Pseudochoinesterase Deficiency

Quick Facts by Core Concepts Anesthesia Review, LLC

What You Must Know

- 1. The deficiency will prolong the action of succinylcholine.
- 2. The deficiency can also cause an increase in the toxicity of ester local anesthetics.
- 3. The dibucaine number reflects the activity (not quantity) of pseudocholinesterase.
- 4. Deficiency inheritance pattern suggests a single gene locus.
- 5. A normal dibucaine number is 76-86% (homozygous normal).
- 6. Homozygous abnormal patients will have a dibucaine number of 18-26% and will experience very prolonged paralysis following succinylcholine administration.
- 7. Evaluation of a patient with a prolonged response to succinylcholine should include a dibucaine number, fluoride number and absolute activity of pseudocholinesterase.

Pseudocholinesterase, also known as butyryl-cholinesterase, is the plasma enzyme responsible for the degradation of succinylcholine and ester-based local anesthetics. Pseudocholinesterase deficiency can occur as an inherited disorder or an acquired disorder. Acquired causes include:

- 1. Pregnancy (third trimester)
- 2. Liver disease
- 3. Cancer, malnutrition and debilitating diseases
- 4. Collagen-vascular diseases
- 5. Uremia
- 6. Exposure to anticholinesterase agents such as neostigmine or organophosphorus agents
- 7. Exposure to cyclophosphamide
- 8. Cardiopulmonary bypass

In addition to the dibucaine number, a fluoride number is also commonly used to assess the activity of the enzyme. A low percentage of inhibition of the enzyme by either dibucaine or fluoride reflects a low level of activity of the enzyme.

Approximately 96% of the population has normal levels and activity of pseudocholinesterase. 2.5% of the population is heterozygous for normal enzyme activity, with dibucaine numbers between 50-70%. Heterozygous patients experience moderate prolongation of succinylcholine activity. Finally, the remainder of the population has dibucaine and/or fluoride numbers of 18-26% and will experience significantly prolonged neuromuscular blockade after receiving succinylcholine.

Additional Reading:

Barash, PG, Cullen, BF, Stoelting, RK, Calahan, MK and Stock, MC. *Clinical Anesthesia*. Philadelphia: Lippincott Williams & Wilkins; 2009:613-614