**POE #5: Hitting the Wall**

**Topic**

Unit 5- Muscular System

The phenomenon below has been simplified for the purposes of this introductory anatomy and physiology course. As you progress in your academic and professional careers you may learn more extensive details related to this scenario.

**Introduction to the Phenomenon**

*As you read through the scenario below underline specific facts and information you find important to the situation*

When running a marathon every runner fears of “hitting the wall”. This metaphorical wall generally occurs around mile 20 in the marathon, and it is a point in the race that every runner hopes to avoid. Hitting the wall, also known as “bonking,” refers to the point when a runner experiences extreme exhaustion and energy depletion. When a runner hits the wall, they typically become fatigued, dizzy, weak, and sometimes incapable of finishing the race. The two images below are of runners at mile 20 in their marathon and based on the images it is clear that runner A has hit the wall while runner B is completing her race…strong!



**Driving Question**

*How are blood glucose homeostasis, electrolyte balance, neurotransmitter levels, and muscle anatomy and physiology affected when a runner hits the wall in a marathon?*

**Initial Hypotheses/Predictions**

*In the box below, please provide your initial ideas about a possible answer to the driving question above.*



**Relevant Data & Analysis Questions**

***ALL analysis questions are italicized in the pages below***

**Runner Information:**

Each of these runners is healthy, young, and fit with no indications of disease or health problems. Both runners are running the same marathon and the pictures above represent the same mile marker (mile 20) in the race.

|  |  |
| --- | --- |
| **Runner A** | **Runner B** |
| **Blood Glucose**  Pre-Race: 105mg/dl  During Race (Image): 58mg/dl  After Race: 70mg/dl | **Blood Glucose**  Pre-Race: 130mg/dl  During Race (Image): 110mg/dl  After Race: 100mg/dl |
| **Caloric Intake**  Day before race: 2550  Race Morning: 220  During the race: 0 | **Caloric Intake**  Day before race: 3000  Race Morning: 665  During the race: 500 |
| **Water & Electrolyte Intake**  Pre-Race: 6oz. water  During Race: Water only at miles 5, 10, and 15 | **Water & Electrolyte Intake**  Pre-Race: 8oz. water & electrolyte mix  During Race: Water & electrolyte mix at miles 5, 10, and 15 / Water only at miles 3, 6, 12, & 18 |
| **Muscle Glycogen Storage**  Pre-Race: 75%  During Race (Image): 0% | **Muscle Glycogen Storage**  Pre-Race: 100%  During Race (Image): 50% |
| **Liver Glycogen Storage**  Pre-Race: 75%  During Race (Image): 0% | **Liver Glycogen Storage**  Pre-Race: 100%  During Race (Image): 30% |

*Why would low muscle and liver glycogen levels affect blood glucose levels; identify any specific cells, organs, hormones, neurotransmitters, and pathways.*

*Low muscle and liver glycogen will ultimately lead to low blood glucose levels. This is because during exercise, the muscles will first use up their storage of glycogen, by breaking it down into intramuscular glucose for the muscle to use and generate ATP for energy. Then as the muscles begin to run low, they will require glucose from an external source which would be from the blood, but the blood is getting its source of glucose from the liver. So if the muscles run low, they demand more glucose from the liver’s glycogen stores, and then once the liver glycogen is used up the only source left is in the blood, which isn’t very much and it will deplete quickly. Specific cells and hormones involved in this process would be alpha and beta cells in the pancreas, which are responsible for controlling glucagon and insulin. Glucagon signals the breakdown of glycogen to glucose, and insulin signals the storage of glucose to glycogen by initiating glucose intake into the cells.*

*Explain how Runner B’s body is maintaining her blood glucose levels; identify any specific cells, organs, hormones, neurotransmitters, and pathways. What would you expect to be different compared to runner A?*

*Runner B is capable of maintain blood glucose for a few reasons. One she fueled properly both before and during the race. By intaking adequate calories before her race she was able to fill her muscle and liver glycogen stores to 100%, whereas runner A started her race with partially depleted glycogen stores (75%) already putting her at a disadvantage. Runner B also ingested carbohydrates throughout her race, so as she depleted some of her muscle and liver glycogen, she replenished these stores by ingesting carbs. Her body was then able to use insulin to pull this external source of glucose into the muscle and liver, and store for later usage during the race. Then when her body demanded more glucose for energy, glucagon was activated and broke down her remaining glycogen stores to provide glucose for her race. This did not occur in runner A, because she did not fuel properly and even if her insulin and glucagon were functioning properly there wasn’t any external glucose or internal glycogen available to help maintain her blood glucose levels.*

