Correspondence

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Succinylcholine-induced Apnea

To the Editor:—In his article,[1] Dr. Oropollo states, without qualification, that a patient when given succinylcholine is at risk from prolonged apnea if the level of pseudocholinesterase in his blood is below normal. Prolonged apneas are not caused by low levels of enzyme *per se* (except in rare cases of complete absence of the enzyme), but by the qualitative abnormalities of the pseudocholinesterase enzyme as determined by dibucaine and fluoride inhibition studies.[2] The most common of the qualitatively abnormal homozygote enzymes is the E_1^a E_1^a genotype, which is associated with an apnea time of two to three hours after a therapeutic dose of succinylcholine.[2] Even though the enzyme level in these instances is usually below the lower limit of normal, it is the qualitative abnormality and not the quantitative deficiency of the enzyme that results in the prolonged response to succinylcholine.

When the normal enzyme is quantitatively deficient, the duration of apnea in most cases is prolonged by only a few minutes, which is considerably less than that seen in people with the abnormal type of enzyme.[3] As these brief periods of apnea should cause no problem for a fully trained anesthetist, it is quite unjustifiable to issue warnings about succinylcholine sensitivity, with all the attendant anxiety this arouses, without first genotyping the enzyme. When this has been done, and only then, those patients (and members of their families) who are genuinely at risk can be identified and warned about the use of succinylcholine.

CHRISTOPHER D. D. HUTTER, F.F.A.R.C.S.
Senior Registrar in Anaesthetics
Queen Elizabeth Hospital
Queen Elizabeth Medical Centre
Edgbaston, Birmingham, B15 2TH
United Kingdom

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2. Lehmann H, Liddell J: Human cholinesterase (pseudocholinesterase): Genetic variants and their recognition. Br J Anaesth 41:235–244, 1969
3. Editorial: Suxamethonium apnea. Lancet 1:246, 1973

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In reply:—Dr. Hutter's viewpoint would be well taken had my article been based on the assumption that low enzyme levels are equated with low levels of enzymatic activity. Rather, it was assumed "that both phenotypes, generally speaking, are to be found in the population with low enzyme levels." This assumption can hardly be construed as an absolute and is not in conflict with the findings of numerous investigators.

The article attempted to make three points: 1) that in a large surgical population (now more than 5,000), we observed that approximately 5 per cent had low pseudocholinesterase levels; 2) that, in this group, there is a small number of individuals who may manifest an untoward sensitivity when exposed to succinylcholine; 3) that, in view of the relatively inexpensive automated methods now available, enzyme levels that could assist the anesthesiologist in providing a more uneventful course of anesthesia could be made available to him. We have found that our patients and surgical colleagues have not in any way been made anxious by a full discussion of the clinical implications of our data.

ANTHONY T. OROPOLLO, M.D.F.A.C.A.
Director of Anesthesia
Montclair Community Hospital
Montclair, New Jersey 07042

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