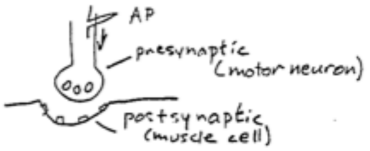
SYNAPTIC FUNCTION:

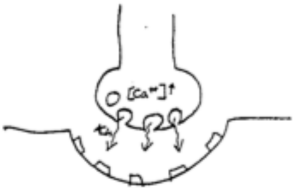
the neuromuscular junction

1) AP in motor axon terminal



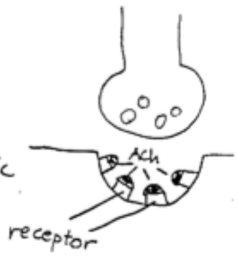
2) $g_{Ca^{++}}$ increases - Ca^{++} enters axon terminal from extracellular medium

3) As a result of higher intracellular Ca^{++} pre-synaptic vesicles bind with pre-synaptic membrane and liberate contents (the neurotransmitter, acetylcholine - ACh) into synaptic cleft.

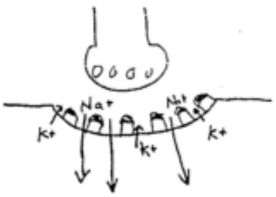


4) ACh diffuses across cleft (~200-500 Å)

5) ACh binds with receptors on post-synaptic (ie. muscle) membrane. The portion of post-synaptic membrane containing receptors is <u>end-plate</u>.



6) The binding of ACh to receptor causes a <u>brief</u> (only so long as ACh not cleaved by enzymes) change in conductance of post-synaptic membrane - $g_{Na^+}\uparrow$ and $g_{K^+}\uparrow$ which results in a net positive current inward (the end plate current or <u>EPC</u>)

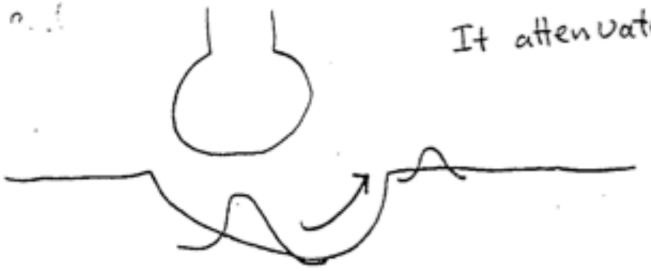


7) The conductance of the membrane quickly returns to normal (ACh broken down from receptors) but the brief EPC resulted in a build up of ⊕ charges in the endplate. This build up of ⊕ charges or this depolarization is called the end plate potential (<u>EPP</u>)

Note: ACh no longer bound to receptors

note: more positive than over here (not an actual reversal of potential the way an AP would be)

8) The EPP moves along the muscle cell membrane (just as any passive depolarization would)



It attenuates with distance - ⊕ charges leak out

9) If the EPP is large enough to drive the E_m of the (muscle cell membrane) zone of AP initiation past E_{crit}, then an AP is initiated and is propagated along the muscle membrane.

10) Properties of EPP

a) non-propagated (i.e. attenuates)

b) graded (size depends on how much neurotransmitter is bound)

3) end plate is not refractory ∴ EPP can summate

Note; neuromuscular junction = high safety factor synapse

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