In *POE #2 we learned that Sandy had low blood calcium. How did her low blood calcium affect her synapses and the communication to effectors?*

*Sandy’s low blood calcium first lead to a lack of acetylcholine release from the presynaptic neuron, because calcium is responsible for signaling neurotransmitter vesicles to dock and release their contents into the synaptic cleft. Then the low calcium also led to sodium gates on the post synaptic cell remaining constantly open which led to unwanted and sporadic action potentials. This is similar to runner A, because she is also depleting her electrolytes such as calcium and sodium, so the low calcium would lead to less acetylcholine release and less action potentials. It could also lead to leaving sodium gates open which could cause cramping, but since her sodium is also low, she likely isn’t getting as many action potentials which would explain her inability to continue running and extreme fatigue.*

*Explain how Sandy’s body would work to maintain her blood calcium levels; identify any specific cells, organs, hormones, neurotransmitters, and pathways. Would runner A experience similar muscle issues as Sandy?*

*Sandy’s body wasn’t able to maintain blood calcium homeostasis, because she was lacking the PTH hormone due to her hypoparathyroidism. But if her body were functioning normally, the low blood calcium levels would alert the parathyroid glands to release PTH, which would then signal the osteoclasts in bones to breakdown the bones and release more calcium into the blood to maintain homeostasis. Because without adequate blood calcium all of the above-described issues would occur at the NMJs within the body. Since runner A was also low on blood calcium, she would likely experience less Ach release and fewer action potentials in the muscle fibers. This would lead to an inability to contract the muscles, and overall weakness and fatigue. Runner A also could experience muscle cramping due to dysregulation occurring in sodium channels on the muscle fibers, ultimately leading to muscle cramps and spasms similar to Sandy.*

*As a quick refresher,* ***circle*** *answers in the chart below that describe the interconnection of neuron and muscle physiology.*

|  |  |  |
| --- | --- | --- |
|  | At Rest | Depolarization |
| Membrane Potential | POSITIVE / NEGATIVE | POSITIVE / NEGATIVE |
| Na+ Concentration | Inside Cell: HIGH / LOW  Outside Cell: HIGH / LOW | Inside Cell: HIGH / LOW  Outside Cell: HIGH / LOW |
| K+ Concentration | Inside Cell: HIGH / LOW  Outside Cell: HIGH / LOW | Inside Cell: HIGH / LOW  Outside Cell: HIGH / LOW |
| Ca2+ Concentration | Inside Cell: HIGH / LOW  Outside Cell: HIGH / LOW | Inside Cell: HIGH / LOW  Outside Cell: HIGH / LOW |
| Contractile State (Muscle Fiber) | Relaxed / Contracted | Relaxed / Contracted |
| Signal Firing State (Neuron) | Firing/ NOT Firing | Firing/ NOT Firing |
| Action Potentials Occurring | YES / NO | YES / NO |

**Runner Electrolyte Levels**

|  |  |  |
| --- | --- | --- |
| **Electrolytes** | **Runner A** | **Runner B** |
| Na+ | Low | Normal |
| K+ | Low | Normal |
| Ca2+ | Low | Normal |

*Now,* ***CIRCLE*** *the components of the NMJ that would be affected in Runner A due to her low electrolyte levels.*

