

- Reactivated by pralidoxime- like an antidote (Given early)
- Another reason for given cholinesterase inhibitors is to reverse effects of non-depolarising NMJ blockers.
 - End of operation
 - Increases ACh levels- compete with antagonist (allows ACh to last longer in synapse so it can out-compete the drug) → increase EPP.
- N.B. effects of depolarising blocker made worse, **ACh acquires Suxamethonium- like action!**
 - If you have someone paralysed with a depolarising blocker, you make things worse because you are adding agonist to agonist, so you allow ACh to acquire Suxamethonium activity because you allow ACh to itself depolarise the membrane so much that you get a depolarisation block.
- Myasthenia gravis:
 - Autoimmune: antibody against nicotinic AChR
 - Muscle weakness → progressive... death
 - Muscle weakness bc failure of NMJ transmission.
 - Cholinesterase inhibitor Drugs: initial benefit:
 - Increase ACh levels to try and overcome the decrease in nAChR's
 - Management:
 - Edrophonium
 - Short duration, used as diagnostic test of drug efficacy.
 - Neostigmine
 - Drug used clinically to control symptoms, Medium duration, muscarinic side effects.
 - Pyridostigmine
 - Another used for symptoms. Better oral absorption, long duration, less powerful.

