

A Theory of Mechanism and Nomenclature of Neuromuscular Blocking Drugs

Mini Review

After practicing anesthesiology and intensive care medicine for about 35 years my idea is: commonly the neuromuscular blocking drugs are classified as:

- A. Depolarizing neuromuscular blocking drugs.
- B. Nondepolarizing neuromuscular blocking drugs.

Really, regarding the mechanism of action of these drugs (Figures 1 & 2), this classification and nomenclature is being to some extent incorrect. Regarding the nondepolarizing neuromuscular blocking drugs there will be a certain way that the drug is blocking ionic channel of nicotinic receptor and by closing the receptor [blocked] no ion exchange will occur and no action potential will initiated and progressed, leading the muscle to be relaxed. However, when dealing with depolarizing drugs, their action on neuromuscular nicotinic receptor is differs completely, as these drugs seems to depolarize the receptor make it opened like the ligand acetylcholine, and ion exchange will occur but as action of depolarizing drugs is much longer than the ligand (minutes versus milliseconds) then a some type of no ion exchange will further occurs [equilibration or inactivation of voltage-gated sodium channels] the condition indeed some call it

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desensitization, when equilibration of ions occur then no further action potential will be initiated and the muscle therefore relax [not because of block but instead because of prolonged receptor state of opening]. In this case depolarizing drugs cannot consider as blocking drugs, rather than like if the acetylcholine prolonged its action. Therefore, I believe it is more logical and accurate to change the classification of neuromuscular blocking drugs as follow:

- I. Depolarizing neuromuscular relaxing [non-blocking] drugs.
- II. Nondepolarizing neuromuscular blocking drugs.

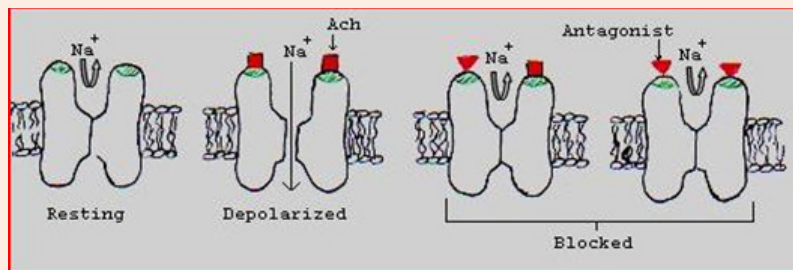


Figure 1: The mechanism of blocking of neuromuscular nicotinic receptor by antagonist (nondepolarizing block).

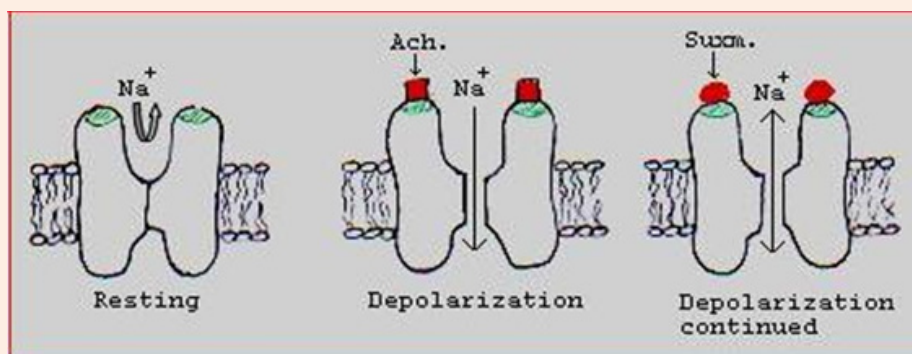


Figure 2: Depolarizing drugs act to open the nicotinic receptor channel and keeping it opened the condition which leading to equilibration of ion exchange through the channel and therefore no further depolarization occurred.