Chapter 3

Treatment of Pathogenic Neuromuscular Dysfunction Utilizing Electromyometric Feedback

All pathologic neuromuscular disorders have two common characteristics: (1) they occur as a direct result of ***damage to nervous tissue*** (supraspinal, spinal cord or peripheral nerve); and (2) the damage results in a ***disruption of a feedback loop*** or communication path between supraspinal structures (brain) or between supraspinal structures and the organs associated with them. These include four separate disorders: (1) the ***post cerebral vascular accident (CVA) syndrome***, (2) the ***peripheral nerve injury syndrome***, (3) the ***spinal cord injury syndrome*** and (4) ***cerebral palsy***. These conditions have common elements that may allow for a similarity in their rehabilitation, but each has a different etiology and differences that deserve exploration and discussion.

THE POST CEREBRAL VASCULAR ACCIDENT SYNDPOME

The rupturing of a blood vessel in the brain commonly causes disruption of the supraspinal feedback loops. The most common etiology of this ***cerebral vascular accident (CVA)*** is a weakened ***cerebral arterial wall*** that balloons out forming an aneurysm as blood pressure exerts its outward push against the vessel wall. Increases in blood pressure cause the vessel wall to be abnormally stretched, and thus increasingly thinner. Eventually the elastic limits and tensile strength of the vessel wall are exceeded and it bursts. As the blood streams out of the rupture, it ***destroys whatever neural tissue it touches*** until blood clotting occurs. A similar phenomenon occurs when a cerebral vessel becomes brittle because of hardening of the arteries (***atherosclerosis*** or ***arteriosclerosis***) and fractures under pressure. Blood spurts through the fracture causing damage similar to that produced by a ruptured aneurysm.

CVA's may occur from ***trauma to the cranium***. Neural damage may result when the brain excessively reverberates within the skull, rebounding from the force of a blow and is bruised against the opposite skull wall. Damage may also occur if the skull collapses inwardly when struck and the brain is directly bruised or lacerated.

A ***CVA*** may occur as a result of necrosis of cerebral tissue caused by a ***failure of blood supply or oxygen deprivation***. A thrombus or external pressure can block arterial blood supply depriving supraspinal structures of oxygen for a lengthy period. The extent of neural damage resulting from vascular blockage will depend upon the size of the vessel blocked and the area it supplies. A blocked arteriole will result, for example, in far less damage than if the carotid artery is blocked. Damage can also occur if capillaries supplying neural tissue are caused to constrict or close sufficiently.

Cerebral damage may occur from ***the trauma of surgery***. The damage may occur directly from neural incision (tumor excision, aneurysm repair or other surgical procedures) and the resultant ***bleeding*** or indirectly from ***blood clotting***, excessive

anesthesia, or ***anoxia*** from cardiac or respiratory failure. ***Chemical poisoning*** of various types may also result in enough neural damage to cause a post CVA syndrome (the detailed mechanism for chemically induced damage is beyond the scope of this discussion).

Regardless of the cause of damage, if neural tissue is injured beyond the body's ability to repair it, a ***post cerebral vascular accident (CVA) syndrome*** will result and may include motor impairment in the form of ***hemiplegia, quadriplegia, hemiparesis*** or ***quadriparesis***. The severity and extent of involvement depends on the amount of damage and individual response. With so many possible sources of cerebral damage, it is not surprising that the post CVA population is large and suffers from a diversity of symptomology.

Classification of Post Cerebral Vascular Conditions

Most commonly, in the post CVA syndrome, the cerebral motor cortex (***Computer B***) is unable, because of intrinsic damage and damage to the internal capsule, to receive ***afferent feedback*** from lower centers including the basal ganglia (***Computer C***) and the cerebellum (***Computer D)***. Essentially, ***Computer B*** is ***deprived of all knowledge of muscle status*** and is faced with a situation analogous to an individual trying to use a telephone with a broken receiver. The individual can talk into the mouthpiece or transmitter, but does not know if the message is getting through because no perceptible reply is forthcoming. The person can choose to keep talking into the transmitter hoping to be heard, or simply hang up the phone. In a very real sense, ***Computer B*** of the post CVA syndrome patient must make the same decision, choosing to send ***efferent messages*** to the musculature (directly to extrafusal muscle via the corticospinal tract or through ***Computer C***), hoping they get to the appropriate muscles, or "to hang up" and to not send any messages.

If ***Computer B*** keeps sending messages to the musculature, the messages are purely ***facilitory***, lacking both ***coordination and inhibition*** provided by ***Computer D***. ***Computer B*** messages are imposed upon the general ***efferent motor activity*** that has been biased by influences from certain uninhibited structures (***red nucleus***, ***vestibular nucleus***, etc.) to produce ***synergistic*** and ***developmental reflex patterns*** (see **Tables 1, 2** and **5**). Any facilitory action by ***Computer B*** either triggers these reflex patterns or reinforces them. The result is called a ***spastic post CVA syndrome***. If ***Computer B*** does not send efferent messages, the involved muscles will appear flaccid (without tone) and the resultant condition is called a ***functionally flaccid post CVA syndrome***.

If the patient has suffered damage of the ***afferent pathways to Computer B*** and ***Computer C*** as well as damage to the ***efferent pathway from Computer B*** to ***Computer C***, the resulting flaccidity will be of a more serious nature than seen in the ***functionally flaccid syndrome*** and is called a ***structurally flaccid post CVA syndrome*** (see **Figure 11**). ***Computer C*** responds to a total lack of input (from ***Computers B*** and ***D***) by turning off and ***shutting down*** facilitory impulse transmission to ***tonic gamma motor neurons***, resulting in an ***inability to generate muscle tone***. The potential for phasic extrafusal

muscle contraction may still exist if the direct efferent pathways from ***Computer B*** to the extrafusal muscle are intact (via the ***corticospinal tract***).

The ***post CVA syndrome*** is usually seen as a "paralysis" on one side of the body, ***contralateral to the side of the brain*** that has been injured and usually both the upper and lower extremities on the involved side are affected. This condition is commonly termed a ***hemiplegia***, or ***hemiparesis***. If the ***CVA*** occurs in the right hemisphere of the brain, the left side of the body will be affected and is called left hemiplegia. Conversely, if the ***CVA*** occurs in the left hemisphere of the brain, the resulting condition is called right hemiplegia. The debilitating effects of a right or left hemiplegia vary among patients and depend on the extent of the neural damage sustained and the hand dominance of each patient. If the patient was right handed, for example, and the left-sided musculature was affected, she would be far less handicapped than if the hemiplegia was on the right side. Likewise, the patient would be far less handicapped if the damage was only enough to affect finger coordination and not enough to involve the entire left side musculature in classical ***developmental*** or ***synergistic reflex patterns***.

Spastic Hemiplegia

The ***supraspinal structural spastic response*** to a ***CVA*** appears much like a ***regression to an early stage of neuromuscular development***. It is as if all previous neuromuscular learning was erased from the involved hemisphere. The patient must begin anew the struggle through the developmental reflex stages, without the aid of ***"muscle sense"*** (***proprioceptive feedback from the muscle spindle***).

One of the initial steps in rehabilitation is to establish which ***developmental stage*** the supraspinal structures have regressed to by identifying which of the ***developmental reflexes*** is dominant. The developmental stage is related to the most primitive reflexes that can be demonstrated by the patient (refer to **Table 1**). If, for example, the patient demonstrates any of the ***first stage developmental reflexes***, she is said to be at the first developmental stage, even if reflexes from other stages are also present. Simultaneous exhibition of reflexes from different stages is not uncommon in normally developing infants, or in CVA patients. The developmental reflex stages for infants, however, are defined in terms of when the developmental reflexes making up that stage disappear, not by which developmental reflexes may be concurrently present (see **Tables 1** and **2**).

In the ***spastic hemiplegic***, the reflexes most typically displayed are the ***tonic labyrinthine prone reflex***, ***asymmetrical tonic neck reflex***, and the ***positive supporting reaction*** (from the ***second stage***), the ***symmetrical tonic neck reflex (I)*** (from the ***fourth stage***) and the ***contralateral*** and ***homolateral associative reactions*** (from the ***fifth stage***). The patient, in this case, is said to have regressed to the ***second developmental stage***. Initial treatment efforts are directed at ***inhibition*** of the ***tonic labyrinthine prone***, ***asymmetrical tonic neck reflexes*** and the ***positive supporting reaction***, and ***facilitation*** of the ***tonic labyrinthine supine reflex*** and the ***negative supporting reactions***. When the former have been inhibited, and the latter facilitated and subsequently inhibited, the next step is to ***facilitate*** the ***tonic lumbar reflex*** of the third stage.

Rehabilitation of the post CVA syndrome would be a simple process of progressive facilitation and inhibition of the developmental reflexes through the five stages of development (as described in **Table 2**) if recovery were not complicated by the fact that the post CVA syndrome patient is not a new born baby and is subject to the continued influences of ***Computer D*** (the cerebellum and the vestibular nuclei). A post CVA patient is unable to coordinate activity between higher centers (***Computer B*** and ***Computer C***). ***Computer B*** can still affect descending gamma motor (***tonic*** and ***phasic***) activity through the common juncture in the ***red nucleus*** (substation complex) (see **Figure 9**). This provides ***Computer D*** not only with the opportunity to affect ***intrafusal muscle activity*** but also, indirectly, to affect ***extrafusal muscle activity***. However, with the loss of coordinating ability (through the loss of necessary pathways), ***Computer D*** continues sending ***pre-CVA learned programs*** to a system no longer able to use them. The nature of the cerebellar programs is inferred from observing the inhibition of gamma motor activity affecting these muscles antagonistic to the synergies toward which the damaged system is biased. Most commonly, the ***flexion synergy*** affecting the upper extremity and the ***extension synergy*** affecting the lower extremity (see **Table 5**) appear in the ***spastic post CVA syndrome***, but the extension synergy affecting the upper extremity and the flexion synergy affecting the lower extremity have also been observed. Exotic combinations of these synergies have been seen, usually in post head trauma patients and in progressing ***functionally flaccid post CVA syndromes*** indicating that certain supraspinal structures may be facilitated while the cerebellum inhibits the action of supraspinal structures that might be inhibitory to them. Brodal (1974) suggests that the ***red nucleus*** is strongly biased toward the ***facilitation*** of gamma motor activity to the flexors of the upper extremities and the extensors of the lower extremities, while the ***vestibular nucleus*** is biased toward gamma motor activity to extensors of the upper extremities and the flexors of the lower extremities (quite a coincidence). This response to cerebellar influence on supraspinal efferent activity is seen when the patient attempts to use the involved extremity and interferes with the ***trial and error*** period necessary for relearning or ***reprogramming*** motor coordination and control.

The synergies not only appear as increased myoelectric activity from ***"facilitated"*** muscles and as overt patterns of motion, but also as spasticity in the ***"synergistic"*** muscles. The spasticity is seen as ***clonic-type motion*** and ***cogwheel rigidity***, or ***hypertonus*** or ***rhythmical deflections*** or wavering as seen in electromyometric visual and auditory feedback.

To teach the patient how to overcome dominant synergistic patterns, the therapist aims not only to balance antagonistic muscles in direct opposition across an involved joint, but also to balance muscles distant from one another in ***opposing synergistic patterns*** (hip flexors versus posterior tibialis, or hamstrings versus biceps brachii and brachialis, for example). In other words, a muscle belonging to the "inhibited" synergy is facilitated while muscles of the "facilitated" or overactive synergy are inhibited. For example, if the upper extremity flexor and the lower extremity extensor synergies are dominant, then the hip flexor could be ***facilitated*** while the quadriceps group, anterior tibialis, posterior tibialis, short toe flexors, biceps group, wrist flexors, finger flexors, long thumb flexors, or short thumb flexor are ***inhibited***, either individually or in combination.

Also helpful in breaking dominant synergies is the ***learned over-control*** of myoelectric muscle activity in the ***balance*** phase. The "spastic" muscle should be inhibited below the minimum level of myoelectric activity that would produce a visible contraction while its antagonist is facilitated to produce an increase in myoelectric activity.[1] This ***over-control*** is effective in inhibiting cerebellar influence and allowing the ***trial and error*** period to occur. Since both the ***synergistic*** and ***developmental reflex patterns*** appear concurrently in ***spastic*** post CVA patients, both must be controlled simultaneously. This dual control is learned as the patient is taught ***inhibition of a developmental reflex***. It can be more effectively dealt with, however, if in the last phase, before continuing on to the next developmental reflex, the patient is asked to myoelectrically inhibit the muscles of the ***dominant synergy*** (two of the muscles making up the synergy are collectively monitored with one EMM utilizing a split-electrode set-up) as opposing muscles are facilitated (in a ***balance*** phase) instead of simply contracting and relaxing the involved muscles on command (see **Table 2**).

When the ***fourth*** and ***fifth developmental reflex stages*** have been reached, weak musculature should be facilitated as components of the ***contralateral*** and ***homolateral associative reactions***, and these reactions should then be inhibited as a ***balancing*** activity (see **Table 3** and **4** for descriptions of direct prime mover agonist/antagonist relationships) between muscles which are facilitory of one another. There will be variations in the program depending on the variations in dominating reflexes seen in the initial evaluation. The spastic post CVA conditions resulting from head trauma, multiple CVA's and developing functionally ***flaccid*** hemiplegic conditions are most likely to demonstrate the greatest variety of reflexes and are the most likely to display reflexes from the ***first stage developmental level***.

As the patient is able successfully to perform ***electromyometric balancing*** at high enough levels to produce joint motion, the patient should be allowed to perform these motions in a free state (without a set or fixed positioning), first with the use of electromyometers and then without. For example, when wrist extension balancing has been achieved, with the wrist flexors producing less than three or four-microvolts (mv) and the extensors producing more than 15-mv, wrist extension motion should be possible. The wrist should then be placed in neutral position and the patient should attempt hyperextension of the wrist using the EMM's as a guide. When the patient can perform this action successfully with feedback, the machines should be turned off and the patient asked to perform the action independently. Many trials may be required, with or without EMM's, but eventually the patient should be able to produce ***free motion without resistance*** or aid. The next step is to ask the patient to perform free motion as a part of a ***functional activity***, developing more muscle control (as in grasping and turning a doorknob).

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1 The minimum level of myoelectric activity that will produce a visible contraction in a spastic muscle has been shown to be three or four- mv in muscles located below the elbow and six-mv across any other joint. These values are for the Toomim EMM's.