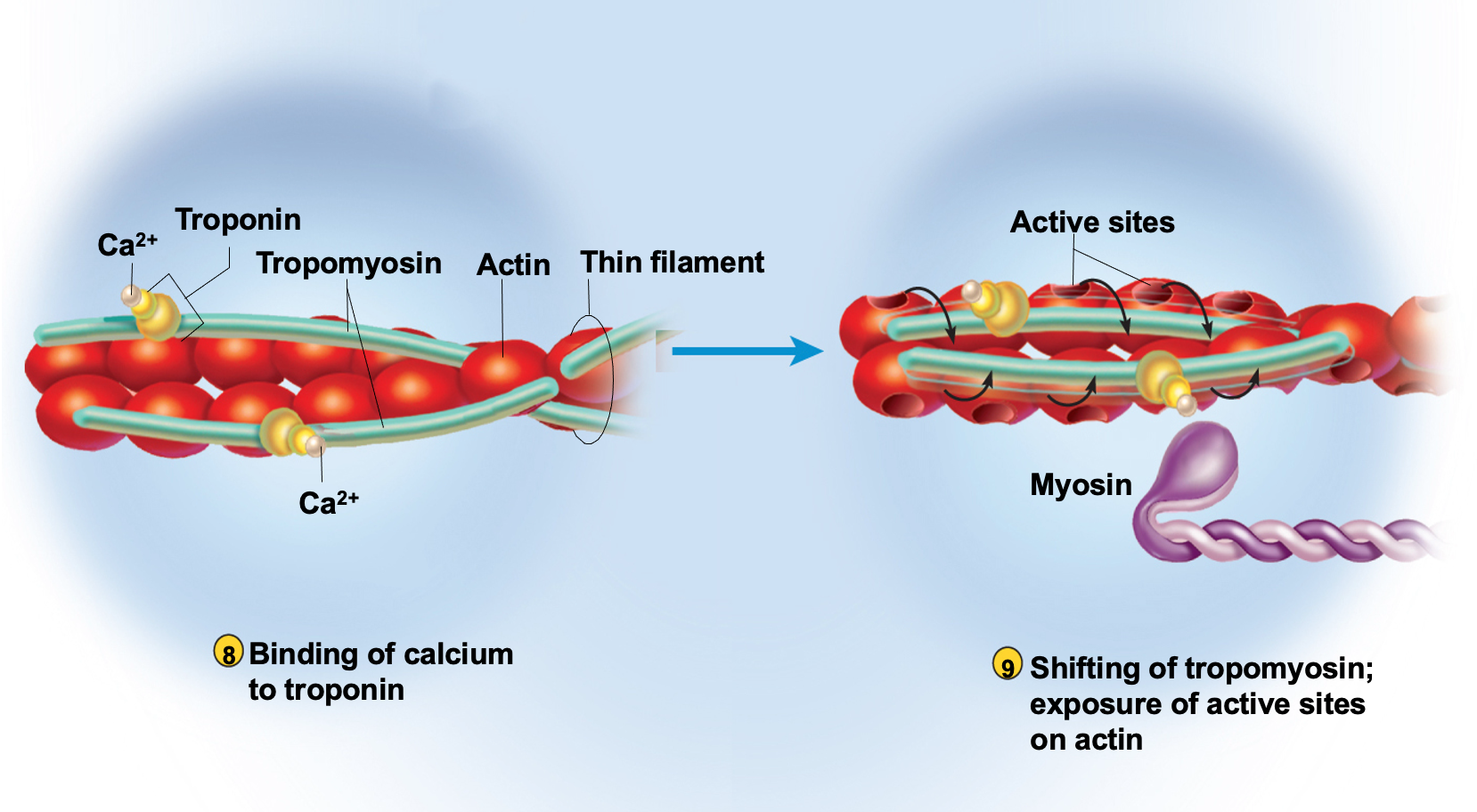
*Explain the role of each ion at the NMJ.*



*Na+ 🡪 Influx of Na+ into the postsynaptic cell will increase resting membrane potential, and generate action potentials (depolarization)*

*K+ 🡪 K+ will rush out of the postsynaptic cell after the action potential has occurred in order to bring the cell back to resting membrane potential (repolarization)*

*Ca++ 🡪 Ca++ is responsible for initially signaling the release of Ach in the pre synaptic cell, but it also controls Na+ channels on the post synaptic cell and will open or close them depending on its binding state*

**Calcium’s Role in Muscle Contraction**

*Explain how the events in steps 8 and 9 would differ between the two runners; identify any specific proteins, cells, organs, hormones, neurotransmitters, and pathways.*

*Due to runner A depleting her electrolytes, by mile 20 her levels of calcium were very low, so the above steps would likely not occur. Since calcium plays a role in binding to troponin to move tropomyosin off of the myosin binding sites on actin, if this does not occur then the muscles cannot contract. In order for muscles to contract myosin must bind to actin to form a crossbridge and pull the filaments closer together to ultimately contract the muscle. In runner B these two steps would still occur, because she has enough calcium available to allow for actin and myosin binding.*

*What other steps in excitation-contraction coupling would be affected in runner A? Identify any specific cells, organs, hormones, neurotransmitters, and pathways. How would these contribute to her hitting the wall?*

*The initial excitation of the muscle fiber would also be affected since this takes place in the neuromuscular junction. If there is not enough calcium available in the presynaptic neuron then it cannot release acetylcholine, and without acetylcholine the ion channels on the target muscle cell won’t open and an action potential will not fire. Without the initial excitation, all of the downstream steps would also be affected, so due to low calcium and a lack of Ach there will be less muscle contraction, and ultimately this would contribute to runner A hitting the wall, because she wouldn’t be able to continue running*

