► Subject: physio (Fakher) Sunday 1 /Mar/ 2020

- Nerve terminal: nerve ending
→ it contains mitochondria: because energy is essential For releasing ACh

- Motor end plate:
⇒ Formed of Folded specialized muscle membran.
1) Folded: to increase surface area.
2) Specialized: because it contains receptor For Ach

⇒ Jumping of excitation from nerve ending to muscle:

1) the impulse reach the nerve terminal, so it opens Na channel and Ca^{+2} channel

↳ بيعمل حركة تحفيز للانتقال العصبي م، ودخول الكالسيوم

Ca wich is present in high concentration in neuromuscular cleft, will pass in to the nerve ending

2) passing of Ca will trigger the viscles (Ach viscles) to move from the cytoplasm of nerve ending, then there will be rupture of these viscles, Ach will released

3) Ach will (or binds to) react with (binding Ach receptor), and Na channels will be opened.

→ types of Na channel:

A) electrical gated Na channel: لما يحدث تحفيز عصبي

B) chemical gated Na channel: لما تصل السيال العصبي تحفز

4) Once there is opening in Na channel, there will be inflate of sodium, so there will be shifting in RMP, and producing A.P + producing excitation of muscle membrane

⇒ the viscles that contain Ach opens spontaneously, but the amount of Ach is small, so this will produce minor changes in R.M.P of NMJ.

*the amount of Ach in viscles called quantum. and once this small amount of Ach reach the Motor end plate, it will shift the R.M.P, but it doesn't reach the Firing Level, so that there will be minor changes in R.M.P of muscle, So it is called ~~mini~~ miniature end plate potential

*Drugs affecting N.M.J:

1) Curare: بتغطي عل الreceptor فما تقدر الAch
تشتغل عالreceptor

2) Hexamethonium: it's structure similar to Ach, but Ach is destroyed by Acetylcholinesterase in N.M.J, but we don't have the substance that destroy hexamethionium, So that if we have Hexa., it cause persistent depolarization by occupying the receptor.
رح يصير فتح لقنوات الصوديوم بدون إغلاق يعني ما رح
يرجع لـ R.M.P وبالتالي يؤدي إلى (paralysis)

⟹ we give Hexamethonium to the patient ~~...~~ during operation, we need relaxation of muscles

3) Botulinum toxin : cause defect in N.M Stimulation.

N.M.J :

3) Hormonal and metabolic (Malignant) :
(Lambert Eaton)

Types of Muscles:

* Skeletal : is responsible for Locomotion.

⇒ Muscle Fiber : multinucleated

Myofilaments:
1) Myosin : Dark bands.

⇒ Bands:
* A : DARK
* I : LIght

→ H Zone : area contain only myosin filaments.

* contraction of the muscle: shortening of Sarcomere Length or shortening of Z-Z line

→ the mechanism of shortening and strecching (relaxing) depends on increasing or decreasing of Z-Z line distance.

→ G protein forms double helix called actin.

→ Troponin:

1) C: bind to calcium
2) T: bounded to tropomyosin.
3) I: inhibitor, that push tropomyosin on the cavity of the actin molecule

* Excitation-contraction coupling.

* Excitable membrane in the muscle include:

1) Sarcolemma (membrane of muscle fiber)

2) endoplasmic reticulum: contain high ~~concentr~~ concentration of calcium + T tubule.

→ calcium will ~~receive~~ the cytoplasm, and receive to (passin)

cross bridges: Site of actin + myosin binding

→ Ca bind to troponin C, once it bind, it will shift the trapomyosin away from the cavity of actin and this will Lead to binding of actin to myosin

⇒ Each Ca will Lead to opening of 7 active site of actin.

→ so if we increase the releasing of Ca, there will be opening of many cavities, So we will have more binding of actin with myosin, So we will have more Strength of contraction

→ we will lose energy during reuptake of calcium to reach relaxation, because relaxation needs ATP

→ If there is no ATP, Ca pump will not work, there is contineous contraction without relaxation, this process called Rigor

Rigor : contineous contraction without relaxation after death

⇒ it occurs if the death occure ~~in~~ at Severe stress

Ca pump ← : تسحب الكالسيوم من السايتوبلازم
للـ EPR وتقلل تركيز الكالسيوم من 10^{-3} لـ 10^{-7}