When a patient has developed ***voluntary gross muscle control***, it should be used ***functionally*** as soon as possible. Repetitive or prolonged shoulder elevation (to help in the reduction of shoulder dislocation) and walking are examples of ***gross muscle functional activities***. When the patient begins to produce tone in the muscles of gross motion or proximal joint stabilization, she should be encouraged to use them in such a way as to increase overall function and promote further development. Shoulder elevation helps facilitate the stabilizing muscles around the shoulder and helps prepare for activity in all the joints of the upper extremity. Ambulation should be encouraged when the quadriceps muscles begin to show enough activity to stabilize the knee. The patient should ***not*** be allowed to hyperextend the knee to provide for stability, contrary to common practice, but instead should be asked to ***bend the knee while stepping on the involved side***. Knee flexion while weight bearing not only encourages quadriceps toning by taking advantage of the stretch reflex in the developing muscle group, it also helps to facilitate musculature all around the knee because of the ***facilitory effect of joint compression***. It also provides the patient with an opportunity to use the musculature of the lower extremity ***without*** reinforcing the ***positive supporting reaction***. The only impediments to walking with knee flexed are ***overstretched or unstable knees and ankles*** (from poor initial gait training or manual joint stretching after injury),

In the upper extremity, functional activity should also follow the balancing of muscles. For example, elbow flexion combined with wrist and finger extension, with thumb abduction and extension will put the hand in position to grasp a doorknob. Once able to assume such a position, flexion can be utilized to grasp the knob while pronation or supination of the wrist turns it. This fine exercise uses many muscles in combination and produces a functional result. Another useful coordination-building exercise is the picking up of a utensil (fork, spoon or knife) placed across the mouth of a cup, between the thumb and index finger, and then returning it. This exercise not only requires multi-muscle cooperation but also ***eye-hand coordination*** and ***timing***. Such an exercise goes far beyond the expertise required to simply squeeze and release, taking the patient closer to the practical functioning of the upper extremity.

Exercises that require multi-muscle coordination may do much more than allow the patient gross or even refined motor function. It may be the element that allows the patient finally to make the complete transition from needing ***"muscle sense"*** to being able to accept other sensory clues of function (or something else) to operate the involved musculature. The common assumption has been that sight was the sense that had to be utilized as a substitute for the missing "muscle sense." Clinical observation has indicated that other senses may be used instead of or in addition to vision. An illustration that supports this contention was the motor behavior developed by a woman who had suffered a CVA resulting in a right ***spastic hemiplegic syndrome*** two years before electromyometric therapy began. Initially her right elbow was chronically flexed with a voluntary range of 30^{o} with her wrist fixed in flexion and her fingers flexed around her flexed and adducted thumb. After a year and a half of electromyometric neuromuscular reeducation, she was able to reach out, grasp an object with the involved hand, lift it and drop it again. At about this time, she was observed performing another remarkable feat. She was standing in her kitchen, grasping a small bag in her involved right hand with the

elbow flexed to 90°, the shoulder slightly flexed and abducted and her wrist in neutral. She turned her head so that her line of vision could follow her uninvolved hand. Her uninvolved arm was abducted to slightly more than 60° and her elbow slightly flexed to 5°. She grasped a kitchen pot with the uninvolved hand, picked it up and brought it back and put it in the bag. The total act was outstanding for a woman with her medical history, but the most phenomenal thing was that she did not drop the bag in her involved hand when she took her eyes off of it and turned her head. She was apparently using other sensory clues besides these provided by vision to help maintain her grasp (joint proprioception, light and deep touch, or perhaps ***skin stretch***). This observation becomes important in light of previous experience in similar circumstances where patients were observed dropping similar objects from their involved hands when they changed visual fields. Research into this area is needed and will be necessary to establish which particular mechanisms are involved in such accomplishments.

The patient should perform these activities that are appropriate to the developmental level of the involved side (see **Table 1**). For instance, when the patient begins to facilitate the ***tonic lumbar reflex***, marking the ***second stage*** of reflex development, the patient should begin attempts at rolling (toward the hemiplegic side). When she reaches the ***fourth stage***, crawling and creeping should be attempted. The accomplishment of these tasks will help achieve mastery of that stage. In the aphasic patient, the performance of the developmentally associated functions may become of paramount importance, since failure to perform these may add to a block in ***cognition and speech development***. Although it may be obvious (to some) that failure to perform the appropriate developmentally-associated functions at the ***first developmental stage*** might impede development of articular control of jaw and tongue for the forming of words, these functions appropriate to higher developmental levels may be just as important for ***cognition, speech development***, and even ***emotional control***. The link between functional neuromotor development and the development of other neuro-functions must be explored further, beyond the limits of conjecture based on observations, before more definitive statements can be made.

It should be mentioned that tactile feedback is the most useful of the feedback modes in the treatment of the right hemiplegic patient. Tactile feedback helps to establish a graphic communication link between muscle response and the patient in a more effective way than auditory or visual feedback (Schandler and Gringe, 1945). The electrodes providing the tactile stimulation are placed over a skin area that retains ***light touch sensory perception***. The selection of the area of application should be made carefully so that the patient will accept the stimulation appropriately. That is, if facilitation of the muscle is desired, the tactile feedback electrodes should be placed on a ***"comfortable"*** skin area (the upper back area, for example) to help reinforce the muscle contraction. If inhibition is desired then the stimulating electrodes should be placed on an ***"irritating"*** area (the ventral surface of the forearm or over the gluteus maximus) to discourage contraction. Since the patients describe the tactile feedback sensation on an irritating area as "prickling" or "needle-like", one can see how this can be used to produce an aversion to undesirable muscle contraction. On the other hand, patients describe the stimulation placed in a ***"comfortable"*** area as being "a moving or rippling sensation", which lends

itself well to reinforcement of desired contractions. The improvement in communication is obvious since it is nonverbal and taps into the basic inclination to avoid discomfort and to facilitate activity that increases pleasurable sensation. Tactile feedback aids in communicating muscle function as it provides a graphic demonstration of desired muscle function.

Flaccid Hemiplegia

The ***functionally flaccid*** response to a ***post CVA syndrome*** is a more primitive response to the disruption of supraspinal feedback loops than the ***spastic*** response. If the ***spastic*** response can be said to be a "regression" on the part of involved supraspinal structures to a neonatal ***developmental reflex state*** then the ***functionally flaccid*** response can be said to represent a regression to a ***pre-developmental reflex state***. Consequently, the rehabilitation of this condition requires ***facilitation*** of all the developmental reflexes. This facilitation should be done, one stage at a time, following the normal ***child developmental reflex sequence***. ***Each of the reflexes of a given stage should be first facilitated and then inhibited before proceeding to the next developmental stage***. This process should proceed from the ***first*** through the ***fifth developmental stages*** (as outlined for the rehabilitation of the ***spastic post CVA syndrome***).

In ***functionally flaccid hemiplegic patients***, not only facilitation of the prime movers of the developmental reflexes must take place, special attention must be paid to the natural necessity of proceeding ***proximally to distally***, facilitating shoulder and hip stabilizers before facilitating more distal muscles. All prime movers should be facilitated in order to create activity throughout the reflex pattern. For example, in the flexor withdrawal reflex, the hip flexors should first be facilitated, then the hamstrings, and then the ankle dorsiflexors. Likewise, when facilitating the ***tonic lumbar reflex*** in the upper extremity, the upper trapezius and supraspinatus muscles should be facilitated first, followed by the middle deltoid, biceps, wrist flexors, finger flexors, and thumb flexors, in that order (see **Table 3** and **4** for a list of prime movers and their related functions). Rehabilitation of the ***structurally flaccid*** post CVA patient should follow the same program as that for the ***functionally flaccid*** post CVA patient.

Quadriplegia

Quadriplegic post CVA syndromes are frequently seen as a result of either ***head trauma*** or of ***multiple CVA's***. ***This*** quadriplegia implies that damage has occurred to both cerebral hemispheres and has produced ***bilateral neuromotor dysfunction***. In such cases, damage on one side of the brain does not usually mirror the damage on the other in the extent of reflex activity exhibited and the total motor involvement. Such a condition should not be viewed as a singular quadriplegic condition, but as two hemiplegic conditions suffered by the same person. The two sides do relate to one another, however, since the ***developmental reflexes of one side affect the other***. For instance, it is not unusual for a patient suffering from a ***quadriplegic post CVA*** condition to demonstrate a ***positive supporting reaction*** on one side and a ***negative supporting reaction*** on the other (indicated by myoelectric activity, if not by observable overt patterned motion). When

such is the case, it is important to establish which ***developmental reflexes*** are dominant on the respective sides to determine their ***independent developmental stage levels***. Once the developmental stage has been determined for each side, a separate electromyometry feedback program should be designed for each. Since simultaneous ***reflex development*** on both sides may not be possible beyond the ***first*** and ***second developmental stage levels***, choices should be made about which reflexes should be worked with, and on which side, to produce the fastest functional improvements. For example, if a patient is dominated by the ***first*** and ***second stage reflexes*** on the left side (including the ***negative supporting reaction***) and demonstrates only the ***tonic labyrinthine prone reflex*** and the ***positive supporting reaction*** from the ***second stage*** on the right (he can support himself on that side during ambulation), the first treatment priority would be to inhibit the ***first stage reflexes*** and facilitate the ***labyrinthine supine*** on the left (preparatory to inhibition of the ***negative supporting reaction***). After the ***negative supporting reaction*** has been inhibited and tone in the quadriceps group muscles has been developed through the ***tonic labyrinthine supine reflex***, then work may begin on the reflexes on the right side. The programs should be designed along the lines suggested for the ***spastic CVA condition***, unless, of course, one side is flaccid (a remote possibility), in which case the treatment approach for that side would have to be based on the program suggested for ***flaccid hemiplegic conditions***.

Electromyometric Considerations

The link between the EMM and the patient is the electrode. It conveys the electrical impulses produced by muscle contraction to the EMM to be transduced into auditory, visual or tactile stimulation feedback. It is very important that the activity reflected by the EMM be representative of the specific muscle contracting. The attachment of the surface electrodes to the skin over the desired muscle should be specific and accurately performed. Placements should be made over the belly of the muscle and as far away from other muscles as possible. The therapist should try to avoid placing electrodes on a skin area that covers two muscles simultaneously.[2]

A difficulty inherent in the use of surface electrodes is skin resistance. Skin resistance is the capacity of epidermis to resist transmission of current either through or over its surface. If skin resistance is ***too high***, it will make electromyometric reception impossible. As a rule, normal skin resistance will not interfere with myoelectric pickup, and no special skin preparation is necessary. However, if the skin resistance is greater than normal, swabbing the area with alcohol or gently abrading the skin with a paper towel may be necessary. This procedure can be performed routinely before each electrode placement. However, in cases where the skin resistance is extremely high or the patient is hypersensitive to alcohol or to rubbing, a suede brush (a brush with fine metal or plastic tines) gently pressed into the skin's surface will suffice. This procedure

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2 *Illustrations for electrode placements are provided following* ***Table 7****. Please note that a pair of dots represent where the "pick up" electrodes should be placed. The "ground" electrode placement is left to the discretion of the therapist, but it is recommended that it be placed near the other two electrodes.*

makes very small punctures in the uppermost layers of the epidermis and breaks down its resistance. Any skin preparation should be performed in an area large enough to accommodate all three electrodes, or the preparation will be superfluous.

Once electrode placement is made, how the feedback is to be presented to the patient should be considered. If only one machine is being used, all modes of feedback available may be used simultaneously. These may include visual, auditory and tactile feedback. This situation will arise in ***post CVA syndrome rehabilitation*** when the patient is being guided through the ***facilitation*** and ***inhibition*** phases of the ***developmental reflex evolution***. However, when the patient enters the ***balance*** phase of training, two EMM's will be necessary to provide qualitative feedback from the muscles in opposition. To represent this balancing between muscles adequately, without confusing the patient, a judicious choice of feedback modes must be made. Various combinations of feedback modes are possible, but the ultimate choice of the appropriate feedback mode must be based on patient response and therapist insight.

Another consideration that must be made is where to put the machines. Machines should never be placed in a position to interfere with the patient's learning. For example, if the patient is being asked to inhibit the hamstring muscle group as he leans forward into the ***labyrinthine prone reflex*** position, the EMM should not be placed on a table at chest height on the involved side, requiring her to turn her head toward the involved side and look up to see the machine. Such a maneuver of the head and neck would be counter productive to the inhibition training since the ***asymmetric tonic neck reflex*** would be facilitated, causing reflex inhibition of the hamstring (see **Asymmetrical Tonic Neck Reflex**, **Table 2**). Instead, the machine should be placed on the floor in front of the patient to allow the patient to see the machine without hyperextension of the neck or rotation of the head, and to deal with the ***labyrinthine reflex*** without interference from any other reflex.

Thought should also be given to the arrangement of the two EMM's used in the ***balance*** phase, relative to each other. Resting, one EMM upon the other is convenient. In our setting the top machine consistently monitors the muscle to be ***facilitated*** and the bottom machine monitors the muscle to be ***inhibited***. This arrangement has helped the patient and the therapist avoid confusion (***especially the therapist***).

The EMM usually provides a meter for ***visual feedback*** (a few provide a digital readout) to allow the therapist an opportunity to record responses, with an assigned number value, and also to allow the patient to quantify visually his electrical responses. This provides both the patient and the therapist with a means of establishing ***quantitative*** and ***qualitative*** goals. A common quantitative short-term goal for a ***flaccid*** hemiplegic patient is to be able to facilitate muscle activity to a 15 to 20-mv level, which would be adequate for a ***trace*** (palpable) muscle contraction. A common qualitative short-term goal for a ***spastic*** hemiplegic patient would be 20-mv of activity from the triceps while keeping the biceps below a six-mv level. This goal would provide enough power to produce a ***trace*** muscle contraction in the triceps, and enough myoelectric inhibition of the biceps to allow a degree of visible elbow extension, linking a ***qualitative***

electromyometric goal with a quantitative functional goal. Such goal setting provides a motivational tool for the patient and progress documentation for the therapist. The idea of limiting the amount of myoelectric activity from the dominant ***prime mover*** muscles of the reflex patterns, in both the upper and lower extremities, to allow their antagonists an opportunity to create joint motion began early in the development of electromyometry as a rehabilitation tool. Through observation, it was established that the general limits for antagonist activity, sufficient to allow agonist joint motion, is six-mv in the elbow, shoulder, hip, knee and ankle, and four-mv or less below the elbow (although microvolt values for these control levels will vary from manufacturer to manufacturer and will have to be established relative to each instrument type). In ***reflex inhibition***, a reading of below two-mv (at low frequency) may be required to represent complete inhibition.

It should be pointed out that non-damaged people would, without practice, be hard pressed to provide the antagonist control levels we have set for our ***post CVA syndrome patients***. Indeed, much normal motion is in the form of eccentric contractions, with one muscle contracting and shortening as its antagonist decreases its tension and elongates, balancing its pull against the agonist as requirements of speed and control are met (see the **Muscle Spindle** and the **Central Nervous System**). In post CVA electromyometric feedback rehabilitation the patient is taught to over-control antagonist inhibition. This over-control is required to help in the inhibition of cerebellar (***Computer D***) interference, while trial and error takes place in agonist facilitation. Once this over-control is learned, the system adjusts to provide for the eccentric contractions seen in non-damaged people (a possible future study).

