Chapter 2

The Human Neuromuscular Cybernetic System

In an effort to simplify the concept of cybernetic communication in relation to the human nervous system, we have provided an illustration, Figure 9. The boxes labeled Computer A, Computer B, Computer C and Computer D represent combined component parts that provide particular functions in the system. Computer A represents the supraspinal components (see Figure 7) that together provide the mechanism of primary decision-making. Computer A is made up of the cerebral frontal lobes and other cerebral cortex areas, as well as some part of the cerebral motor cortex. Computer A’s function is to make the decision to perform a motor act and communicate this decision to Computer B. Computer B is primarily made up of cerebral motor cortex that is not a part of the primary decision process. Computer B is the initial source of the facilitation of phasic extrafusal muscle activity. Computer B also affects intrafusal muscle activity through its influence upon Computer C. Computer C is made up of the basal ganglia and other subcortical structures. Computer C initially facilitates both tonic and phasic intrafusal muscle activity. Computer D is primarily made up of the cerebellum and the vestibular nuclei. Computer D modifies or reorganizes motor commands from Computers B and C. Computer D coordinates motor commands with data from the proprioceptive sensory mechanisms (i.e., muscle spindles, Golgi tendon organs, skin sensation, optical and auditory impressions, organs of balance, etc.). Computer D’s influence is basically inhibitory. Historically, it has been credited with modifying gross motor activity into refined, precise activity (Llinas, 1975). Computer D does this by “learning” programs of selective inhibition. Learned selective inhibition programs are developed in the cerebellum as the individual experiences trial and error motor attempts to acquire coordinated motor skills. Cerebellar programs influence motor activity through afferent and efferent interconnections between Computer D, Computer B, Computer C, and the Substation Complex. The Substation Complex is comprised of the red nucleus, substantia nigra, olive, subthalamus, reticular formation, and others (see Figures 7 and 8). The substation complex plays a key role in the interrelations between the Computers by providing coordination centers for afferent and efferent data being sent among them.

In the muscle process sequence, Computer A makes a phasic or tonic motor decision and sends appropriate signals to Computer B to implement action. Computer B balances Computer A’s signal against incoming data from Computers C and D and passes signals to Computer C. Computer C signals are relayed through the Substation Complex to Computer D. If the Computer A proposed action is a “learned” motor skill, Computer D responds by sending the appropriate selective inhibition program through pathways to Computer B and C and the Substation Complex. Both Computer B and C interpret a
Figure 9
THE NORMAL HUMAN NEUROMUSCULAR CYBERNETIC SYSTEM
learned program and appropriate messages are sent to the tonic and phasic gamma nerve innervation (see Figure 7) of intrafusal muscle to carry out the program. If the Computer A proposed action is not a learned skill, Computers B and C send respective signals to alpha and phasic gamma innervation and Computer D simply notes the results of the trial and error extrafusal and intrafusal muscle actions and begins to formulate a new program. Tonic and phasic stretch reflexes are utilized as controlling mechanisms for tonic and phasic actions (see Chapter 1).

