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| **Issue: #8** | **August 2011** |  |

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| **Dear Exercise Physiologist,** Thank you for being part of our community. **ASEP is the specific voice for (historically under-represented) Exercise Physiologists.** Please use this Newsletter as a link to ASEP resources from scientific journals to professional papers, to employment and related opportunities. And be sure to click on "More On Us" at the left for the ASEP-Newsletter's parent web site.Yours in health, -Lonnie Lowery and Jonathan Mike, ASEP-Newsletter Editors  |

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| **Editor's Corner** |  |
| editorial **Overqualified and Undercertified**It's likely I've commented on this before but it's worth revisiting in any case. I once had a friend - a doctorally trained exercise physiologist (EP) - who, after a few years of teaching, went looking for clinical work. After running into a few brick walls he was finally told: "Doctor, you need a specific (bachelor's level) certification for this position". Imagine. The interviewer went on: "Sir, you are overqualified and undercertified."  This begs the question, "when did university degrees become so devaluated?" The need for some level of standardization aside, this scenario puts professional groups and their certificates in the driver's seat as opposed to even the best universities. This is saddening. If professional groups (some with corporate and financial interests, some at odds with others) are monitoring the universities' curricula (via accreditation), who is monitoring the professional groups?   Further, how is it that an individual who has actually been involved in composing various certification exams gets shut out by a "certified" interviewer looking for him to hold that same certificate? This happens in fields other than exercise physiology as well. Did you know there are currently law suits and contentious pleas submitted to various state licensure boards and assemblies, in which PhD-level persons are pointing out how ludicrous it is that they are prohibited from giving advice in their own field? Especially when some of their students, with 1/4 their training, can? Again, there is indeed a need for standardization in knowledge, skills and abilities (KSA). Certificates and even licenses should play a role in assuring competency in practice. But when, in a healthcare practice setting, a two-year Associates degree trumps a PhD, things have gone awry. It's little wonder there is massive growth in two-year vocational-type programs and a serious decline (or at least a flatlining by comparison) in graduate school enrollment.  Some professional groups, like physical therapists, do require doctoral-level training (not a PhD per se) while others, like dietitians, voted down the need for graduate schooling (and have a tiny percentage of their population at the PhD level). What is YOUR stance? Should the need for standardization in "KSAs" allow certificates to become more important than a university degree? If a professional needs both, what level of schooling is required? Is the comparative loss of growth in PhD programs a "dumbing down" of America? These are not easy issues, and the above thoughts are not those of ASEP as an organization. But they do suggest careful thinking is required as academic disciplines become professions. Yours in health,Lonnie Lowery PhD    |
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| **Ask the EP** |  |
| **Q: What are the detailed steps of overall muscle contraction, from resting membrane potential to the contractile process?**Our knowledge of muscular contraction and the sliding filament theory originated from H.E. Huxley, and A.F. Huxley in the early 1950's. These steps of muscle contraction have been modified over the last several decades due to advances in neuromuscular research, which have added to our overall understanding of the contraction process. The steps of neuromuscular contraction are detailed in this section. RMP and Action Potential The resting membrane potential (RMP) is -70mV. Once input is received at the dendrites of the neuron, it moves to the axon hillock, and passes along the entire axon to the nerves terminals, where it connects the muscle. Once input reaches the axon hillock, the membrane potential reaches threshold at -50/-55mV. At this point, depolarization occurs as the sodium (Na+) gates open and sodium rushes in. This creates a more positive charge on the inside of the cell and the membrane potential now reaches +30mV. This creates the overall action potential. Repolorization occurs immediately following depolarization, resulting in the return of the RMP. Due to an increased time delay, the depolarization causes an increase in the permeability to potassium. Potassium then exists the cell making the inside more negative. After the depolarization stimulus is removed, the sodium gates close and entry is slowed. These activities quickly restore the RMP to the original value of -70mV. However, the action potential does not move in a wave like formation along the entire axon. It actually jumps or hops from one area to another. There are two primary mechanisms by which this occurs. The first mechanism involves the insulating cells called the schwann cells, that contain a lipid protein substance called myelin. This myelin sheath provides an insulation effect around the outside of the axon. The primary benefit of the myelin sheath, or the myelinated fiber increases the overall conduction velocity of the action potential, which can increase up to 100 times faster. The second mechanism involves the influx of sodium. Once sodium rushes into the cell, this creates more positive charges on the inside of the cell, and pulls the negative charges over into a more positive direction, which initiates the action potential. This process continues along the entire axon but can only occur in specific gaps or spaces between the myelin segments. These gaps are known as Nodes of Ravier. These Nodes assist in increasing the conduction velocity of the action potential and are important for the process of transmitting these signals. This "hoping", or "leaping" of the action potential is known as Saltatory Conduction. ContractionThe action potential then moves along toward the synaptic cleft and activates the L-Voltage Gated Calcium Channels. Once the action potential (AP) activates the calcium channels, calcium rushes into the cell. Calcium is sensed by a variety of proteins in the cells, specifically the calcium dependent protein kinase. This allows the addition of phosphate groups on other target proteins. A specific target protein that originates inside the vesicles in termed Synapsin.Moreover, the action potential activates the calcium channels, which activates the protein kinase, which then phosphorylates synapsin. This results in the exocytosis of the vesicles. The neurotransmitter acetylcholine is release from the synaptic vesicles and diffuses across the synaptic cleft on the neuromuscular junction (NMJ) and binds to the acetylcholine receptors. The binding of acetylcholine to its receptors causes an influx of sodium and results in depolarization of the sarcolemma. Depolorization of the sarcolemma results in the action potential moving into the transverse tubules, which stimulates a specific receptor called the Dihydropyrodine, or DHP receptor. These receptors are linked to another receptor termed the Ryanadine, or RyR receptor, which is attached to the sarcoplasmic reticulum, creating calcium release from the SR. Calcium then binds to Troponin C to produce the change in Tropomyosin. The Troponin- CA++ complex induces a conformational shift in Tropomyosin uncovering the actin binding sites, allowing myosin to bind to actin. The actin/myosin binding allows the S-1 units on the globular region of the myosin head to be moved into the cocked or energized position. When ATP binds to myosin, it hydrolyzes via the ATPase enzyme into ADP and Pi and produces angular movement causing the 'power stroke', and is initiated when myosin cross-bridges bind to actin. If ATP is replenished, and if calcium is still present due to neural stimulation, contraction cycling, or excitation contraction coupling (ECC) will continue. Relaxation occurs when calcium is no longer being received at the NMJ, causing calcium to be pumped back to the sarcoplasmic reticulum via active transport. The removal of calcium from troponin causes tropomyosin to recover the actin binding sites, and overall contraction ceases. Jonathan Mike, MS, CSCS, USAW, NSCA-CPTDoctorate StudentCo Editor, ASEP Newsletter        |

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| Thank you for perusing our opinions, facts and opportunities in this edition of the ASEP-Newsletter.  **Sincerely,** Lonnie LoweryAmerican Society of Exercise Physiologists  |

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