To promote not only the ***over-control*** of antagonist myoelectric activity but also the ***toning*** of the agonist muscle, a six-second attempt interval followed by a six-second relaxation interval is used, providing the patient with the best opportunity to reinforce immediately a ***successful attempt*** or to try again if the attempt fails. This method not only provides an opportunity to apply good learning theory, but also the best chance of toning the agonist muscle. To illustrate, the patient's wrist is fixed in hyperextension with the fingers in extension. After waiting for the patient to inhibit the antagonist activity to a satisfactory level (usually below four-mv), the therapist asks the patient to contract the finger extensors (as reflected by the EMM) while attempting to inhibit the finger flexors (below the four-mv level) for six-seconds, followed by a six-second rest. The task is repeated until the patient is able easily to produce the desired myoelectric responses. Not only is the correct muscle balance established for the patient on the unconscious level, but some toning of the extensors may also occur. Also physiological relengthening the flexor muscles and shortening of the extensors is produced since the exercise is an isometric contraction of the extensor muscles in a shortened condition, while the flexor muscles are being inhibited in a lengthened condition. This procedure helps reduce ***hyper-reflexia*** of the inhibited muscles.

Special Problems with Adaptive Devices

The adaptive devices or appliances that may pose problems in rehabilitation are the arm and forearm slings, short-leg braces, canes, walkers and wheelchairs.

Arm and forearm slings can increase the patient's disability by forcing the involved shoulder to ***sublux*** or ***dislocate***. The sling, by virtue of its construction, pulls the arm ***forward and down***, overtaxing ***ligaments and tendons*** that would otherwise hold the shoulder in place. In a flaccid CVA condition, unless electromyometry training starts shortly after onset, a subluxed or dislocated shoulder may be unavoidable because ***little or no muscle tone exists*** to relieve the strain on the ligaments produced by the weight of the arm. In the ***spastic CVA syndrome***, however, the sling may be the ***sole*** reason for shoulder subluxation. Its use decreases the hyper-reflexic response to stretch by the shoulder muscles, which could help retone these muscles (***after the central nervous system "shock" wears off***) during ambulation. When a sling is used, the shoulder muscles do not contract in response to the proprioceptive traction demand provided by gravitational pull that would maintain joint integrity. The muscles tend to progressively weaken, allowing the sling to pull the shoulder out of place. For a flaccid hemiplegic, the sling may be initially justified, but there is little evidence to support the contention that such application inhibits shoulder subluxation or dislocation. In the end, its only function may be to ***keep the arm out of the way***. Therapeutically, such an approach is as justified as dressing the patient, for expediency, who could dress herself. An unsupported shoulder, if given a little early electromyometric feedback encouragement, will develop muscle tone rapidly while encouraging elbow flexion and internal rotation to help get it out of the way. It is hypothesized that utilizing this approach from the onset of injury (after the patient is medically stable), the therapist would see far less or no shoulder subluxation (even in the flaccid hemiplegic condition), nor the ***"pampered extremity syndrome"*** discussed later in this chapter.

Bracing the involved lower extremity poses an even greater problem for rehabilitation of the post CVA patient. The ubiquitous ***short-leg brace*** inherently provides the patient with some ankle or knee stability during ambulation, conditioning the patient to rely upon it for security. The dependence may go on long after functional development has rendered it obsolete. This is unfortunate, especially since some types of short-leg braces are actually ***detrimental*** to the patient's overall well being. A good example is the ***Klenzak dorsiflexion-assist brace***, which is often issued routinely to both flaccid and spastic post CVA patients, without regard for the cause of ankle or knee instability. In the ***spastic lower extremity***, the dorsiflexion-assist spring action increases the tendency to ***toe-walk*** by reinforcing the synergistic hyperactivity of the posterior tibialis and gastrocnemius muscles by providing a ***quick stretch*** of these muscles, setting off the ***phasic stretch reflex*** during the ***swing phase*** of gait. The muscles soon overpower the spring, and while maintaining plantar flexion, ***force the knee into hyperextension*** during ***stance phase***. This gait pattern reinforces the ***positive supporting reaction***, discouraging natural development of muscle tone in the hamstrings, peroneals and long toe extensor muscles. Other braces that can cause problems are the simple wire-spring ankle brace (attached to the shoe) and the ***cosmetic shell brace*** (worn in the shoe). They may be helpful in providing lateral platform support and in preventing ankle inversion, but because of that support ***they discourage the development of the ankle everters***. They also do not prevent the knee from hyper extending in response to the ***positive supporting reaction pattern*** nor do they prevent ***knee-snap***. Consequently, it is recommended that these braces be used only if the patient can keep the knee ***slightly flexed*** during the stance

phase to avoid damaging knee ligaments. When the musculature supporting the ankle develops with electromyometry, the wire brace or cosmetic shell should be discarded. If the knee is slightly flexed during stance phase, the decreased ankle support will ***promote*** muscle activity in the ankle everters and, with care in foot placement, the patient will avoid ankle strain.

If the knee and ankle are totally "flaccid" and unstable, the recommended brace is the short-leg brace with ***double uprights and adjustable fixed-ankle joints*** (the ***Becker or Pope joints*** are examples) with a ***long-shank sole support***. If adjusted correctly, it will provide (by virtue of its mechanical advantage) adequate ankle and knee support while preventing the knee from hyper extending. Ambulation skills will progress as tone develops in the calf muscles.

Canes pose a problem for the patient by providing a ***false sense of security*** if used excessively. If the cane is used appropriately, it can do much to increase the stability and ambulation range of the patient by helping with balance (***by providing a broad base of support***) by forming a tripod between the lower extremities and the cane. Although this stance is more stable, both speed and mobility are sacrificed. The coordination of the cane with leg motion is accomplished at a rate limited by the ***speed of cane placement***. Bipedal walking is a habit that is practiced over many years and becomes automatic. It becomes so efficient that we can even run without conscious attention to the complex mechanics involved. This automatic quality is not usually available for the upper extremity as a functional component of ambulation. It requires both training and practice to learn to use the arm in ***tripodal ambulation***. Even after years of use, a modicum of attention must be paid to the coordination required. It decreases the speed of ambulation and consumes extra energy. Other liabilities include ***increased space*** required to ambulate with a cane and ***increased chances of collision*** (which are further increased if a ***quad-cane*** is used). The coordination and stability problems of stair walking and ambulating on broken or irregular terrain are also problems. ***Slippery surfaces*** are far more dangerous for a person using a cane because of the large amount of weight intermittently communicated to the ***small surface of the cane tip***. Should the cane slip, a fall is inevitable. Danger also lies in careless cane placement. A person who uses a cane unnecessarily may forget its presence and inadvertently place it in a hole or on a slick surface, causing a fall. People have even been known to trip over their own canes. The major problem caused by cane use, over a long period of time, is the development of the habit of tripodal balance, and it may be quite difficult to reorient the patient to a bipedal gait pattern with its inherent feeling of instability. It is a necessary step, however, in the developmental sequence, to achieve bipedal ambulation, even affecting some of the mental ***processes including speech and emotional control***. It is axiomatic then that when the musculature is available (barring other orthopedic or neurological problems) the patient should, with careful training and supervision, discard the cane in favor of independent ambulation. Walkers and quad canes can also be problems. They may foster much ***greater dependency*** problems than these produced by the cane because of ***their innate stability***. Very little balance ability is called upon, especially with the walker, since the ***broad base of independent support*** of four legs is provided. Because of their stability, walkers and quad-canes can support much of the patient's body weight,

and as such, they become enemies of not only the ***patient's balance*** but of ***muscle tone*** as well. They may also prove dangerous if all four legs of the cane are not securely on the ground or if the patient is too close to the front legs when weight is put on them. In such circumstances, the patient may be literally ***thrown to the floor without a chance of stabilizing herself***.

After discussing the dependency problems of brace, walker and cane use, it seems almost superfluous to discuss the problems connected with ***wheelchair*** use, since they are so blatant. In order to follow the developmental sequence, the patient ***must*** leave the sitting position. It is essential, not only for ***reflex development***, but for ***balance*** as well. Beyond these considerations, there is the possible development of a ***"dependent personality"*** arising from ***functional limitations***. Also muscular atrophy due to disuse associated with long-term use of the wheelchair may foster physiological problems including ***bacterial infections of the kidneys and bladder*** and ***circulatory problems in the legs and feet***. The patient may also develop problems of ***bowel "kinking" and blockage***. All these problems stem from not allowing gravity to have its proper affect on these organs. Normal human beings stand on their feet for respectable amounts of time, and the organs take advantage of the ***straight-line downward pull of gravity***, especially for circulation and elimination (why use the muscles, when gravity will do it). Gravity does not, however, work around the corners imposed upon vessels and tubes by continual sitting. To leave a patient in a wheelchair not only seriously limits functional and rehabilitation potentials but may also decrease survival potential.

Special Considerations

Rate of Learning

Observation has confirmed that ***children with post CVA syndromes*** learn at a faster rate than do the majority of post CVA patients (see **Neuromuscular Dysfunction in Cerebral Palsy Conditions**). They learn much faster than their counterpart adults because the ***cerebellum*** has not yet had the time or the demand put upon it to develop many functional motor programs. Children generally have not yet learned enough ***fine motor*** skills to have prior programming that causes interference to their rehabilitation. The speed at which they learn is often startling. Three or four training sessions may provide functional gains that would require months for an adult. An example of this extraordinary learning speed was seen in a seven-year-old right hemiplegic girl, injured by trauma to the head at the age of one month. When first seen at the age of seven, she demonstrated a gait pattern consisting of stepping forward with the involved leg and bringing the other up behind it, with her pelvis rotated to a 45^{o} angle to the line of motion. Her elbow was habitually flexed, as were her wrist and fingers, which were flexed around her thumb. She had some control of her right shoulder but could not fully flex it. In three sessions, despite a very short attention span, she was able to ambulate with a normal gait pattern with her pelvis at 90^{o} to the line of motion, and her step-through length was nearly normal, bilaterally. In a year, she was able to perform simple hand tasks with her involved handy including feeding herself with the involved hand, and she was able to roller skate. Her progress was amazingly rapid and gratifying, and

illustrates well the ***effects of cerebellar interference on learning rates by its notable absence***.

Time

Another special problem is the time that has elapsed between the CVA and when electromyometric feedback training begins. It becomes progressively more difficult to make functional gains if ***five years*** have gone by before the program begins. From a theoretical point of view, the time post-onset should make little difference in neuromuscular reeducation with the electromyometry, since the pathology is not progressive if no additional necrosis of neural tissue occurs, and if the rehabilitation depends on supplementing negative feedback loops. It does make a difference, however, and this difference lies in the formation of habituated patterns of accommodation that have been necessary for dealing with the ***activities of daily living***. In other words, new programming may develop to handle normal living tasks (like walking and eating). This programming takes the reflex response for granted, making it a part of the program. If ***accommodative programs*** are habitually reinforced over a lone period of time, they become almost impossible to deprogram. For example, a left spastic post CVA patient was first seen ten years post injury; he demonstrated the classical synergistic flexion pattern in the upper extremity. We were able to teach him to inhibit wrist and finger flexor muscles well enough so that he could open his hand to full finger extension with the wrist in a neutral position, and he could keep it there without it being fixed or taped. However, as soon as he stood up, his elbow, wrist and fingers all went into the ***upper extremity flexion synergistic pattern***. It was part of his habitual standing and ambulation patterns. The likelihood of this patient gaining a great deal of functional improvement was very remote. We have found this to be true with most patients who exceed five years post injury before electromyometric feedback begins, with an ***increasingly poor prognosis as tine progresses***.

Another element that may play a role in the poorer prognosis of those who are many years post-injury is at least partly neurological and has to do with the effect of a ***lack of sensory input on the function of some of the brain's structures***. In the absence of sensorimotor input from the peripheral structures, certain imbalances may be produced in supraspinal structure function that may interfere with the neuromotor relearning process, and these imbalances may become ***more pronounced as time goes by without the effects of negative feedback supplementation.***

Sensory Difficulties

One of the extra problems that a post CVA syndrome patient may have to confront is the ***loss of tactile sensation in the involved areas***. This sensory loss is not universally suffered and does not seem to be directly related to the type of post CVA syndrome suffered, or to the amount of neural damage sustained. ***Spastic*** post CVA syndrome patients may demonstrate a total tactile sensory loss in the involved areas while some ***flaccid*** patients show little tactile loss. An example of this is a patient who was first seen two years after her CVA. She had a right ***spastic*** hemiplegia with minor involvement in

the lower extremity, (she hyper extended her right knee when she walked) and a more involved upper extremity. The elbow and shoulder ranges of motion were severely limited. The wrist was flexed, and the fingers were flexed around the flexed thumb in a ***chronic flexor synergy***. The patient had no apparent difficulty with speech or cognition. ***She lacked all sensation in her upper extremity***, but ***did not*** demonstrate this sensory loss in her lower extremity. When she began therapy, she often displayed deep burns on the back or her right hand, sometimes up to an inch and a half long, sustained while cooking. She reported that she experienced no pain either during or after the acquisition of each burn. She also reported that she had slammed her involved hand in a car door, approximately a year before beginning therapy, fracturing the thumb's proximal phalangeal bone. The fracture was ***commuted*** and never set, leaving her with two bones instead of one. She claimed that she had never experienced any pain as a result of the accident. As therapy proceeded, with the help of electromyometric feedback, she gained more and more muscle control of the involved right upper extremity. She learned to open the right hand, reach out, grasp, lift and drop small objects. Eventually, she was able to toss small objects from the involved side to the uninvolved side. As she developed these skills, she reported that the sensations in her right upper extremity gradually returned, including ***light and deep touch***. She no longer burned the involved hand while cooking because she could ***"feel"*** the nearness of heat, and she began to complain about pain from the unset thumb when pressure was applied to the general area. It was as if the supraspinal structures decided to ***relate to or turn on*** the arm's sensations as it learned to use its musculature (***the arm became worth feeling because it could be used***). In other words, the higher supraspinal structures were deprived of muscle sense but could appreciate the advantage of having ***tactile sensation in a functioning extremity***.

Pain

Most pain problems in post CVA patients are related to joint abuse, muscle weakness or strain and sensory dysfunction. The most common of these is the pain associated with shoulder ***subluxation or dislocation*** resulting from overstretched ligaments and tendons. This condition is found in the majority of post CVA syndrome patients, flaccid or spastic. The best remedy for this condition is to teach the patient, through the use of electromyometric feedback, to contract the deltoid and shoulder external rotator muscles to draw the medial head of the humerus up. After the patient has learned to selectively contract these muscles, and has engaged in isometric exercise to further tone and shorten them, the pain will disappear, even before complete reduction of the dislocation or subluxation occurs. The isometric exercise involves having the patient elevate the shoulder and hold it up for a six-second count (while remembering to breathe) and then relax it for six-seconds.[3] The exercise should be repeated 20 times, four times a day.