Alterations of Neuromuscular Cybernetic System by Disrupted Feedback Loops

All components of the neuromuscular cybernetic system depend on feedback loops. Each element in the system receives positive feedback from other elements and in turn responds (either directly or indirectly) with negative feedback. Should any of these feedback loops be disrupted, the entire system may be rendered functionally inoperable. A disruption can occur if one of the supraspinal pathways or one of the pathways between the structures is damaged or destroyed. In humans, nonfatal damage to the supraspinal neuromuscular system most commonly occurs to Computer B (the cerebral motor cortex) and to the afferent pathways from Computer D and C to Computer B (as they traverse the internal capsule). The usual initial response of Computer B to the resultant cessation of afferent signals from computers C and D is a discontinuance of efferent motor signals to extrafusal muscle activity, and an interruption of facitulatory activity to intrafusal muscle from Computer C. Damage of such magnitude usually results in a temporary total flaccidity of the involved musculature. However, after a brief period of several days to six weeks, or more, Computer B and C may adjust to not having incoming negative feedback from Computer D and will begin to send facitulatory motor messages to extrafusal and intrafusal muscle fibers, respectively. Without the coordinating-inhibitory influence of the cerebellum, hypertonous and reflex patterns begin to appear (“spasticity”). The resulting altered cybernetic system is illustrated in Figure 10. In the altered cybernetic system, Computer A may make a motor decision and send the appropriate signals to Computer B. Computer B relays the decision to Computer D. Computer B then prepares to receive a “learned” coordinating program from Computer D or data on the status of ongoing muscular activity to make it possible to coordinate gross motor activity. Because of the lost pathways from Computers C and D, that data never arrives and Computer B must make the choice of taking blind action or doing nothing at all. If Computer B chooses to take action, it simply “facilitates” extrafusal muscle activity (via the corticospinal tract) and activity in Computer C. Computer C, failing to receiving selective inhibition programs from Computer D can only act blindly (via the reticular formation) by facilitating intrafusal muscle contraction and thereby extrafusal muscle contraction (through the tonic stretch reflex). The clinical

Before any supraspinal action can be taken, however, information on muscle status (length, degree of stretch, initial tone, etc.) must be received by the supraspinal structures. Much of this information is transmitted directly from the muscles (muscle spindles, etc.) to Computer D. Muscle status information serves not only as the basis for Computer D coordination of the other computers, but also serves as a primary element in the cerebellar programmed-learning process (see Chapter 1).
picture of this disruption varies from patient to patient as the extent and site of the supraspinal damage varies, and differences in individual response to the supraspinal...
insult occur. If Computer B does not adjust to having no incoming information from Computers C and D, Computer B may “choose” not to act or activate, and a state of functional flaccidity will result. As time goes by, their systems may respond to proprioceptive stimulation (delivered to the supraspinal structures via pathways to Computer C not diagramed here) and begin to generate some activity (especially in the lower extremities) and eventually hypertonus will develop. This response is different from the flaccid response provoked by severe damage to either Computer C or to the supraspinal efferent motor pathways from Computer B (see Figure 11). Damage to these structures will produce a condition which will not allow “muscle tone” to develop because of the consequent lack of Computer C facilitation of tonic gamma motor activity (structural flaccidity). Some such individuals develop a modicum of voluntary phasic extrafusal muscle control, but they are unable to maintain unconscious prolonged muscle contractions to any degree or able to benefit from the motor programming provided to the tonic gamma system by Computer D. In such conditions, even small movements of the involved extremities require continuous fully conscious attention and are therefore of small functional value.

If the peripheral spinal nerve to the muscle is severed, both the motor and sensory nerves must regenerate to both the extrafusal and intrafusal muscle fibers. Clinical experience has led us to postulate that the motor nerve to the extrafusal muscle fibers is likely to reinnervate its end organs, because the reinnervation occurs primarily on the surface layers of the muscle, even if the deeper lying motor and sensory end organs of the muscle spindle fail to be reinnervated, resulting in the neuromuscular cybernetic system resembling the schematic illustration, Figure 12. Theoretically, this condition leaves the neuromuscular system with only the option of contracting extrafusal muscle fibers, without the potential of maintaining tone or taking advantage of Computer D programming provided through the tonic gamma system. This type of singular reinnervation is very rare. Most patients suffering from long-existing (one to ten years) peripheral nerve injury syndromes develop muscle tone after neuromuscular reeducation with electromyometric feedback. It is likely then that in such cases the tonic gamma motor and possibly the phasic gamma motor and the tonic sensory nerves have reinnervated their end organs. However, the annulospiral sensory end organ may have failed to be reinnervated on the equatorial segment of the nuclear bag and nuclear chain fibers by virtue of its complex nature or the depth of tissue penetration required of the sensory neuron for end organ reinnervation. In either case, any muscle spindle sensory deficit will cause Computer D (without outside aid) to allow a state of functional flaccidity to exist because prior “learned programming” is dependent upon constant phasic afferent input. However, Computer D can be “reprogrammed” through electromyometric feedback neuromuscular reeducation to do without phasic afferent input (see Peripheral Nerve Injury, Chapter 3).

