**Succinylcholine-Induced Hyperkalemia: New Perspectives**

Hyperkalemia occurs in a small subset of patients after succinylcholine (SCh) administration and can be severe and fatal. In a review of cases and the pathophysiology of succinylcholine hyperkalemia (SChK), the author describes 2 mechanisms that lead to the disorder: upregulation of acetylcholine receptors and rhabdomyolysis.

Upregulation is caused by a change in the subunit type of acetylcholine receptors and by an increase in their density as they spread over the muscle surface outside the motor endplate area. Causes of upregulation include burns, severe muscle trauma, upper or lower motor neuron denervation (e.g., stroke or spinal cord injury, respectively), and prolonged ICU care (bed rest, steroids, prolonged neuromuscular blockade). In such situations, SChK can occur if SCh is given at least several days after the underlying condition has become established. If recognized, SChK can usually be treated successfully (mortality, 11 percent). This syndrome is not prevented by pretreatment with a competitive neuromuscular blocking agent.

Rhabdomyolysis occurs when already-myopathic muscle is exposed to SCh. Hyperkalemia caused by rhabdomyolysis can be severe and rapidly fatal (mortality, 30 percent) despite treatment, presumably because of the sheer surface area of abnormal muscle membrane capable of leaking potassium. The most common cause is underlying muscular dystrophy, but a few idiopathic cases have been reported. SChK associated with crush injuries is mediated by upregulation, not rhabdomyolysis.

**Comment:** This precisely written review article on a major toxic effect of one of emergency medicine's most important drugs is worth reading. SCh remains the drug of choice for ED intubation, especially after the recent withdrawal of rapacuronium from the market. All who use SCh should be aware of its side effects.

— **RM Walls**

*Published in* Journal Watch Emergency Medicine *May 16, 2001*

**CITATION(S):**