Once the patient can hold the humerus in the glenoid fossa she is asked to do this while ambulating. Through this vehicle, the patient gets in the habit of maintaining tone in the

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3 *Special care should be used with patients with heart conditions in regard to the number of repetitions and attention to breathing. One must not over tire such a patient, and holding the breath may produce a Valsalva maneuver that could precipitate a heart attack.*

shoulder muscles, and eventually causes the muscles to shorten sufficiently to maintain joint reduction without conscious effort. The patient should also forego the use of any arm or forearm sling, and stop carrying the arm with the uninvolved extremity.

Having the involved arm continually in a sling or carrying it with the uninvolved hand produces another pain problem, the "***pampered extremity syndrome***", resulting from ***overprotection of the involved extremity***. Such ***"protection"*** seems to be an ***expression of rejection*** of the extremity and of the patient's situation. The patient may ***unconsciously*** wish to ***deny the reality of the disability***, and does so by protecting the ***"sick"*** arm and waiting for it to heal. The patient ignores the arm by avoiding any touch or motion that might draw attention to it. A forearm sling adds to this problem by wrapping the arm up, protecting it and ***making it easy to ignore***. Eventually this protection leads to an extremity so ***unaccustomed*** to sensory input from the environment that any stimulation of the skin or joints is perceived as ***traumatic and painful***, a further reinforcement that the arm is "sick." It is quite possible that a hemiplegic patient, especially with a ***flaccid*** condition, can inadvertently develop this condition simply by carrying the arm in the sling recommended by the rehabilitation staff. Whatever the mechanism of onset, the results remain the same and must be dealt with quickly and effectively to facilitate the patient's progress. One way of dealing with a hypersensitive extremity is to have the patient actively engage in whatever voluntary muscle activity is possible. She must once again focus on the involved extremity and must not be allowed to treat it as "sick." ***The patient must relearn to relate to the involved extremity*** (no longer ignoring it) to be able to learn to use it.

Another large problem that plagues many post CVA patients is the common ***trigger point syndrome***. This syndrome is one of the most misunderstood and ***unrecognized*** syndromes to confront the medical and physical therapy fields. It is one of the ***"big foolers"*** in physical medicine, and can look like many different pain syndromes (Taylor, 2002). It can look like sciatica, radiculitis, neuritis or neuralgia; various types of low back pain syndromes, arthralgia and the ***thalamic syndrome***. They usually include those muscles responsible for shoulder, neck, hip, knee and ankle stabilization (see **Table 3** and **4**). The supraspinatus, upper trapezius, scaleni, serratus anterior, pectoralis major, infraspinatus, subscapularis, gluteus minimus, hamstring, gastrocnemius and anterior tibialis muscle trigger point pain patterns are seen in combination as the great thalamic syndrome look-a-likes. Close scrutiny of the pain patterns involved and their overlap will do much to confirm the possibility of this deception. The referred pain problem develops from a weakening of the involved muscles during the first stages following the CVA when the patient experiences a false flaccidity of musculature as a result of the temporary suspension of all efferent nerve impulses by the involved cerebral motor cortex and basal ganglia (both to extrafusal and intrafusal muscle), as the brain reacts to the insult to its tissues. As the supraspinal structures begin to respond to sensory reflex demand (if they can), and as voluntary muscle activity becomes possible, the patient may tend to be ***overly active*** in an attempt to make the muscles work more and thereby ***strains*** of the weakened muscles occur. This same response may also occur in shoulder, neck and jaw muscles, which may produce headaches and trigeminal neuralgia-like ***burning pain*** patterns. The treatment for the trigger point syndromes not only includes encouraging the

patient to stop over-taxing the offending musculature by teaching them to facilitate and inhibit selectively muscle activity with electromyometric feedback, but also includes treating the trigger points. Treatment modalities may include electrical stimulation (surged faradic or pulsed-galvanic current), soft tissue manipulation (breaking associated adhesions), phonophoresis of an effective anti-inflammatory, and toning the involved muscles isometrically. Isometric exercise tones the involved muscles without causing further strain, and serves to remove one of the principle sources of the trigger point syndrome, ***weakness***. With this eclectic approach to treatment of the syndrome, complete relief from the symptomology may take only a few sessions. If isometric muscle toning is not possible, periodic modality treatment may be necessary.

Aphasia

Of the many effects of the post CVA syndrome, few are as disabling as the ***aphasia*** that may accompany a ***right hemiplegia***. The neural areas responsible for symbolic interpretation are located in the ***left cerebral hemisphere***, near the motor cortex. If the CVA damage involves these areas, not only will the person have difficulty speaking, but may also be ***unable*** to understand either the ***spoken or written word***. The effects on cognition and speech vary from one individual to the next and are dependent on the areas affected and the extent of the damage. This condition can occur in both the flaccid and spastic post CVA syndromes and may not reflect the extent of damage to supraspinal structures. It should be noted that ***aphasia might be directly related to the developmental level of the patient's involved side***. Clinical observation has led to a single conclusion that the ability to understand and produce symbolic communication (speaking and writing) is ***directly related to the individual's ability to inhibit activity in the wrist and finger flexors of the right upper extremity***. While it has been demonstrated that a right hemiplegic may be able to understand and speak (because the damage did not involve the communication centers), the aphasic right hemiplegic patient will not develop normal speech until she is able to learn inhibition of the right wrist and finger flexors.[4] What this means is that if both the cerebral motor cortex and the communication centers were damaged enough to produce both hemiplegia and aphasia, ***the development of cognition and speech will be markedly affected by neuromuscular reeducation***. This association is seen in the normally developing infant. As the infant begins to open the right hand, she begins to recognize objects and faces. As more sophisticated inhibition develops, she begins to experiment with sounds. When she stands and walks, she begins to imitate and initiate words. A similar relationship was demonstrated in individuals who were still aphasic two to five years post CVA. As the patients' command of right wrist and finger flexor inhibition developed through the use of electromyometry, the patients developed the ability to handle symbolic interchanges; first cognition evolved, then imitation of sounds, initiation of words and, finally, meaningful speech. The success of this approach did not and does not rule out the necessity for careful guidance in speech development. The ability to communicate is not an automatic achievement, and ***many verbal skills may have to be relearned***.

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4 *It should be noted that aphasia may result without right hemiplegia being present and therefore unrelated to right upper extremity spasticity.*

One example of this type of development occurred in a patient who was more than two years post CVA. He was a right hemiplegic with severe spastic involvement in both the upper and lower extremities. He demonstrated many of the ***second-stage developmental reflexes*** and had severe aphasia. On the first visit, he was only able to understand a few of the simplest commands and was able to verbally respond with only a ***few obscenities***. After the history was taken and gross motor and EMM evaluations were performed, a program of electromyometric feedback neuromuscular re-education was designed, and the patient was placed in the direct care of ***affiliating physical therapy students***, attached to our staff. The students' written and verbal reports allowed the supervising staff to follow the patient's progress (a full reevaluation was to take place later. Consequently, this author was not involved in direct observation of the patient's functional progress. Although the patient's progress appeared to be good, judging from the reports, one could not be prepared for what happened six weeks after the initial visit. The patient came in for his treatment, walked up to me and said, ***"You know, you're going to have to explain to me what we're doing."*** After recovering from the shock, I proceeded to my office to figure out what had happened. The students had been systematically and concertedly working on wrist and finger flexion inhibition and had noted that he had steadily been improving this inhibition. They had not mentioned a concurrent improvement in his communication ability. The students may have felt that such an improvement in cognition and speaking ability in an aphasic two-year post CVA spastic hemiplegic a natural and normal consequence of their ministrations. I can only say that although our knowledge and technical expertise has improved markedly since these early experiences, I would be hard pressed to find a better example of the parallel between ***neuromotor and communication skill development***.

Psychological Considerations

The effects of a CVA that result in a ***hemiplegic syndrome***, are far more extensive than simply the affects on neuromuscular function. They also include affects on the personality and mental processes. The hemiplegic patient not only suffers from a change in physical abilities but may also face a sense of disorientation and personal alienation as a result of the cultural response to a change in body image and physical abilities. This may result in the ***disintegration of many work and family roles*** necessary to maintain the person's self image and sense of reality. Personal relationships may have to be re-established or redefined to insure that the person's identity survives. Therefore, it may be necessary for the individual and her family to begin an intensive program of psychotherapy or family counseling to aid in the difficult task of readjustment and accommodation.

PERIPHERAL NERVE INJURY

The peripheral nerves of interest here include the ***efferent motor neurons*** and the ***afferent sensory neurons*** of voluntary muscle. The motor neuron arises from the ***ventral "gray" horn*** of the spinal cord and proceeds via the ventral nerve root to join the sensory neurons to make up the spinal nerve. The sensory neuron has its origin in the ***dorsal root ganglion*** and terminates in both the ***dorsal gray matter*** in the spinal cord and

in the ***muscle, skin and tendon***. The spinal nerve proceeds distally from the joining of the motor sensory elements, branching as it proceeds to innervate end organs in the various structures. These organs include sensory receptors in the skin, muscle spindles, Golgi tendon organs and motor end plates (see **Figure 17**). The sensory receptors in the skin include ***free nerve endings***, ***Meissner's corpuscles***, ***Krause's end-bulbs***, ***Ruffini's corpuscles*** and ***Pacinian corpuscles***.[5] As a group, these organs serve to provide the individual with the means to perceive superficial pain, light touch, cold, warmth, deep touch, pressure, vibrating sense, deep pain, the appreciation of movement or limb position and to judge shape and form, in all their variations and combinations.[6]

Within voluntary muscles are ***neuromuscular spindles*** that serve to provide an ongoing gauge of muscle length and the ***speed of stretch*** for "***muscle sense***," as well as the primary source of ordinary muscle contraction. The ***Golgi tendon organ*** acts (with other deep sensory end organs) to influence the gamma loop reflex as an ***inhibitory counterbalance*** to the excitatory effects of ***increased tendon tension*** on the muscle spindle to provide precise integration and timing of the stretch reflex activity. ***The motor end plate*** serves to provide a receptor for ***efferent motor nerve impulses***. These impulses are transduced into chemical reactions, which translate into extrafusal and intrafusal muscle fiber contraction. The neurons innervating these end organs interrelate with one another and the supraspinal structures in the ***spinal cord gray matter***, either directly or indirectly through interneurons, to provide neuromuscular feedback loops, both positive and negative (see **The Muscle Spindle and the Central Nervous System**, **Chapter 1**).

The **motor neuron** *is composed of a* **trophic** *and* **receptive** *segment, made up of the* **cell body** *and its* **dendrites***, and a conductive segment, the* **myelinated axon***. The neuron responds to stimulation (electrical impulses) from other neurons by producing its own electrical impulses that are transmitted down its axon to its* **motor end plates** *(see* ***Figure 18****). The* **sensory end organ** *stimulates the sensory neuron to produce electrical impulses that travel up the receptive myelinated axon to its* **trophic segment***, the* **dorsal ganglion***, made up of the* **cell body** *and its* **satellite cells***. The dorsal ganglion allows the impulses to continue via its transmissive segment to dendrite synapses with other neurons in the spinal cord gray matter (see* ***Figure 19****).*[7]

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5 *Meissner's corpuscles, Merkel's corpuscles, and hair follicle plexus subserve the tactile sense; Pacinian corpuscles subserve pressure, speed of tissue displacement, and vibratory senses (and possibly serve as pain sensors of a given order); Ruffini corpuscles and Krause end-bulbs subserve thermal sensors of warmth and cold respectively; and free nerve endings subserve pain sense as do other end organs (some of the muscle spindle sensory functions also include the pain sense of over-stretch). No doubt other organs of sense will be defined in the future.*

6 *Sensations are probably perceived by relative stimulation. Light touch is perceived, for example, because the area surrounding the touched area is not being stimulated. Sensory nerves send a steady stream of electrical impulses to the central nervous system. When something is felt, it is because some end organs surrounded by others have increased their impulse rate, above the norm, while the surrounding organs did not.*

7 *Note that the neurons, both motor and sensory, which innervate the muscle spindle, are required to penetrate the conglomerate collection of muscle bundles and fascial layers making up the extrafusal muscle, as well as the muscle spindle capsule to reach their respective transmissive end organs, unlike the neurons to the skin and extrafusal muscle end organs.*