*In POE #3, we learned that inhibiting the release of ACh affected the communication at a synapse. Fatigue can reduce the amount of ACh released by motor nerve fibers. Compare and contrast these two scenarios. Then give two examples discussed in class where neurotransmitters, toxins, or receptors are affected and explain the results on the body.*

*In POE #3 the reason Ach wasn’t being released was because the botulinum toxin was preventing the docking and exocytosis of Ach from the presynaptic neuron. This is different from the running scenario, because the reason runner A is releasing less Ach is because her electrolyte levels are depleted, so there isn’t enough calcium available to signal the release of Ach. But the effects experienced in both scenarios are similar because in POE #3 Fred experienced flaccid muscle paralysis, which is similar to runner A, because her muscles essentially were not able to contract due to a lack of Ach, leaving them in a similar state as Fred but in her case, there is no toxin so it isn’t necessarily paralysis likely its muscle fatigue. Muscle fatigue also almost acts as a positive feedback loop, where the muscles use up all of the ATP, and once glycogen is depleted ATP generation declines, also the motor nerve fibers use up their Ach which makes them less capable of stimulating muscle fibers ultimately continuing the cycle of fatigue.*

*Curare is another toxin example from class, and this competes with Ach for binding sites on the postsynaptic receptors, ultimately blocking Ach from binding and initiating an action potential leading to flaccid muscle paralysis. Certain pesticides can also affect the NMJ, because they contain cholinesterase inhibitors that bind acetylcholinesterase and prevent the degradation of Ach. This will lead to spastic paralysis where there are constant action potentials causing the muscles to remain in a contracted state, because they are incapable of relaxing due to the continual Ach stimulus.*

*Explain different strategies the body uses to remove neurotransmitters from the synapse.*

*In order to remove NTs from the synapse, the body will first discontinue the nerve stimulation to signal the muscle to stop contracting. Then acetylcholinesterase will be released into the synaptic cleft to breakdown Ach and reabsorb the fragments into the synaptic knob. This will ultimately halt the stimulation from Ach, because it is no longer present to initiate action potentials.*

*From POE #4 we learned that the sympathetic nervous system is responsible for excitatory responses. Complete the graphic below to understand how the SNS plays a role in running a marathon.*

|  |  |
| --- | --- |
| Physiological Process/System | Describe the Response During a Marathon |
| Nervous System Activation | *SNS activation, because we want excitatory pathways to be initiated to prepare for exercise* |
| Heart Rate | *Increased to supply more blood and oxygen to the muscles* |
| Respiration | *Increased to supply more oxygen to the blood and muscles* |
| Blood Glucose | *Increased blood glucose in order to provide the muscles with enough ATP to contract at a high rate*  *High glycogen to glucose conversion* |
| ATP Production | *High ATP production in order to support long and sustained muscle contractions* |
| Blood Flow | *Increased blood flow to muscles*  *Decreased blood flow to digestive system* |

**Answer the following questions, connecting as much of the material learned in Units (and POEs) 1-4.**

*List 4 variables that affect muscle force. How might the previous 4 POEs alter each variable.*

1. *# of fibers stimulated to contract (POE #4: SNS activation- More activation will stimulate more fibers / POE #2 More calcium = More Ach and stimulation of muscle fibers)*

1. *Thickness of each muscle fiber (thick fibers = more power)*

1. *Frequency of stimulation (POE #2 & #3: NMJ functions- More calcium available = more muscle stimulation due to increased Ach release; But if there is a toxin present (botulinum toxin) it will prevent Ach release and decrease the frequency of stimulation)*

1. *Initial length of muscle fibers when at rest*

*Describe the metabolic pathway used to create ATP in these two women. Which runner would likely have less ATP production? Describe specific processes during muscle contraction that would be affected from low ATP levels.*

*Aerobic cellular respiration would be the metabolic pathway used, and Runner A would likely have less ATP production, because she has less glucose available. She also probably suffered from oxygen debt, because her fatigued muscles required more oxygen to generate ATP but this higher demand could not be met and thus, she was producing less ATP than runner B.*

*Low ATP levels could result in:*

* *An inability to contract muscles (excitation-contraction coupling not occurring)*
* *Muscle fatigue, because ATP declines as glycogen is consumed*
* *Slowing down of Na/K pumps, so there is an inability to control action potentials and RMR*
* *Less capable of stimulating muscle fibers*
* *CNS fatigue which would lead to less SNS activation and overall slowing down of various metabolic processes (i.e. muscle contraction/function)*

