Powers of Stress

by Richard Utt

When a muscle is monitored and observed to be in any of the states of stress other than homeo-sta-stress, it is impossible to immediately determine exactly how far the muscle is away from the homeo-sta-stress condition. It is possible that a single correction - perhaps a spindle organ squeeze technique - will return the muscle to homeo-sta-stress. When a muscle is returned to homeo-sta-stress by a single appropriate spindle organ technique, then the muscle was at the threshhold stress level or, in other words, exhibiting 1 power of stress. The other possibility is that the muscle will require a number - 2 or 20 or 200 - of spindle organ stimulations (or other modality stimulations) to return the muscle to the homeo-sta-stress state. Each of these manipulations takes the muscle one power of stress closer to homeo-sta-stress. These powers of stress represent degrees of muscular chemo-electro-magneto polarity that must be reduced in amplitude bit by bit before the muscle can be returned to homeo-sta-stress. Long before a tissue or organ demonstrates anatomical or physiological changes that are obvious, it will manifest powers of stress that an Applied Physiology (AP) practitioner can observe and work on.

In a real sense, the complex and coordinated tissues and cells of the body - including neurons, synapses where neurons meet, and motor end plates where neurons meet muscles - can be compared to spark plugs. The internal combustion engine in our prize automobile will not run at peak performance if all the spark plugs in all its cylinders are not firing correctly. In order for an electrical charge to travel across the poles of a spark plug, the poles must be appropriately spaced, or "gapped". If the gap is too wide, no spark will jump across it and the spark plug won't fire at all.

How does this relate to muscle function? It can be compared to the condition when a muscle is underfacilitated or overinhibited. When we ask the muscle to respond to commands from our central nervous system, it is unable to contract appropriately and play its part in the "team" of muscles that work together to create graceful, purposeful movement. At its extreme, this condition can be observed as flaccid paralysis, when the muscle does not respond at all!

If, on the other hand, the spark plug gap is too narrow, the jumping spark will be uncontrolled, and the plug will overheat. This is often compared to the states of stress called overfacilitation or underinhibition, where the muscle being observed is unable to relax appropriately during extension - this may result in pain and certainly results in movements that are less graceful and efficient than possible. At its extreme, the muscle may be unable to relax at all, a condition called spastic paralysis.

Even though this analogy to a spark plug, its "gap" width, and its optimal function may seem to be comparing apples and oranges, there are many interesting parallels with living tissues. Within the motor, sensory, and autonomic nervous circuits of the body are motor end plates and synapses that represent real "gap" sites in these nervous circuits. The width of the "gap" between a neuron and an extrafusal muscle fiber, for example, must be within a certain width tolerance (homeo-sta-stress) or the nervous impulse will either be very weak or will not "jump" across the gap at all. The muscular circuit connecting an agonist, the spinal cord, and antagonists will not function optimally because of this constriction or dilation (see the Chapter on the Histology of Stress) of the cells at the neuromuscular junction. When an Applied Physiologist applies the appropriate interventions to the system, he or she is reducing the powers of stress that have created distorted tissues, incorrect "gapping", and observable states of stress.

Sometimes the entire circuit, including the afferent and efferent nervous cells that carry information from the big toe to the brain and back, is compared to the action of a transistor. In a transistor, a small voltage change at one place in the circuit (the sensor or dimmer switch) is used to control or regulate much larger voltages elsewhere in the circuit. In this model, the "dimmer switch" represents the neuromuscular spindle organ (NMS) in a muscle which sends sensory information to the central nervous system (brain and spinal cord). This sensory information informs the brain about the overall tone, action of contraction, and action of relaxation in the muscle itself. The circuit is completed as the central nervous system sends motor impulses back to the muscle which cause it to contract (facilitation) or relax (inhibition).

In this "transistor circuit model" the output voltage (which generates the nerve cell "voltage" that stimulates muscle actions) is adjusted by changing the input voltage (at the NMS). Let's consider the agonist muscle first. If the input voltage is too low (+0.1 mV to +0.38 mV) then the output voltage will also be too low (+1 mV to +38 mV), and the agonist muscle will be unlocked and underfacilitated (see Figure 2 on pg. 34). The muscle is capable of movement but not a solid lock. In that critical moment when we call upon that particular muscle to support us or lift a fragile object, it fails to give us adequate stability.

On the other hand, if the input voltage is too high (+0.6 mV to +1 mV), then the output voltage will also be too high (+60 mV to +100 mV) and the agonist muscle will be overfacilitated (see Figure 3) and it is capable of movement but is tight and rigid, like a weight lifter who is "muscle-bound". While we may not notice this rigidity during large muscle movements - perhaps driving our car - we certainly experience limitations as we try to carefully adjust the tiny screws in our sunglasses.

In the homeo-sta-stress state, the range of input voltages (+0.39 mV to +0.59 mV) creates a range of output voltages (+39 mV to +59 mV) which allow the agonist muscle to contract (locking firmly into positions 1-7) optimally and appropriately so that the muscle accomplishes tasks accurately and gracefully (see Figure 4).

In this model, the agonist muscle exhibits spastic muscle paralysis when an output voltage of +101 mV or more exceeds the range of values we associate with overfacilitation . Flaccid muscle paralysis is observed when the output voltage is 0 mV (see Figure 5).

