

Skeletal Neuromuscular Blocking Agents
Self-Study Objectives and Questions for Pharm 210, Fall Term 2009
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Note: You do NOT need to read the section of the chapter that is devoted to ganglionic blocking agents.

Terms that you should be able to recognize or define

curare	nondepolarizing (neuromuscular blocker)
depolarizing (neuromuscular blocker)	blocker)
malignant hyperthermia	pseudocholinesterase
neuromuscular blocker (blockade)	quaternary
neuromuscular junction	

Drugs

tubocurarine (“curare” – prototype nondepolarizing skeletal neuromuscular blocker)
pronounced TOO-bo-cure-AR-een
succinylcholine (depolarizing skeletal muscle blocker)
pronounced suck-sin-ill-KO-leen

Objectives

After reviewing your notes from class, and reading the corresponding chapter in Lehne (16), you should be able to state, describe, or recognize the:

1. the anatomy of the somatic nervous system; the key neurotransmitter and receptor type involved in skeletal muscle activation; and what happens, physiologically, when those cell receptors are activated. Compare and contrast those characteristics with cholinergic/parasympathetic neural control of smooth muscle and cardiac muscle.
2. the mechanisms of actions of nondepolarizing and depolarizing neuromuscular blocking agents, and state how these actions affect using one class, rather than the other, in specified clinical situations.
3. three specific uses for neuromuscular blocking agents, and monitoring and other measures that are necessary when they are used.
4. the main risks and main cause of death of administering neuromuscular blocking agents; describe steps to manage potentially fatal responses.
5. the class of drugs used to reverse the effects of nondepolarizing neuromuscular blockers, and describe the mechanism by which they cause that reversal; and explain why pharmacological reversal is not used when succinylcholine is the neuromuscular blocker.

6. the etiology, signs, and symptoms of malignant hyperthermia; the drugs associated with a high risk of that condition; and interventions to be implemented should it develop.

Questions to “think about”

1. What will usual pharmacologic doses of atropine, the prototype muscarinic receptor blocker, do to activation of skeletal muscle? Why?
2. What are the likely *direct* effects of giving tubocurarine, in usual pharmacologic doses, on airway smooth muscle tone, heart rate, and motility of the GI and urinary tracts? Why?
3. Why is it essential to ensure, as best as possible, that a patient who is going to receive a neuromuscular blocker have normal serum electrolyte levels, particularly potassium and magnesium?
4. Some texts describe the effects of tubocurarine and similarly classified neuromuscular blockers as “stabilizing” skeletal muscle cell membranes, and membrane potential. In contrast, succinylcholine destabilizes them. Can you envisage how these descriptions relate to the mechanisms of action of these two distinct classes of drugs, both of which paralyze skeletal muscle?
5. Explain the basic biochemical process that enables skeletal muscle to contract and then relax normally, and what becomes abnormal such that it contributes to many of the consequences of malignant hyperthermia. Identify the drug used as an adjunct, and explain in simple terms how it works on abnormal skeletal muscle metabolism.
6. A patient with no history of cancer has had abdominal surgery and develops malignant hyperthermia in response to an interaction between succinylcholine and halothane. She has to be admitted directly to the ICU. Given the negative history for cancer, how can she develop *malignant* hyperthermia?
7. A patient with a genetic deficiency in serum cholinesterase activity is given an “otherwise correct” dose of succinylcholine to facilitate endotracheal intubation. How would the response to this neuromuscular be different from normal? What drug would be administered to reverse the succinylcholine’s effect, and would that approach be used only for a cholinesterase-deficient patient?