” due to the standardization procedure is apparently compensated for by the loss of activity due to performing the assay in the presence of the inhibitors phenol and aminoantipyrine. Although the method, as published, cannot be recommended, the kinetic approach has obvious potential for automation if these problems can be overcome.

TABLE 1. Interference of Phenol and Aminoantipyrine with Cholinesterase

Inhibitor	Inhibitor Concentration	
	Low*	High†
Phenol	91‡	40
Aminoantipyrine	95	68
Phenol + aminoantipyrine	84	22

\* Phenol 20 mmol/l, aminoantipyrine 4 mmol/l.

† Phenol 1 mmol/l, aminoantipyrine 0.2 mmol/l.

‡ Activity as a percentage of a normal plasma pool in the absence of any inhibitors (determined as ref. 1).