Remember that an agonist muscle always works as part of a muscle "team" which includes at least one antagonist muscle. In this "transistor" model, hypothetical positive millivoltages are used to represent the chemo-electro-magneto events in the agonist. In order to expand the model to include antagonist activities, we use negative

values for the millivoltages (see Figure 1 for a summary of these circuits). All these voltage values (positive or negative) are hypothetical and are only used to represent the different and varied chemo-electro-magneto events in the neuromuscular circuitry of this model.

Let's consider the antagonist muscle now. If the input voltage is too high (-0.1 mV to -0.38 mV) then the output voltage will also be too high (-1 mV to -38 mV), and the agonist muscle will be weak and overinhibited (see Figure 6). The muscle is capable of movement but not a solid lock.

On the other hand, if the input voltage is too low (-0.6 mV to -1 mV), then the output voltage will also be too low (-60 mV to -100 mV) and the agonist muscle will be underinhibited (see Figure 7 on pg. 35) - it is capable of movement but is rigid and will not unlock. Once again, we may not notice this rigidity during large muscle movements but fine, accurate movements are difficult.

In the homeo-sta-stress state, the range of input voltages (-0.39 mV to -0.59 mV) creates a range of output voltages (-39 mV to -59 mV) which allow the antagonist muscle to relax (unlock) optimally and appropriately so that the muscle team accomplishes its tasks accurately and gracefully (see Figure 8). In this model, the antagonist muscle exhibits spastic muscle paralysis when its output voltage of –101 mV or more exceeds the range of values we associate with underinhibition (-101 mV or more). Flaccid muscle paralysis is observed when the output voltage is 0 mV or less (see Figure 9). A summary of this model is shown in Figure 10.

If the output voltage remains consistently too high, then this creates a situation which can be compared to a circuit in which there is too much current flow and a fuse (or other electronic device, such as a transistor) burns out. In human tissues, this "overheating" may take place over short time periods (perhaps one second) or long periods (maybe 20 years) and culminate in the "burning out" of an organ that will need to be replaced in a heart or kidney transplant! It is important to remember that there are a range of values which represent homeo-sta-stress (39 - 59 mV in our example). When the voltages are regulated within optimal range, the circuit behaves according to design. It is also important to remember that there are a range of values associated with either overfacilitation or underfacilitation - the individual voltage values (+61, +62, +63, etc) can be compared to the powers of stress exhibited in overfacilitation . If the model voltage value (perhaps +63 mV) is only a few mV (or powers of stress) away from the homeo-sta-stress range, then the muscle circuit can be restored to homeo-sta-stress with a small number of NMS manipulations. If, on the other hand, the model voltage value is many mV away from the homeo-sta-stress value (perhaps +93 mV!), then a much larger number of NMS manipulations are required. In an extreme example, when many hundreds or thousands of powers of stress are affecting muscle function, spastic paralysis may occur.

Changes which occur in the "voltage" of this anatomical and physiological circuit may have several different causes. They may be due to chemical events, including our individual reactions to the many different foods - sweets and meats and vegetables - we eat or the alcoholic or caffeinated drinks we consume. Environmental toxins may be inhaled as we wait for the bus on a smoggy intersection, or ingested along with our polluted tap water. Mechanical events, such as sports injuries or strains related to over-exercise or under-exercise may create powers of stress in these body circuits. Finally, our complex mental processes - all of our thoughts, attitudes, and feelings - including happiness, sadness, fright, grief, or anger can contribute to the voltage in these neuromuscular circuits and create powers of stress.

The result in both of these cases is that tissues become distorted and the polarities which create neuromuscular homeo-sta-stress are disturbed. As the powers of stress increase, the amplitude of this tissue distortion and polarity disturbance increases. Individual cells are unable to do their "job" in the cellular community as they have problems receiving nutrients and ventilating wastes through constricted or dilated blood vessels. Neurons cannot transmit impulses effectively along their axons and dendrites. Neurotransmitters flood across overly narrow synaptic gaps or are lost and diluted in overly wide gaps. The nervous messages which coordinate and create appropriate muscle actions are not transmitted accurately. The AP practitioner observes these departures from homeo-sta-stress and understands the appropriate methods using body hardware to create changes in the polarity and chemo-electrical condition at the cellular level which restores the entire muscle to homeo-sta-stress.

In AP we quantify the Powers of Stress by understanding that each Fac/Inh balancing technique we apply is counted as one power of stress. It is important to be aware that when we begin to build a muscle's chemo-electrical condition toward the homeo-sta-stress state that we do not know how many powers of stress it is manifesting. The AP practitioner understands that he or she must be persistent in applying appropriate spindle organ techniques or other interventions until a sufficient number of powers of stress are removed to enable the muscle to accept the final spindle organ technique applied. It may be necessary to be extremely patient and persistent as we manipulate spindle organs multiple times, store the information from these manipulations in Pause Lock, and slowly adjust the chemo-electrical condition of the the muscle towards the homeo-sta-stress state. Ultimately, our repeated use of spindle organ technology will allow the muscle to accept this information and return to the homeo-sta-stress state and stored in Pause Lock. The P/L is now holding the gravity of the powers of stress and we have successfully created a hardware circuit that may be worked on by using an appropriate indicator muscle. The modality of correction is only limited to the expertise of the practitioner at this point. Whatever modality is used, the subsequent release of endorphins and enkephalins by nervous tissues in the brain will affect the motor and sensory components of the multiple nerve circuit that were created in the original P/L, including its powers of stress (Amplitude of Circuit). After all corrections have been made, close the Pause Lock and recheck the original OF/UI/UF/OI circuit observed.