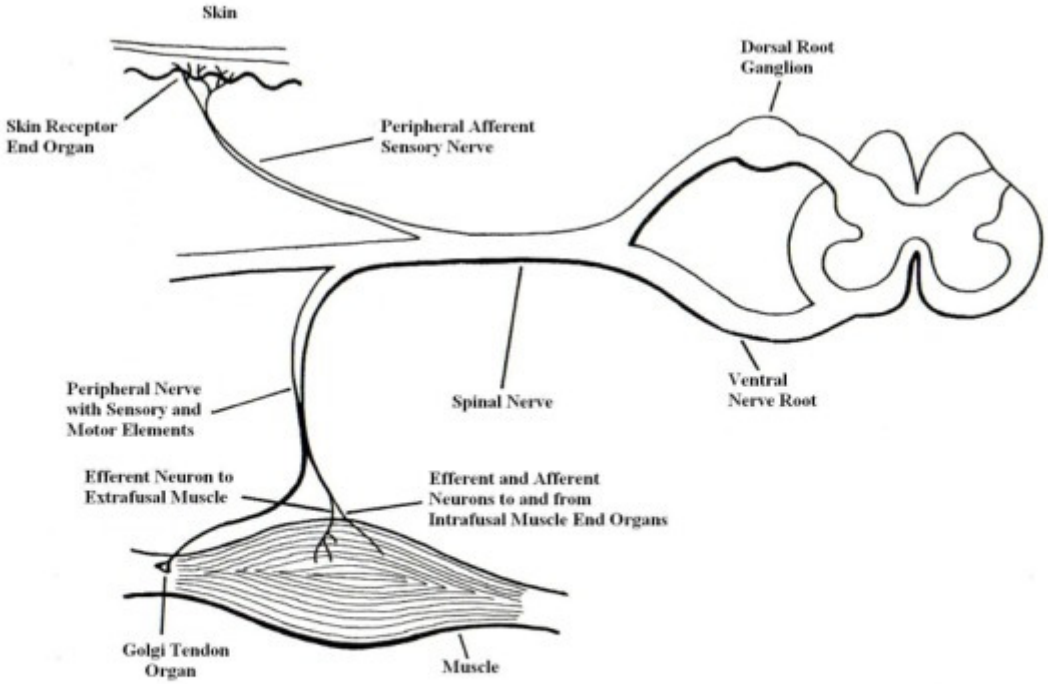

Figure 17

A REPRESENTATIVE VIEW OF THE SPINAL NERVE, SUPPLYING BOTH AFFERENT NEURONS TO THE SKIN AND MUSCLE

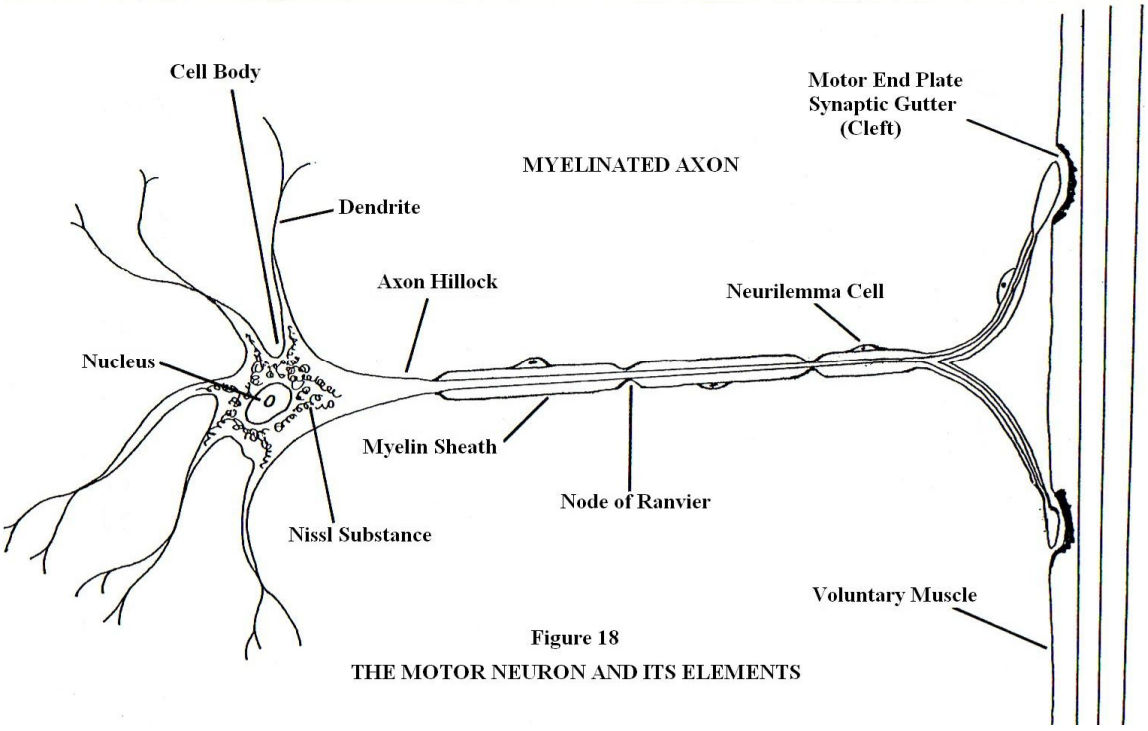

Figure 18

THE MOTOR NEURON AND ITS ELEMENTS

When a peripheral spinal nerve has been damaged by transection, crushing, blood deprivation or toxic substance, each neuron that has been damaged (sensory and motor alike) must go through a process of ***degeneration and regeneration***. The entire neuron responds. ***Wallarian degeneration*** of the axon occurs proximally from the first ***node of Ranvier*** to the end organ, distally. The cell body goes through changes that include swelling, becoming turgid and increasing protein synthesis. A breakup of the myelin sheath and axon cylinders occurs in several of the internodes proximal to the site of injury, proceeding distally to the end organ, leaving only the ***neurilemma shell*** to maintain a pathway for future nerve regeneration.

Regeneration normally begins when the severed axon forms a new cell membrane and sends out branches to follow the neurilemma. If the injury site is bridged successfully, normal ***axis cylinder*** regrowth occurs at the rate of four millimeters per day with the myelin sheath ***regenerating at two millimeters per day***. After several months, the axis cylinder reaches the end organ, which is ***reinnervated*** or (as in the case of motor end plates) ***reconstituted***. Any branches that fail to reinnervate end organs degenerate, as do ***collateral branches*** from undamaged neurons that have been replaced at the end organ sites by regenerated axons.[8] Impediments to regeneration of injured neurons include displacement of the severed neurilemma ends (out of line with the regenerating axonal branches), continued pressure, and scar tissue (collagen fiber growth) obliterating the ***neurilemma axis cylinder pathway***. Clinical observation has also suggested the probability that the failure on the part of sensory nerves to regenerate to the organs of "muscle sense" (the muscle spindle) is due to the failure on the part of the neurilemma to maintain an open pathway for axis cylinder regrowth through the various layers of muscle and related tissues, thus falling prey to muscle pressure or stricturing fascial layers. This postulate would explain why so many examples exist of ***extrafusal muscle motor innervation coupled with a complete lack of "muscle sense"*** (See **Figures 12** and **13**).

Motor Nerve Damage Paralysis

If a motor nerve is damaged and fails to regenerate from the neuron to the motor end plate, a ***flaccid muscle*** condition results. This condition is illustrated in **Figure 14** of the previous chapter, which shows the effects of a complete ***lack of positive feedback*** to the muscle. The assumption is that the sensory nerve does not survive. Should afferent sensory neuron reinnervation occur without efferent motor neuron reinnervation, ***voluntary muscle activity and neuromuscular reeducation is impossible***. However, motor nerve destruction without regeneration to the motor end plates on extrafusal muscle is unusual. Usually paralytic problems stem from afferent sensory neuron failure to reinnervate muscle spindle sensory end organs, thus depriving the cerebellum (**Computer *D***) of negative feedback from the muscles. ***Computer D*** is therefore unable

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8 *The process of regeneration stimulates undamaged neurons to attempt reinnervation of denervated end organs by collateral sprouting through their Modes of Ranvier. These collateral branches are capable of reinnervating denervated end organs but with the loss of independent response of the co innervated structures, if they are not replaced by the proper regenerating axons (Noback, 1967).*

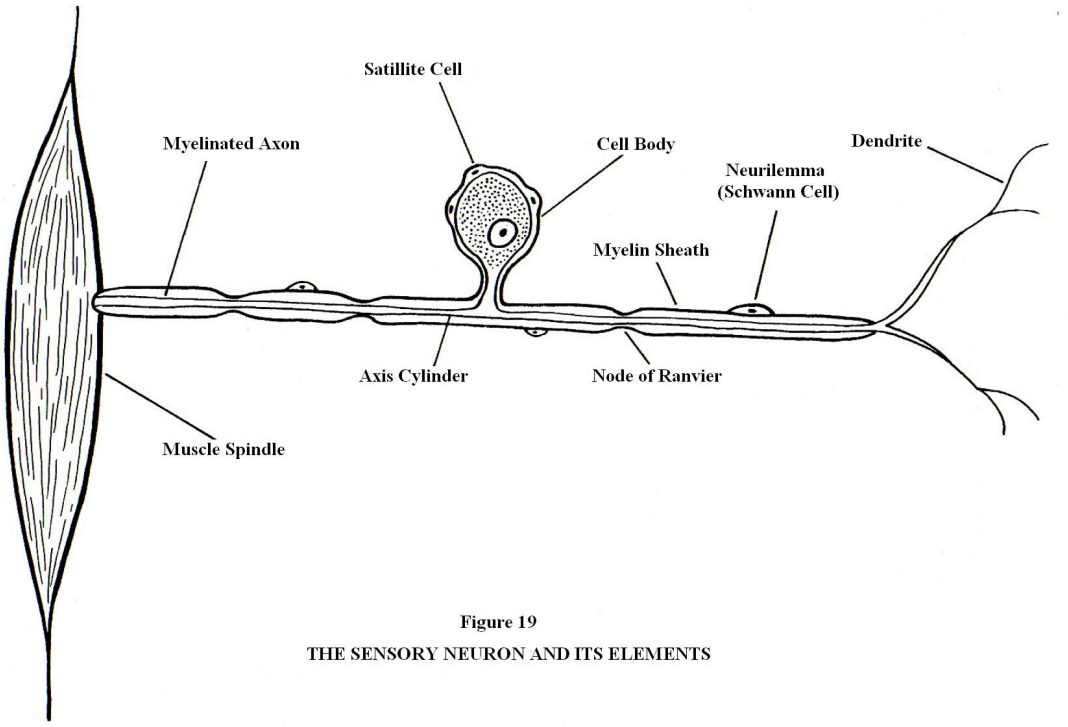

Figure 19
THE SENSORY NEURON AND ITS ELEMENTS

to coordinate supraspinal structure activity because of its inability to perceive what affect supraspinal efferent activity has had on muscle activity. The initial supraspinal response to a suspension of ***Computer D*** coordination resulting from a peripheral nerve injury is similar to the initial supraspinal response to a CVA, ***a complete suspension of efferent neuromuscular activity***. However, the subsequent supraspinal long-term response to the peripheral nerve injury varies a great deal from subsequent responses to a CVA. For instance, the functionally flaccid long-term response to the lack of negative feedback seems much more prevalent in the ***post peripheral nerve injury syndrome*** population than in the post CVA population, and ***the spastic response is relatively rare***.

When a spastic response does occur as the result of a peripheral nerve injury, the muscles involved seem more susceptible to the influence of ***contralateral*** and ***homolateral associative reactions*** and to ***synergistic patterns*** more than to developmental reflexes (see **Tables 2** and **5**). This probably occurs because the supraspinal pathways have not been damaged and are not susceptible to ***sensory-triggered reflexes***, indicating possibly that the cerebellum is still able to provide some of its inhibitory function, even without negative feedback from the musculature. The central nervous system is spared full regression to primitive developmental stages.

Rehabilitation of Peripheral Nerve Injury

Rehabilitation of the post-peripheral nerve injury syndrome has been thought to be simple, involving electrical stimulation of involved musculature. Galvanic or sinusoidal currents have historically been used to provide high amplitude long-duration stimulation that provoke involuntary contractions of the denervated muscle. The use of the electrical stimulation has been justified by the idea that the muscle so stimulated could maintain a modicum of tone until the damaged nerve had regenerated. This approach may be based on some sound rational, but it was also implied that such stimulation might even help the muscle or central nervous system to ***"remember"*** how to perform contractions of the involved musculature. Such implications may be ***without foundation*** since the contractions so caused are involuntary. It is like assuming that a person can learn to throw a ball by hitting him with it. The real drawbacks to the use of galvanic or sinusoidal currents include the fact that the currents used are painful (and in the case of galvanic current, dangerous), when delivered at amplitudes that cause involuntary contraction of the denervated muscle and any "remembering" would be dependent upon afferent sensory feedback to the supraspinal structures, but if such input were available such "remembering" would be unnecessary.

Exercise is another standard treatment used to encourage the return of muscle activity, even before any visible muscle contractions are evident. The assumption is that activity in uninvolved muscles related by virtue of proximity or function (homolaterally or contralaterally), helps facilitate activity in involved muscles.

Exercise, as well as the applications of galvanic or sinusoidal electrical stimulation may indeed aid neuromuscular rehabilitation, even without the return of the originally injured neurons. These programs possibly encourage collateral reinnervation of denervated end organs from other damaged neurons in the proximity of the damaged neurons. However, if true, this type of reinnervation might limit integrated complex functional activity to nothing but ***patterned, gross motion***. For example, should the hypoglossal nerve collaterally innervate the distal portion of the facial nerve, the muscles of expression so involved might only respond when the tongue moves (Noback, 1967).

Appropriate exercise is advisable, in any case, for the toning of muscles which show evidence of returning function with visible or palpable muscle contraction, especially in light of the observation that regenerated neurons have a ***diameter and conduction velocity of 80% of normal***, indicating a compensation may have to be made for decreased efficiency in speed of response and contractibility of the involved muscle mass (Noback, 1967). However, if the musculature involved is fully reinnervated by the motor and sensory neurons necessary for a complete neuromuscular feedback loop, limited compensation should be necessary for normal functional usage (with proper patient motivation). If the negative feedback pathway from the muscle spindle is impaired, it is advisable to provide at least a temporary completion of that loop with electromyometric feedback. The protocol involved is much like that followed in the rehabilitation of post CVA syndromes.

Initially, an evaluation of the involved muscles utilizing standard muscle testing techniques should be made, followed by an in depth evaluation utilizing electromyometry (Daniels, Williams and Worthingham, 1969). In most cases of paralysis resulting from peripheral nerve injury, the involved musculature will initially demonstrate either a flaccid state with no overt motor activity or, more likely, fasciculation's with a low steady state of myoelectric activity from randomly-firing myofibrils.[9] In both instances, a ***baseline of myoelectric activity*** should be established at rest (the ***maximum level of involuntary myoelectric activity***). The patient should then be asked to try to increase myoelectric activity (as reflected by the electromyometer) from the muscle monitored during six-second attempts. The best response (total mv achieved) should then be compared with attempts made with the help of the ***contralateral*** and ***homolateral associative reactions*** combined with ***synergistic reflex patterns***. If these attempts fail to produce any myoelectric activity above the established baseline, then an attempt should be made to elicit the desired responses through the use of ***developmental reflexes***. Measurements of the myoelectric responses should be made ***without*** the patient being able to observe either the visual or auditory feedback from the electromyometers. If any of these attempts are successful in increasing myoelectric activity in the involved muscles, then rehabilitation may begin utilizing the technique that evoked the response. If all of the attempts prove successful in producing increased myoelectric activity above the established baseline reading, then the reflex mechanisms that produced the greatest response should be the reflex used initially in the patient's rehabilitation program.

As in the functionally flaccid post CVA syndrome rehabilitation program, the next step is to begin the long treatment process of ***facilitation, inhibition, balance and function*** (refer to **Tables 2, 3, 4** and **5**).

To illustrate, I was asked to evaluate the condition of an 18-year woman who twelve months earlier had her leg completely severed at the knee. Successful surgical reattachment was accomplished. The surgery was obviously remarkable, but the orthopedic surgeon had given her no hope of regaining muscle control below the knee because of what he called "scar tissue", which (in his opinion) had to have obliterated regenerating nerve pathways, both motor and sensory, precluding any reinnervation of sensory or motor end organs; this scar tissue was thought to have developed as a consequence of the soft tissue repair. She was told that she would have to continue wearing a short-leg wire brace to compensate for foot drop. When initially evaluated, she showed ***demonstrable voluntary myoelectric activity*** of several mv in each of the involved muscles. This activity was not enough to generate visible or palpable contractions, but it verified motor neuron regeneration. A rehabilitation program was begun, taking advantage of the contralateral associative reactions, continuing until visible contraction of the involved musculature could be demonstrated without the use of the contralateral associative reaction. The ***homolateral associative reactions*** were then used

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9 *Fasciculation's appear as irregular bursts of independent myoelectric activity and are associated with denervated muscle (without efferent innervation). Spasticity appears as regular rhythmic "pulses" of activity when the muscle is stretched and is associated with hypertonus (increased* ***tonic*** *gamma neuron activity) seen in central nervous system lesions of the internal capsule and primary motor cortex, though not exclusively.*

in a facilitory manner until functional mv levels were demonstrated by each of the involved muscles. The ***homolateral associative reactions*** were then inhibited. Differentiation of the muscles involved in the homolateral associative reactions is necessary for the development of complex muscular ***differentiation, integration and function***. Once independent visible contractions were demonstrated in this patient, she was asked to perform them without electromyometry both in the clinical setting and at home. After a year and a half of this program, the patient had gained enough control of the involved musculature to return to training as a tap dancer.

