So far here are some of the points that you should have gained from the readings, the lectures, and the discussion board:

**Resistance training programs can produce a 25% to 100% improvement in strength within 3 to 6 months.**

**Muscular power is the product of strength and speed of the movement.**

Though two individuals can lift the same amount of weight, if one can lift it faster, she is generating more power than the other.

**Muscular endurance is the ability of a muscle to sustain repeated muscle actions or a single static action.**

So the question becomes what roles do neural and hypertrophic pathways play in the gain in strength. Here are some things to think about:

On the neural side, we rely on the following:
- **Recruitment** - Recruitment of additional motor units for greater force production.
- **Counteraction of autogenic inhibition** allowing greater force production.
- **Synchronization** - Reduction of coactivation of agonist and antagonist muscles.
- **Rate Coding** - Changes in the discharge rates of motor units.
- **Changes in the neuromuscular junction**.

On the muscle fiber, we rely on the following:
- **Increase of muscle size after long-term resistance training** due to changes in muscle fiber number (fiber hyperplasia) or muscle fiber size (fiber hypertrophy).

Where hypertrophy is:
- The numbers of myofibrils and actin and myosin filaments increase, resulting in more cross-bridges.
- **Muscle protein synthesis** increases during the postexercise period.
- **Testosterone** plays a role in promoting muscle growth.
- Training at higher intensities appears to cause greater fiber hypertrophy than training at lower intensities.

And the subfactor of hypertrophy; hyperplasia would be:
- Muscle fibers split in half with intense weight training.
- Each half then increases to the size of the parent fiber.
- **Satellite cells** may also be involved in skeletal muscle fiber generation.
- It has been clearly shown to occur in animal models; only a few studies show this occurs in humans too.

What we see in strength gains from studies are:
- Early gains in strength appear to be more influenced by neural factors.
- Long-term strength increases are largely the result of muscle fiber hypertrophy.

See the attached figure for a visual description of strength gains.

Dr. E.

As I described before, on the neural side, we rely on the following:

**Recruitment** - Recruitment of additional motor units for greater force production. Theoretically speaking, the body should only recruit the number of fibers necessary to cause contractile forces that will overcome the external force on the muscle. However, this is actually a training adaptation. Remember back on Selye and the stress response. If you are a novice lifting a sufficient weight that produces the stress response (above homeostatic levels), the response to this weight is immediate and overwhelming. To generate the force, the body contracts ALL of the muscle fibers in the affected area to ensure adequate contractile force. As the body adapts to the weight over time, it limits the contractile response to only those fibers that are necessary to generate an adequate response. Again generally speaking; we should see the following response for muscle recruitment:
- Force<60% of maximum contractile force should only recruit type I fibers.
- ~60%Force>80% of maximum contractile force should recruit all type I fibers and ALL type IIa fibers and only those type IIb fibers necessary to generate an adequate response.
- At maximal force generation, all fiber types would be recruited. How do we come to this conclusion? Well studies have
shown that at low weight high reps, we see improved hypertrophy and strength generated by the type I fibers but not much change in the Type Ila & Ilb fibers. However in studies where the weight training included forces greater than 85% of 1 RM, we see increases in size and contractile force of Type 1 fibers, but we also see significant increases in Type Ila & Type Ilb fibers.

The counteraction of autogenic inhibition allows for greater force production. This is the suppression of the Golgi tendon organ (tension receptor). The Golgi tendon produces feedback to the CNS that protects generating too much contractile tension in the fibers, to protect from muscle damage. As a training adaptation, the Golgi tendon is suppressed, allowing for greater force production that equates to greater muscle fiber tension through improved and coordinated action potentials of the neuron. The whole idea of plyometrics is to increase suppression of the Golgi tendon in and effort to desensitize its response further, which in turn allows for greater contractile force. Now for a little neural science: Action potentials are the language of the nervous system. They are generated at the axon of a cell by a strong depolarization that opens voltage-gated channels. Once initiated, they travel along the axon and reach the axon terminal. Here, the action potential triggers the influx of Ca++ into the cell, which, in turn, causes the release of a neurotransmitter from synaptic vesicles. An excitatory neurotransmitter will open Na+ channels, causing a depolarization of the postsynaptic membrane called an excitatory postsynaptic potential (EPSP). An inhibitory neurotransmitter, on the other hand, will hyperpolarize the cell, producing an inhibitory postsynaptic potential (IPSP). Both EPSPs and IPSPs can occur on the same neuron and can be spatially and temporally summed. Synaptic integration is the interaction of many IPSPs and EPSPs. Summation of many EPSPs is essential for the production of an action potential great enough to create an action potential across the neural muscular junction which will cause a muscle contraction. The Golgi tendon regulates the IPSPs which will negate the any postsynaptic action potential. It is the post synaptic action potential that we need to generate force; therefore we need adequate EPSPs to create an action potential at the post synaptic cleft, which will in turn create an action potential at the neural muscular junction, thereby generating muscle fiber contraction. It is important to distinguish between an EPSP and an action potential. The EPSP, which occurs only on the dendrites and cell body, will decrease with time and distance from its point of origin, while the action potential is all-or-nothing and is usually only found on the axon. Also, EPSPs can be added one on top of the other while the action potential cannot.

Synchronization - Reduction of co-activation of agonist and antagonist muscles. This actually allows for a reduction in the muscle groups that are activated in response to the external forces. Think about a novel task that you have completed recently, for me it was removing the motor from Scout. Did you happen to notice that the next morning; there were muscle aches in areas that you did not knowingly use? That is because, much like the fiber recruitment theory, the body activates all of the agonistic and antagonistic muscles necessary to assure task completion. As the adaptive process begins, the body allows for the reduction in and timing of contractions (synchronization) of only the necessary muscles to generate both the force and stability necessary.

Rate Coding - Changes in the discharge rates of motor units. Rate coding is the final actual neural control that we can measure. What would happen if you stimulated the muscle with an action potential, causing a muscle twitch, and then stimulated it again before the muscle fully relaxed? This would trigger another twitch that would add on top of the first one. The resulting tension in the muscle would be doubled. This can take place because the twitch is a type of mechanical event relying on protein interactions. It can therefore be added. An action potential is an all-or-nothing event that cannot be summed due to the absolute refractory period caused by the inactivation of Na+ voltage gated channels. Therefore, you can increase the force of contraction by increasing the number of action potentials per second that travel down the nerve (increasing the frequency). The attached figure shows the result of increasing action potential frequency producing a step-wise summation of individual twitch contractions. As action potential frequency increases, so does force generation. At high frequencies, this will produce a maximal tetanic contraction as shown by the plateau in the muscle tension.

Changes in the neuromuscular junction, the sensitivity to EPSPs, amount of ACh (neural transmitter to create an action potential at the muscle site)and ACh esterase (the enzyme which allows for the reabsorption of choline and production of new ACh).