*Which muscle fiber type would be dominant in each of these female marathon runners? Which muscle fiber type would be dominant in a sprinter? Before her next marathon, what type of training could runner A work on to improve the performance of this fiber? Give examples and provide an explanation.*

*The fiber type that would be dominate in the marathon runners would be Type I fibers which are slow twitch and slow oxidative fibers. Muscle fiber types that are dominate in sprinters would be Type IIA and Type IIX fibers because these are fast oxidative and fast glycolytic fibers.*

*Runner A would benefit from endurance training, because this style of training improves fatigue resistant muscles, which means there will be more slow twitch fibers composing her muscles. If she has a greater number of slow twitch fibers, they will aid in producing more mitochondria, glycogen, and they will contain more blood capillaries. Endurance style training would include activities such as running, cycling, rowing, swimming, etc. Also performing these exercises for longer durations would aid in increasing type I fibers, so going on longer runs and performing longer run workouts such as tempo runs would help runner A improve her performance of this fiber type.*

*List and describe 5 variables that affect muscle strength.*

* 1. ***Muscle size****- Larger muscles will contain more fibers and aid in muscle strength*
  2. ***Fascicle arrangement****- pennate are stronger than parallel, and parallel stronger than circular*
  3. ***Size of motor units****- larger the motor unit = stronger contraction*
  4. ***Multiple motor unit summation*** *–* ***recruitment****- when stronger contraction is required, the nervous system activates more motor units*
  5. ***Temporal summation****- nerve impulses usually arrive at a muscle in a series of closely spaced action potentials*

*greater frequency of stimulation, the more strongly a muscle contracts*

* 1. ***Length – tension relationship****- a muscle resting at optimal length is prepared to contract more forcefully than a muscle that is excessively contracted or stretched*
  2. ***Fatigue****- fatigued muscles contract more weakly than rested muscles*

*What are some strategies runner A could have implemented before and during her marathon to prevent her from hitting the wall? (****Hint: Discuss nutrition, electrolyte intake, and training****)*

*Several strategies runner A could have implemented include:*

***Nutrition****: Runner A could have improved her nutrition both before and during her race. For example the night and morning before her marathon, runner A did not intake enough calories to fully replenish her muscle and liver glycogen stores, so she already started the race at a slight deficit (75% capacity). If she had eaten more calories and carbohydrates both the night and morning before her race, so could have started the marathon with more glycogen and ultimately lasted longer potentially avoiding hitting the wall. Then during the race she didn’t intake any carbohydrates, so her body had no external source of glucose to use as her body’s internal stores began to run out. Without any additional glucose during the race she was unable to continue to perform and run once she depleted her glycogen and blood glucose stores.*

***Electrolyte Intake****: Electrolytes play a major role in muscle function, so maintaining a proper balance and amount of electrolytes such as sodium, calcium, and potassium is important for performance especially during a long endurance event like the marathon. Runner A did not intake any electrolytes during her race, so as she was sweating and losing electrolytes, she had no way of replacing them within her body. She could have prepared some water and electrolyte mixes to intake at different points during the race to avoid depleting her sodium, calcium, and potassium.*

***Training***: *Runner A could have benefitted from a few different training strategies before her marathon race. The first is that she could have increased her endurance training to prepare her muscles and increase her type I fibers to aid in lasting longer during the race. She also could have trained in a similar fashion to the way she raced. Since she chose to not intake calories or electrolytes during the race, she should have done more fasted training in order to prepare her body to become used to not having additional glucose or electrolytes after she depleted her own stores. The body can slowly adapt to using fat as a source of fuel instead of glucose, but in order to do so one must train in a way that encourages fat oxidation. This means more fasted running, switching to a very low carbohydrate diet, and training without additional fuel or glucose. Without preparing the body during training it is impossible for the body to be ready for these conditions on race day.*

**Observations**

*After examining the data and answering the analysis questions above, describe interesting observations and patterns you believe are relevant to explaining the phenomenon. You can include both textual and visual observations in order to help organize the data from above. (Include at least 10 important pieces of data and evidence that will aid in your final explanation of the phenomenon below)*



**Explanation**

*Based on the data and analysis questions above, please provide an answer to the driving question(s) in the box below. Remember to include data from above as evidence, important ideas from previous units, and the concept of homeostasis in your response.*

**Driving Question(s)**

*How are blood glucose homeostasis, electrolyte balance, neurotransmitter levels, and muscle anatomy and physiology affected when a runner hits the wall in a marathon? Compare Runner A to Runner B in order to explain a normal homeostatic response to exercise (runner B) to an abnormal homeostatic response to exercise (runner A).*