Assistive Devices

Assistive devices can be as big a problem to the post peripheral nerve injury syndrome patient as they are to the post CVA syndrome patient, if they are utilized beyond their necessary role as protective devices or for joint stability and protection. If they are used after reinnervation has occurred (as established by electromyometric evidence, or visible or palpable contraction), they may promote weakness by preventing joint pressure, joint traction and muscle stretch from making proprioceptive demands on the muscles for support. Outstanding in this regard (as discussed earlier) are the wheelchair, pickup and rolling walkers, arm and forearm slings and short-leg braces. All of these not only provide inhibitory influences on muscle toning and proprioceptive demand, but also serve as ***impediments*** to the development of ***balance*** and other ***functional skills***. They can do damage to the patient's self-image and can promote inappropriate emotional and physical dependencies on these devices or family members (or other individuals in their environment). Much of the responsibility for the development of these dependencies must be laid upon the therapists, who have the job of determining the appropriateness of the use of any adaptive or supportive devices at all stages of the patient's recovery. It is well within a therapist's talents and training to constantly reevaluate a patient's progress and to promote appropriate functional skills aimed at cutting down the use of assistive devices. Without such attention, the patient's development may be arrested with the resultant failure to attain optimal functional levels or performance. Needless to say, the elimination of assistive devices must be timely and made with regard to the patient's safety and well being, with additional attention paid to joint integrity and stability. It is not suggested that the patient be relieved of all responsibility for situational judgment or cooperation, or that motivational factors do not play a major role. The therapist is, however, responsible for careful ongoing evaluation of the patient's abilities and for providing skillful guidance toward realizing their full potential.

Bell's Palsy (Facial Nerve Paralysis)

The most commonly seen peripheral nerve injury syndrome is ***Bell's palsy***, or facial nerve (cranial nerve VII) paralysis. The frequency of its occurrence may be blamed on the nerve's close proximity to the mastoid process as it makes its way to the face. Should the tissue associated with the mastoid process become swollen, the facial nerve may become compressed. The effect may vary between patients from a numbness of the skin and a temporary loss of voluntary muscle control (some three days to a week in duration), to profound sensory and motor damage resulting in complete ***anesthesia*** of the entire

involved area and total loss of ***voluntary motor control*** on a long-term basis (from months to ***permanently***).

In cases of a long-term syndrome, the damage is sufficient to produce peripheral degeneration of the axons, but the involved neurilemma sheathes are usually intact. The problems of regeneration are the same as discussed earlier with the potential problems of reinnervation of the involved muscle spindles (the neurilemma sheath may not be able to hold the pathway open to the sensory or motor axonal branches to the muscle spindle) as it pierces the various tissue layers. This may preclude the redevelopment of muscle sense. On the other hand, the motor neuron to extrafusal muscle (and its suspected intrafusal muscle) may meet no such impediment and is likely to be reinnervated. In such an event, the patient is left with muscles that could be voluntarily controlled but for the fact they ***cannot be "felt."*** In this case, electromyometric feedback can provide aid to rehabilitation by supplying a temporary substitute for the ***lack of "muscle sense"*** and (as seen in post CVA syndromes and other like conditions) make possible the reestablishment of full functional control of the involved musculature (by making possible the ***reprogramming of the cerebellum***).

The ***contralateral associative reaction*** may help to facilitate voluntary myoelectric activity in the involved facial muscles. Once visible contractions appear, the patient must be taught to contract the muscle ***independently on the homolateral side***, ***while inhibiting the contralateral musculature***. To promote differentiation and integration of muscle function, the homolateral associative reaction must be broken. This can be done by facilitating myoelectric activity in one muscle while inhibiting myoelectric activity in the other on the homolateral side. As voluntary visible contractions are observed, the patient should be encouraged to practice these contractions at home in front of a mirror while performing facial exercises designed to promote toning (see **Table 7**), first employing both sides of the face and then only the involved side, with differentiation between the various muscles. The success of such a program will be evident in the return of unconscious muscle activity of involved musculature during facial expression (smiling and frowning). Some habitual mannerisms may remain because of the problem of habituation (discussed in regards to post CVA syndrome rehabilitation) (such as talking with only the uninvolved side of the mouth) but normal facial wrinkles should reappear as well as other functional unconscious responses like normal eye blink, and sniffing.[10]

SPINAL CORD INJURY

The Spinal Cord

The spinal cord is made up of gray and white matter. The gray matter achieves its color by virtue of its ***vascularization and its lack of myelin***. It is made up of cell bodies, unmyelinated nerve fibers, and lightly myelinated nerve fibers. The majority of these

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10 *See* ***Table 6*** *and* ***7*** *for facial muscle functions and exercise and* ***Electrode Placement Illustrations*** *for electrode placement on facial muscles.*

nerve fibers are said to be oriented at right angles to the long axis of the cord. The gray matter composes the central part of the spinal cord, as illustrated in **Figure 20**. It is divided into three regions: (1) the ***anterior ventral gray horns***, which are made up of motor cell bodies and synapsing nerve endings; (2) the ***poster dorsal gray horns***, made up of endings from somatic afferent fibers of the spinal nerves, interneurons and cell bodies whose axons project fibers to both the gray and white matter; and, (3) the ***intermediate gray zone*** made up of interneurons that communicate to the general visceral afferent fibers from the spinal nerves and descending nerve fibers from the supraspinal structures and other spinal cord levels.

The white matter is made up of **unmyelinated and myelinated nerve fibers** *that are oriented parallel to the long axis of the cord. The myelinated fibers are responsible for the white color. There are* **no nerve cell bodies** *in its composition. It is divided into* **funiculi***, or* **tracts***. These tracts include* **ascending tracts** *to the supraspinal structures (comprised of sensory nerve axons), tracts of motor nerve axons* **descending** *from the supraspinal structures and of ascending and descending tracts* **between spinal cord levels***. The* **sensory tracts** *include the fasciculas gracilias, fasciculas cuneatus, lateral spinal thalamic, ventral spinal thalamic, ventral spinal cerebellar and dorsal spinal cerebellar tracts. The* **motor tracts** *include the lateral corticospinal, vestibulospinal, tectospinal and rubospinal tracts. Other tracts are included in the illustration provided by* ***Figure 21***.

The spinal cord provides the pathways necessary for positive and negative feedback between supraspinal structures and the neuronal end organs (both motor and sensory), as well as between infraspinal structures. It provides the optimal opportunity for quick and refined coordination of function (the stretch reflexes and the gamma reflex loops, for example). These pathways are necessary for normal neuromuscular function. Should these pathways be damaged, normal neuromuscular function may be altered or prevented.

The spinal cord may be damaged in many ways. It can be crushed, cut, bruised, overstretched, chemically corrupted, starved of circulation, abnormal formations, or deposits that may obstruct its pathways. All such damage may result in the destruction of axons or nerve cell bodies and the interruption of afferent or efferent spinal cord pathways.

Recovery from Spinal Cord Injury

When the spinal cord is injured, the individual nerve cells respond much like peripheral nerves that have been damaged. The damaged axons die and are reabsorbed back to the originating cell body. If the cell body has survived the trauma, it begins the process

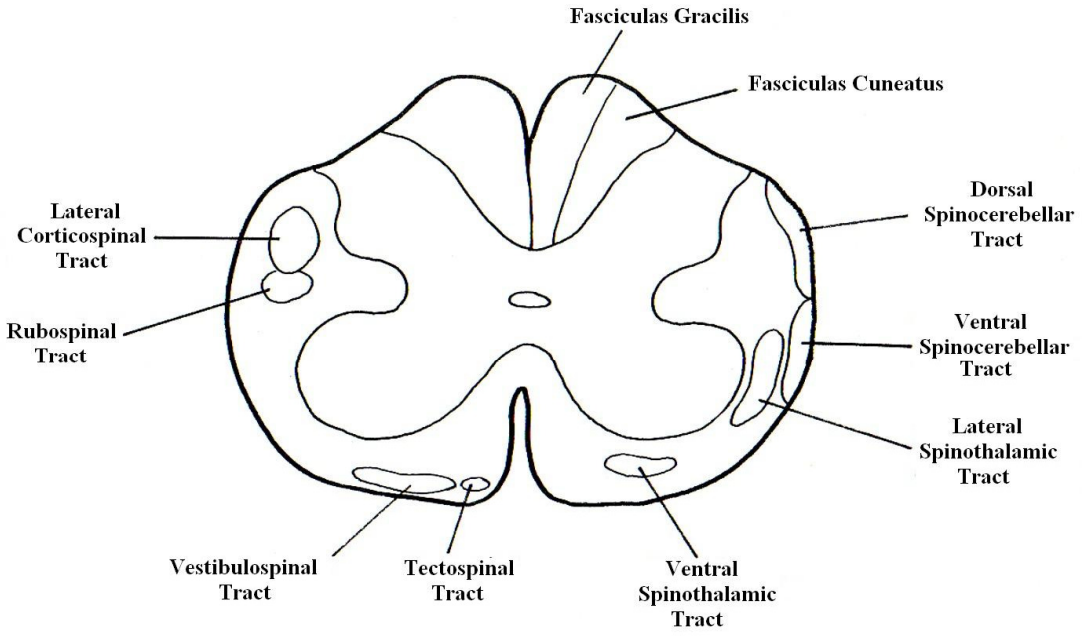

Figure 20

CROSS SECTION OF THE SPINAL CORD WITH SENSORY AND MOTOR TRACTS

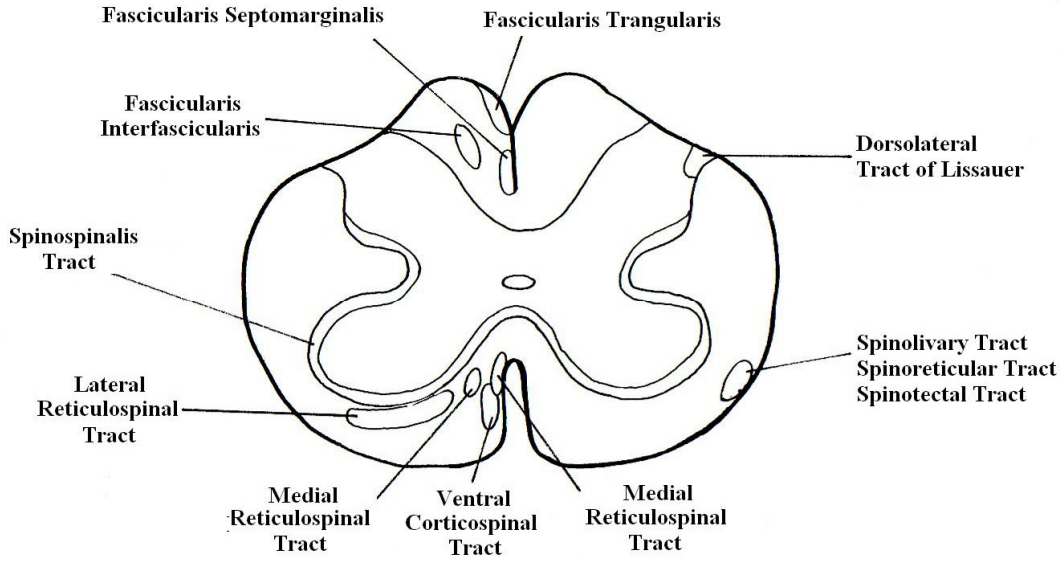

Figure 21

CROSS SECTION OF THE SPINAL CORD WITH SENSORY AND MOTOR TRACTS

of regeneration. Unlike the peripheral nerve, however, the regenerating spinal cord nerve is faced with barriers it may not be able to overcome. First, the spinal cord must overcome the edema or swelling resulting from the pressure of increased fluids in the immediate area of the lesion (pooled blood, etc.); this recovery may take as much as two to three weeks. Second, it must overcome an initial reflex decrease in local blood flow, as well as the circulatory decrease resulting from damaged vessels; collateral circulation must develop with new blood vessels and an expansion of existing ones. It may take hours or days for the reflex decrease in blood flow to be overcome and often a period of months will elapse before collateral vascular development is completed. During these stages, all nerve transmitters, both ascending and descending, are ***halted below the site of lesion***. As the edema is reduced and circulation is restored, this "***spinal shock***" is eased and some of the reflexes may return. These may include the knee jerk, the flexor reflexes and the crossed extension reflexes (first stage developmental reflexes). Much depends on the extent of the injury. If the lesion is not a complete transection of the cord there may be a great deal of "***spontaneous recovery***" of both motor and sensory elements. If the cord is completely transected, traditional theory states that only the ***stretch reflex*** and some of the ***autonomic reflexes*** may reappear.[11] The third step in spinal cord recovery is the ***regeneration of axons and dendrites from the involved nerve cell bodies***. If it is indeed possible, it would entail the regeneration of the involved axons to and across the lesion gap and down the remaining cord to their synapses with interneurons and spinal cell bodies, dorsal root ganglia, etc. The obstacles to such regeneration are many, but numerous examples exist of motor and sensory return months after injury (sometimes over a year later) in patients said to be without hope of any return because of a complete spinal cord transection. These examples continue to provide the medical and paramedical fields with material for controversy, speculation and continued investigation.

The controversy over the possibility of spinal cord regeneration has been raging for nearly 100 years. It is generally accepted that the adult mammal is incapable of replacing lost neurons or causing neuronal division in the central nervous system, and no hard evidence has been brought forward to refute this view (Altman, 1962). Without the replacement of damaged neurons or mitosis, the adult mammal must depend on ***axonal nerve sprouting and collateral sprouting for reinnervation***. Unlike the peripheral nerve, the spinal cord nerve is ***not*** provided with Schwaan cells to provide a pathway for axonal regeneration (Weiss, 1934, 1936). This does not preclude sprouting but, as we see in peripheral nerve regeneration, without preexisting intact pathways for axonal regeneration regrowth may be easily deterred or misdirected and prevented from reaching the appropriate end organs. Inappropriate connections may even be established with denervated structures in close proximity to the lesion site, thereby halting axonal growth (Bernstein and Guth, 1962; Sperry, 1945). Further, it has been shown that while denervated tissue is a stimulus for spinal cord nerve regeneration, it also promotes collateral nerve sprouting from undamaged neurons. The collateral sprouts may reinnervate the denervated end organ prior to the arrival of the regenerating axons. It has

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11 *The spontaneous return of motor and sensory components varies a great deal between individuals with apparently similar injuries. The differences range from (apparently) complete flaccidity below the site of lesion years after injury to dramatically spastic and hyper reflexive musculature six weeks after injury. Sensory return is also as varied.*

been demonstrated that these sprouts have the ability to deny these sites to the more appropriate regenerating axon (unlike their peripheral nerve counterparts) (Windle, 1970).

