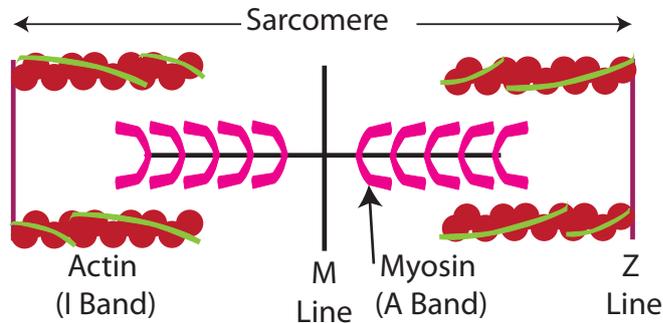


First 2010  
VIVA 4

This station is concerned with the physiology and pharmacology related to the musculoskeletal system. Candidates were asked to draw and label a sarcomere, outline the physiological events relating to muscle contraction, length – tension relationship, muscle relaxants, malignant hyperthermia and dantrolene.

**“Please draw and label a sarcomere”**



**“Outline the physiological events leading to contraction”**

- There is a propagation of nerve signal
- Release of ACh at the presynaptic junction
- Activation of nicotinic receptors at the neuromuscular junction
- Propagation of action potential in the muscle
- Release of calcium from the sarcoplasmic reticulum
- Exposure of actin to myosin
- ATP dependent process of muscle shortening due to the movement of the myosin tails

**“What is the tension length relationship?”**

- is based on the sliding filament theory
- can be represented by a Starling force curve, optimal position enables good overlap
- contracted is too much overlap and over stretched is insufficient overlap

**“Discuss the pharmacology of suxmethonium”**

- is a depolarising neuromuscular blocking agent used primarily in rapid sequence induction
- is presented as a clear colourless solution in vials of 100mg in 2 ml it has a shelf life of 4 weeks
- its structure is two linked ACh molecules
- mechanism -mimics the action of ACh, binding to the two alpha subunits and depolarises the muscle
- side effects: raised IOP/gastric pressure, myalgias, arrhythmias, MH, anaphylaxis, sux apnoea, increased K<sup>+</sup>
- The effective dose for 95% of patients having NMB is around 0.3-0.6mg/kg, but is usually given 1mg/kg
- Only about 20% reaches the NMJ due to metabolism by hydrolysis and plasma esterases
- It has a very short half life of several minutes, and is excreted in the urine as inactive metabolites

**“Can you explain the mechanism of malignant hyperthermia”**

- the mechanism of MH is due to a genetic susceptibility to the accumulation of intracellular Ca
- this only occurs in the presence of certain anaesthetic agents
- it is believed that it is the result of an abnormal ryanodine receptor
- the symptoms include rigidity, which increases ATP use, causing hyperthermia and lactic acidosis

**“What is the treatment for MH?”**

- appropriate history taking before the anaesthetic can avoid the risk of MH
- early recognition look for unexplained tachycardia, hypercapnia (ETCO<sub>2</sub>) or masseter spasm as first signs
- treatment with dantrolene which binds the ryanodine receptors and prevents further Ca release
- increased ventilation, and oxygenation, monitoring for hyperkalaemia and ECG changes
- cooling and insertion of an IDC is important supportive measures