Other authorities have claimed that interruption and damage of the vascular system around the lesion site precludes functional regeneration of spinal cord nerves and precludes the dissolution of all supportive tissue in the area, causing an ***"isolation dystrophy"*** (Hunter and Royal, 1924). This points to a hypersensitivity of neural tissue to nutritional deprivation. One writer said, "It is common knowledge that neurons cease to function normally within a few seconds following alterations in circulation" (Clemente, 1964).

Another deterrent to functional spinal cord nerve regeneration is the formation of scar tissue. ***Scar tissue*** can potentially obliterate the access across the lesion site, stemming the advance of regenerating axons. This mechanical barrier ***becomes the single greatest impediment to spinal cord nerve regeneration*** (Rose, Malis, Kruger and Baker, 1960).

Scar tissue may even be a threat to those who have had minimal immediate disruption of function from spinal cord injury because of a tendency to develop over a long period of time (often years) at the site of lesion. It gradually constricts the spinal cord, cutting off nerve pathways, and stricturing the vascular supply. For example (sited by Windle, 1955), a young woman had her spinal cord severed by a bullet at the tenth thoracic vertebra. The surgeon involved was able to approximate the ends of the severed cord so successfully that by the sixteenth month he reported that the patient showed voluntary muscle activity throughout the lower extremities, with Fair (+) muscle grades in the hip flexors. Touch, position sense and temperature awareness had returned to a great extent throughout, as had partial bowel and bladder control. At the end of 19 years, she was reexamined, and it was found that she had become totally paralyzed below the waist and had lost all sensory perception below the site of lesion. Upon her demise, five years later, an autopsy was performed which showed the spinal cord constricted by scarring at the site of lesion and surgical repair. Similar results have been noted in studies utilizing experimental animals (Windle, 1955).

Studies have been developed which have supported both sides of the controversy over spinal cord regeneration. Some have shown varying degrees of recovery from spinal cord transections, while others reported complete failures to recover. The proponents of recovery claim that studies showing no recovery utilized surgical techniques that produced wide-spread cord damage and alterations of vertebral structures and that there was poor postoperative care (Windle, 1955, 1970; Clemente, 1964). The opponents of recovery suggest that studies showing recovery were victims of optimistic interpretation, that ***incomplete surgical transections*** of the cord were made and that recovery of "function" was a result of coordination of reflexes in the cord below the transection by upper body manipulation (Davidoff and Ramsloff, 1948).

Regardless of the criticism, research evidence has come to light that supports the possibility of spinal cord recovery, at least in part. Research has shown that regenerating

axonal fibers may be capable of ***carrying across the lesion gap*** (Clearwaters, 1946, 1954; Liu and Chambers, 1957, 1958) and even of ***penetrating scar tissue*** (Berstein and Berstein, 1967, 1969; Berstein and Gelderd, 1970). Missing from the literature reviewed were studies making it clear that regeneration was equally prevalent for ascending as well as descending neurons. Most, if not all, attention seemed to be focused on efferent innervation. Indeed, evidence suggests that what regeneration has been demonstrated has occurred in ventral areas of the cord (Clearwaters, 1946, 1954) and that ***unmyelinated fibers*** might be responsible (Hoy, Bittner and Kennedy, 1967). It would seem that such evidence suggests that the ***ventral horns of the gray matter*** may be the sites for ***potential spinal cord reconnection***. If that is the case, such reinnervation would provide the patient with the potential for interneuronal communication which could provide a channel for the positive feedback required to produce the first stage reflex activity and spasticity cited earlier. It would not provide for the negative feedback required for normal neuromuscular function. Judging from the clinical evidence available through our work with electromyometric feedback, it seems far more likely that some reinnervation takes place, even in complete transections, than that none does.

Rehabilitation of the spinal cord injured patient is similar to that faced by the peripheral nerve injury syndrome patient. They are faced with having the possibility of sending messages down (from supraspinal structures), but are unable to receive negative feedback from the muscles. Cases of intense spasticity (***first stage developmental reflexes***) and other cases of near total flaccidity (showing only low level facilitory activity) are as evident in this population as they are in the post CVA syndrome population.

To illustrate, I was called in as a special consultant to one of the rehabilitation hospitals to evaluate a young man who had broken his neck at the C3-C4 level. He had been told that he would be a quadriplegic, without the hope of developing functional activity in any of his extremities. It was six weeks after the injury when I evaluated him. The spinal cord injury had been initially judged by his neurosurgeon to be a complete transection, and this was confirmed by a second neurosurgical opinion. This diagnosis was later modified by the attending physiatrist as being an ***"almost complete transection"*** because the patient had maintained the ability to breathe without the aid of a respirator. The patient had experienced an early onset of ***extreme spasticity*** in his lower extremities, which took the form of ***exaggerated clonus*** in the ankles when sharp pressure was applied to the balls of the feet. It was reported that he had been placed on a spasticity inhibitory medication but that it had not been effective, and that the clonic episodes could continue unabated for very long periods (one-half hour or more). It was also reported that the patient had demonstrated no voluntary control of any skeletal muscles of the upper or lower extremities or of the trunk. Upon entering the patient's room, I found him lying flat on his back with a pillow under his head and a footboard against the soles of his feet. The clonic activity of his ankles was so severe that he was rocking the bed. I had the footboard removed and began my evaluation. The upper extremities showed passive range of motion within normal limits and no visible voluntary motion throughout, but the patient was able to demonstrate several mv of voluntary muscle activity (above the baseline readings) in literally all the muscles in his upper extremities, including the finger flexors and extensors. This activity, of course, was below that necessary to demonstrate

visible or palpable contractions. I then began the electromyometric evaluation of the lower extremities. The patient was unable to generate any voluntary activity in any of the skeletal muscles below the waist. Frustrated, I decided to approach the problem from another point of view. I had the footboard put in place against the planter surface of his right foot and gave the gastrocnemius a quick stretch to evoke the clonic response. It began immediately. I had previously attached an electromyometer, with auditory and visual feedback, to the right gastrocnemius. I asked the patient to decrease the apparent auditory and visual activity that was being produced in regular high amplitude bursts. In approximately five minutes, the clonus stopped. I started it up again with the quick stretch and asked the patient to stop it once again. This time it stopped in approximately three minutes. This procedure was repeated several times, and he was eventually able to stop the clonic activity at one-minute intervals. One of the physical therapists, which had been observing the evaluation, suggested that the clonus was abating from simple fatigue. To check this premise, we set up the footboard against the plantar surface of the left foot with the electromyometer attached to his left gastrocnemius. I then triggered the clonic activity, and he was able to stop it in less than two minutes; subsequent attempts took less than a minute each. I left the family with my recommendations that he receive electromyometric feedback neuromuscular reeducation from a colleague who lived in the area. I received a report some eight months later that the patient was able to sit up on his own and feed himself. No further follow-up was made, so we can make no statements about long-term scar tissue development in this case.

I have had occasion to work with several quadriplegic or paraplegic patients five to ten years after their injury before beginning electromyometric feedback neuromuscular reeducation. All of them were able to demonstrate voluntary control of myoelectric activity from the musculature below the site of lesion, regardless of the fact that their spinal cord injuries had reportedly resulted from complete transections. One patient diagnosed as having a complete lesion at C4-C5, was able to produce enough myoelectric activity to demonstrate visible finger flexion. This movement did him little good, however, because the finger joints (the metacarpophalangeal and proximal interphalangeal) had been fused to provide a ***tenodesis pinch***. On my recommendation, an outside neurologist performed a diagnostic EMG. The neurologist reported voluntary muscle potential down to the T6 innervation level.

All this points to the suggestion that regeneration may occur across the spinal cord lesion. This regeneration may occur only in central gray columns of the spinal cord, and although some functional neuromuscular reeducation may be possible, it may only be possible through the use of electromyometric feedback supplied as a supplement to disrupted afferent feedback flow.

The program suggested for the peripheral nerve injury syndromes patient should be applied to the spinal cord injured patient with special attention paid to the use of developmental and synergistic reflex patterns for helping the patient learn to facilitate and inhibit muscle activity as noted in the section on the post CVA syndrome.

Special Considerations

The time elapsed since the injury is especially crucial for the post spinal cord injury syndrome patient for the initiation of the electromyometric feedback neuromuscular reeducation program. If the patient is flaccid, the electromyometric feedback program should begin within a year. If she is spastic, she should begin the program before two years has elapsed. In the former case, atrophy is the deciding factor. After a year of inactivity, much muscle tissue may be lost, and the prospects of functional gains dwindle. In the latter, the accommodation to the wheelchair lifestyle may defeat efforts to modify and keep up the necessary routines of electromyometric feedback training and exercise. Beyond that, it has been speculated (Margaret E. Ayers, 1975, unpublished) that continual lack of ***afferent proprioceptive neuromuscular feedback*** may produce a condition in the supraspinal structures that will inhibit any attempts at neuromuscular reeducation by ***surreptitiously blocking efferent neuromotor activity***.

Bracing is a large problem for many paraplegic and quadraparetic patients. The general practice of long-leg bracing paraplegic or quadraparetic patients is usually a waste of time, money and energy. Most long braces eventually find their place in dark closets with the Canadian crutches that were issued with them, displaced by the general use of more convenient and less energy-consuming wheelchairs. It is our opinion that if a patient does not have the potential for using short-leg braces (the ***metal double upright adjustable fixed-ankle joint short-leg brace***, or the ***short-leg cosmetic plastic shell***), bracing should not be attempted, and the patient should be taught to accommodate to the wheelchair and to compensate for the physiological need for gravity through the regular use of a ***standing table***.

Surgical intervention has proven a problem with some of our patients. Fused joints or lengthened or transplanted tendons have limited rehabilitation of recovered neuromuscular function (as mentioned earlier). If ***no*** efferent nerve pathways exist down the spinal cord below the lesion site, then a ***true flaccid paralysis will exist without the hope of reactivation or neuromuscular re-education***. In such cases, surgical intervention is certainly justified to improve functional status, but ***the decision to use surgery should be made only after all other possibilities have been explored***. This means: (1) better and more thorough periodic electromyographic evaluation; (2) waiting for any possible spinal cord regeneration; and, (3) giving the patient the opportunity to reactivate or assume neuromuscular control through electromyometric feedback.

If a patient is spastic, it is recommended that the patient ***not*** be treated with spasticity inhibitors. ***Spasticity is helpful in maintaining muscle tone*** until electromyometric feedback can be used to being it under control. If a patient is spastic, it is because of a degree of influence from the supraspinal structures, implying some intact efferent pathways. It is clinically apparent that those patients who cannot increase myoelectric activity from the muscles below the site of lesion, through the use of electromyometric feedback, are those who are structurally flaccid and have not achieved spinal cord regeneration (illustrated in **Figure 14**). ***All*** spinal cord injury patients, in our experience, who have demonstrated marked spasticity clinically, have been able to inhibit that

myoelectric activity to varying degrees through electromyometric feedback. Their condition is best illustrated cybernetically in **Figure 22**.

Bowel and bladder control may be enhanced through the use of electromyometric feedback or through the use of pressure transducers that provide adequate feedback modes, but it must be applied through suppository or catheter appliances.

CEREBRAL PALSY

Cerebral palsy is usually defined as a ***paralysis or lack of muscle control as a result of injury to an immature nervous system***, usually occurring before, during, or shortly after birth. This condition is often accompanied by seizures, mental retardation, abnormal sensory perceptions, and impairment of sight, hearing or speech. The basic causes of this condition include oxygen deprivation (interference in blood circulation), blood/chemical disorders, disease and direct cerebellar concussion. Oxygen deprivation can result from premature separation of the placenta and the uterine wall, strangulation of the umbilical cord or arterial blood supply to the brain, premature birth, or labor that is too short. Blood/chemical disorders, which may damage immature neural tissues, include chemical poisoning (alcohol, tobacco or other drug poisoning) and incompatible blood factors (Rh factor, or AB blood type mismatching between fetus and mother). Sequellae of viral and bacterial disease causing meningitis or meningitis-like conditions (rubella, for example) are said to be the greatest known causes of cerebral palsy conditions. Cerebral concussion can occur as a result of attempts to aid in infant delivery through the use of forceps or from a direct blow to the head.

Obviously, so many causes of damage will result in widely divergent symptomatic profiles. Especially complex are the syndromes arising from damage caused by blood factor problems and disease. Children affected by these often have perceptual problems accompanying the neuromuscular dysfunctions. From a neuromuscular point of view, however, this whole group closely resembles the adult population of post CVA syndrome patients, especially when matched according to cause, extent of damage and the site of damage. Both groups could be classified together in three broad categories usually reserved for differentiation of cerebral palsy syndromes alone: (1) the ***spastic*** group, who move involved joints stiffly and with difficulty; (2) the ***athetoid*** grou***p***, who experience involuntary and uncontrolled movements; and, (3) the ***ataxic*** group, who have problems with balance and depth perception.

The athetosis experienced by the cerebral palsy patient involves ***slow involuntary writhing movements of the extremities*** (often of the face) and extreme hypertonus (rigidity) of the involved muscles. Agitated, emotional responses often increase the strength and violence of the involuntary movement and hypertonus. Voluntary movement is often difficult or impaired, and in extreme cases, impossible. Traditionally, this condition is attributed to damage to the ***globus pallidus*** or to interruption of the feedback loops between elements of the basal ganglia (***Computer C***), thalamus and the cerebral motor cortex (***Computer B***). The abnormal movements are said to be the result

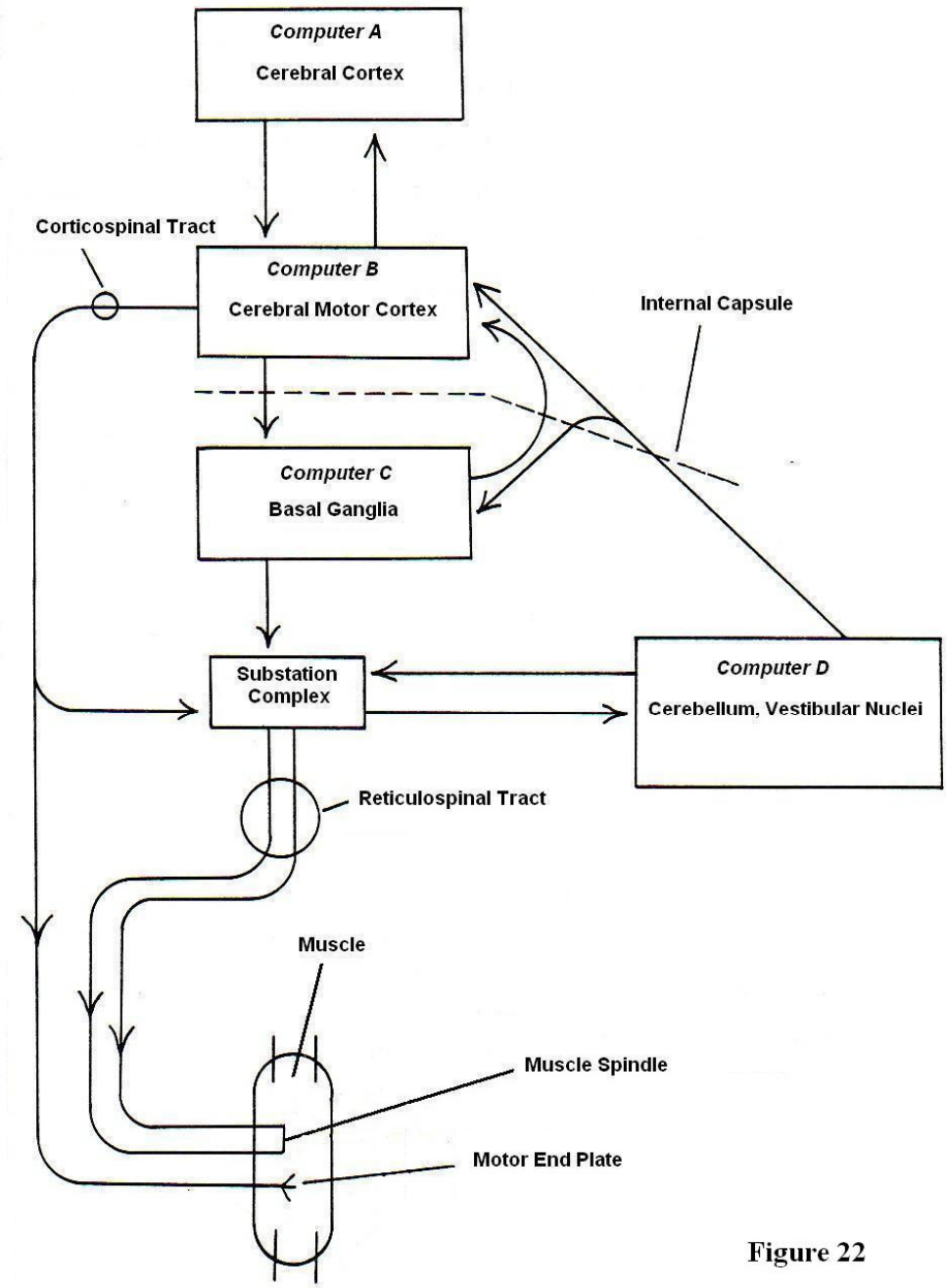

Figure 22

THE HUMAN NEUROMUSCULAR CYBERNETIC SYSTEM WITH DISRUPTED INFRASPINAL FEEBACK LOOPS: THE PHASIC AND TONIC PATHWAYS FROM THE MUSCLE SPINDLE TO COMPUTER D HAVE BEEN LOST

of impulses detouring and taking devious routes through the involved structures (Guyton, 1971).

The spastic cerebral palsy syndromes are usually the result of head trauma and demonstrate the same symptomology as adults with similar damage. If the damage

occurs on one hemisphere, then the patient will have the same symptomology as the hemiplegic post CVA victim. If the damage is to both hemispheres, then the symptomology will resemble the ***quadraparetic post CVA syndrome***, with rather exotic movement patterns due to ***asymmetric damage to the hemispheres***, creating bizarre patterns of motion as the reflexes on one side of the body affect the reflexes on the other side when voluntary movements are attempted. The adult post CVA patient may not be subject to the variety or violent extremes of the counterpart cerebral palsy patient. The differences lie in the previous programming of **Computer D** experienced by the adult CVA patient before onset. This program plays a role in preventing many of the extreme experiences by some of the cerebral palsy syndromes by instituting dominant synergistic patterns. The cerebral palsy child does not have the advantage of having previously learned proximal joint (scapula, hip, trunk, neck and face) stabilization or coordination. Consequently, the **Computer D** programming will include more random and uncoordinated motion of both the distal and proximal joints.

The electromyometric feedback neuromuscular reeducation program utilized for cerebral palsy patients is the same as that utilized for the counterpart adult post CVA syndrome patient, whether unilaterally or bilaterally involved. The cerebral palsy population has a singular advantage over the adult CVA syndrome population in such a program, however, because previously learned neuromuscular programs usually do not exist to any great extent to interfere with the trial and error periods required for neuromuscular reeducation (reprogramming). They have the opportunity to learn faster than the adult group. After all, they are building brand new programs without the necessity of simultaneously inhibiting previous programming, thus they expend less energy and train much faster. I estimate that children respond to electromyometric feedback neuromuscular reeducation five to ten times more rapidly than adults with similar symptoms.

The ***ataxic cerebral palsy*** and post CVA syndromes that display ataxia often resemble each other to a greater degree than the counterparts of the previously discussed groupings. Unfortunately, neither appears to be amenable to biofeedback training, utilizing conventional instrumentation. Such training may be possible, but it will have to involve the development of instrumentation designed to feedback changes in body positions, or reflect perceptual interpretations. ***Any future treatment course of this nature will have to be built around the facilitation and then the subsequent inhibition of the righting reflexes and will have to be incorporated with the Bobath techniques and other approaches that are concerned with vestibular function***.

One of the possible symptoms of cerebral palsy is ***mental retardation***. This term is commonly defined as an inability to learn. While is indeed possible that the immature nervous system can be damaged in the areas that affect learning ability, it may be that this diagnosis is arrived at ***too quickly and without adequate testing***. Many examples can be found of individuals who have demonstrated all or most of the possible symptomology of cerebral palsy and have been diagnosed as being mentally retarded because of an inability to speak or write intelligibly or understand proper communication when tested for the intelligence quotient. A youngster, for example, whose mother had rubella while in the first trimester of pregnancy, demonstrated spastic cerebral palsy symptoms, bilaterally.

As he developed, he proved to be legally blind and "totally deaf." At the age of two years, either because of poor testing or a diagnostic assumption, he was judged to be mentally retarded and made a ward of the state and given up to an institution specializing in the care of the mentally retarded. At approximately five-and-a- half years of age, his intelligence quotient was retested and shown to be at least 90 (verbally and nonverbally), which is at the lower end of normal range. Consequently, he was taken out of the state hospital environment and placed in the ***"better"*** environment of a nursing home. Here, surrounded by the old and infirm, he was expected to mature and grow. Without the necessary environmental stimuli, he might have been developmentally lost, but an adult mentally retarded female (afflicted with ***Down's syndrome***) took an interest in him. She began playing with him and providing him with some of the nurturing he needed for physical and social development. Eventually, at around seven years of age, he was included in a recreational program for orthopedically handicapped children, provided bimonthly by a community volunteer group. While participating in this activity, he came to the attention of an educator, who specialized in the education of blind and emotionally handicapped children. He informally evaluated the child and came to the conclusion that the child was probably more intelligent than the tests indicated (formal testing later confirmed his IQ to be at least 112). Moved by the prospects of a fine mind being trapped in a closed environment, he took the child into his own home as a foster child. Though legally blind, the child had functional sight, and this proved a useful tool in the long ordeal of socialization and educating the child to communicate. The educator and his wife (also a specialist in the education of the blind) learned and subsequently taught the child the fundamentals of sign language. They had the child's hearing tested and found that he had some hearing and, with special, very powerful hearing aids, could discern some articulated sounds.

I was asked to evaluate him for any indication that he might be a candidate for neuromuscular reeducation. He proved to be dominated by first, second and third stage developmental reflexes, which had left him wheelchair-bound. He demonstrated a deformation of the spine (kyphosis) and "paddle" or flipper-hands. He suffered from general muscle atrophy, or lack of muscular development, throughout the trunk and upper and lower extremities. This evaluation was performed before neuromuscular reeducation utilizing electromyometric feedback had been developed. A program, utilizing Bobath and other functional exercise techniques, including gait training with bilateral long-leg braces with adjustable fixed-ankle joints, was outlined and recommended for home use (they lived 350 miles away). Physical therapy was available in the patient's local area, but the therapist involved would only ambulate the patient with long-leg braces and Canadian forearm canes. At that time, he could only ambulate with a bilateral ***"slide-through gait."*** Three years later, I once again evaluated him. This time a gross evaluation was performed, along with an electromyometric evaluation. This evaluation showed him to have spastic elbow, wrist, finger, hip and knee flexors. His ankles were over-stretched by previous physical therapy, and were almost flaccid. He demonstrated almost symmetrical involvement bilaterally throughout with slightly more involvement on the left side. Provided with adequate visual and auditory feedback (through headphones) he immediately demonstrated the ability to facilitate myoelectric activity in the involved extensors and to inhibit myoelectric activity from the involved flexors. An

electromyometric program was recommended and outlined for home use. The patient's father (he had been formally adopted by then) had acquired a pair of electromyometers and had begun a valiant, if irregular, program of neuromuscular reeducation.[12] Through the efforts on his behalf, the patient improved his hand functions, communication ability, his ability to perform the activities of daily living, and eventually developed a fairly normal gait pattern, utilizing the long-leg braces with a pickup walker. Eventually, he developed enough independent function and adequate skills to enter a private school for the deaf, living in the dormitory environment. This heroic effort and accomplishment is not an example of overwhelming success with electromyometric feedback neuromuscular reeducation (although the patient did respond well to irregular treatment with electromyometry, especially when the program was modified to directly deal with dominant developmental reflexes). It is an example of how a patient's lack of communication skills can ***bias an inadequate tester*** and result in a misjudgment of IQ and competency levels. ***The patient's ability to communicate or understand symbolic communication should not be used a yardstick to indicate a person's ability to learn***.[13]

We have shown clinically with both adults and children that cognition and the ability to communicate (speak) are closely related to the ability to voluntarily inhibit (to a given degree) the wrist and finger flexors of the right hand.[14] Work with aphasic patients indicates that a much greater appreciation of the role of neuromuscular development, as it relates to symbolic communication, must be realized before adequate IQ and competency testing of cerebral palsy children can exist (refer to **The Post Cerebral Vascular Accident Syndrome: Aphasia**).

Another example of misjudged IQ is the case of a seven-year old child who had been evaluated by the public school authorities as being a mentally retarded right hemiplegic, spastic cerebral palsy victim. Gross motor and electromyometric evaluations indicated she was a ***spastic*** right hemiplegic patient dominated by ***second stage developmental reflexes***. A question of her acceptability as a patient rose because of a very short attention span, and apparently a poor cognitive ability. These deficiencies were attributed to her ***"mental retardation."*** This premise was soon questioned because of subsequent events. The electrodes for the evaluation had been attached to the forearm skin with adhesive paper collars. When we pulled these collars off at the end of the initial evaluation session, they pulled her forearm hair, which was abundant. She was scheduled for further assessment the following week. Just before her appointment (the mother reported) this "mentally retarded" child asked her mother to shave the involved forearm so that it would not hurt when we had to pull the electrode collars off. Obviously, the child was ***not very*** mentally deficient. She had been able to judge cause and

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12 Initially, this program proved to be inadequate because it did not include the current approach of dealing directly with developmental reflexes.

13 Should a Nobel Prize winner be examined and tested for oral and written competency in a language foreign to that individual, she would undoubtedly be adjudicated incompetent.

14 *While (theoretically) it could be possible that some individuals may have the speech and cognition centers on the right side of the brain, and therefore, have these skills owing to inhibition of the wrist and finger flexors of the left hand, we have not been able to demonstrate this phenomenon clinically.*

Effect and was able to figure out a solution. Noting this, and the patient's ability to inhibit wrist and finger flexors, we had the patient reevaluated by a teacher in special education. He found that the patient had a higher than normal intelligence quotient, but she suffered from ***receptive aphasia*** and had almost ***no self discipline***. It was also suggested that she had developed an appreciation of the secondary gains of being considered mentally retarded (***no responsibility***, ***no discipline*** whatsoever, the privilege of acting out in any environment and the advantage of parental manipulation). He maintained that her facade was maintained by ***feigning a short attention span***. It was not surprising that she had been labeled "mentally retarded" by the school system. The mother was informed, and was requested to begin structuring the child's emotional environment and to try to get her into an adequate educational setting. Through the use of electromyometric feedback and operant conditioning, we saw immediate results. She not only responded neuromuscularly, but also improved her communication skills as inhibition of wrist and finger flexors developed. Skilled speech therapy was instituted to take advantage of developmental speech gains. The following year, she was able to reach out with the involved hand, grasp and release with the wrist and finger flexors almost fully extended, and had almost caught up with her age group in school.[15]

Any evaluation of the intellectual skills of the cerebral palsy population, which does not include an assessment of developmentally linked communication and neuromuscular skills, and equates deficits in communication skills with intelligence, is guilty (mostly by virtue of ignorance or prejudice) of the mishandling of a great number of better deserving individuals.

The development of the ***tactile feedback mode*** may prove to be a great boon in a clinical setting serving the needs of the cerebral palsy patient if the clinical staff has the expertise to apply it. Correctly applied, tactile feedback will allow neuromuscular reeducation of even the very young (three years of age and perhaps younger). The common modes of visual feedback are often totally ignored by the very young, but auditory and tactile feedback can serve as modes that are ***not*** ignored. The patient can perceive both auditory and tactile feedback as ***aversive or pleasurable***. For example, light tinkling bell sounds are often pleasurable, but buzzing sounds are irritating. Tactile feedback applied to the stomach may be irritating, but the same stimuli may be perceived as pleasurable if applied to the upper back. Using the pleasant feedback to facilitate one muscle group, while using aversive stimuli to inhibit another muscle group can make the earlier described neuromuscular reeducation program utilizing electromyometric feedback possible for the very young.[16]

Conclusion

Most neuromuscular disorders that result from an interruption of afferent feedback from the muscle or efferent and/or afferent feedback between supraspinal structures may be

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16 *Auditory and tactile feedback coupled together may make neuromuscular reeducation possible for the blind, while coupled visual and tactile feedback may make neuromuscular reeducation possible for the deaf.*

theoretically susceptible to electromyometric feedback neuromuscular reeducation, if appropriate feedback modes (audio, visual and/or tactile feedback) are utilized correctly. This reeducation is made possible because electromyometric feedback may qualitatively substitute for missing feedback loop pathways on the level that allows previously lost supraspinal coordination by providing a ***new "muscle sense."*** This reeducation must be presented in a manner that is consistent with supraspinal structure requirements for learning, and may entail guiding the system through the five stages of developmental reflexes. The theme around which electromyometric feedback neuromuscular reeducation is built is (1) ***facilitation***, (2) ***inhibition***, (3) ***balance*** and (4) ***function*** or functional use. During the first three phases, electromyometric feedback can be used to develop myoelectric control before visual or palpable contractions can be seen or felt.

It has been demonstrated clinically that this approach permits successful rehabilitation conditions resulting from (1) ***cerebral vascular accidents***, (2) ***peripheral nerve injuries***, (3) ***spinal cord injuries*** and (4) ***damage to the immature nervous system*** (cerebral palsy). Other conditions that might be amenable, at least in part, to electromyometric feedback neuromuscular education are ***multiple sclerosis***, ***muscular dystrophy***, ***dystonia musculorum deformans***, ***Frederich's ataxia*** and (theoretically) any neuromuscular condition that leaves efferent pathways to the musculature intact.