The Use of Electromyometric Feedback in the Treatment of Syndromes Resulting From the Disruption of Feedback Loops

Both “spastic” and “flaccid” responses to disrupted afferent pathways are rehabilitation problems. Many schools of thought and techniques have been developed to cope with the
Figure 11

THE HUMAN NEUROMUSCULAR CYBERNETIC SYSTEM WITH DISRUPTED SUPRASPINAL FEEDBACK LOOPS: THE AFFERENT PATHWAYS FROM COMPUTER D TO COMPUTERS B AND C, FROM COMPUTER C TO A, AND THE EFFERENT PATHWAY FROM COMPUTER B AND C HAVE BEEN LOST.
overwhelming task of patient recovery. Some schools have directed their efforts at facilitation, for example, on both “spastic” and “flaccid” extremities, hoping to either redirect reflex-dominated musculature or to initiate motion (Knott and Voss, 1956; Semans, 1967). Other efforts have been directed at evoking and directing developmental
reflexes by forcing the patient through “patterned motion” (the Doman-Delacato
technique, for example) (Doman, Spitz, Zucman, Delacato, and Doman, 1960). Others
have developed techniques of applying various sensory stimuli to evoke desired
neuromuscular response or inhibit undesired neuromuscular response (the Rood and
Ayres Techniques) (Stockmeyer, 1967; Ayres, 1974). Still others have advocated
maintaining joint range, strengthening available musculature, gait training, and teaching
the patient to accommodate to his disability. All of these approaches have proven to be
extremely valuable, and many patients have benefited from their application. However,
each of these approaches has a common limiting element, they all depend upon
changing supraspinal function from without. None provide a means for completing the
disrupted feedback loop and the chances of reprogramming Computer D, through an a
priori trial and error phase, are remote. Electromyometric feedback provides a
mechanism for temporarily completing the disrupted loops through the mirroring of
neuromuscular activity, thereby making the a priori process possible for the
reprogramming of Computer D.

To function properly, the central nervous system is dependent upon continuous positive
and negative feedback between its active components. If the pathway of one of these
feedback loops is damaged sufficiently, it results in certain predictable deficits of
function. It may result in paralysis, paresis or incoordination of musculature. The
resulting deficit varies with the extent of the injury (how much communication was
interrupted), the location of the injury and individual response to insult. What appears
obvious upon inspection (see Figures 11, 12, 13 and 14) is that damage to an afferent
pathway of a feedback loop, although apparently complete, is preferable to damage to an
efferent pathway of a feedback loop. Even though muscular dysfunction results, damage
to the afferent pathway of the feedback loop leaves the potential for motor function
return because facilitory channels still exist. Until the advent of electromyometric
feedback such potential appeared clinically meaningless, and the resulting “spastic” syn-
drome was considered by many to be worse than the “flaccid” syndrome produced by a
lost efferent pathway of a feedback loop. “Spasticity” was often deemed far less
desirable than “flaccidity” because of the accompanying contortion of involved
extremities and the seeming rigidity of involved joints. Early research was able to
demonstrate that the human central nervous system (the supraspinal structures) could
learn to use electromyometric feedback to compensate for missing afferent components
of feedback loops (muscle sense) and to restore a degree of neuromuscular function
(Marinacci, 1960). This discovery is not surprising, since the feedback used was
qualitative (normal supraspinal structures are dependent upon qualitative feedback from
muscle sensory receptors), and the electromyometry (or electromyography) used in such
investigations demonstrated myoelectric change simultaneously as it occurred in the
muscles monitored. It became startling, however, when it was demonstrated that through
such feedback the supraspinal structures could learn to adjust to the loss of the afferent
(and some times efferent) supraspinal feedback loop components and retain the
neuromuscular control after the electromyometric feedback was removed (Basmajian, et
al., 1977).
THE HUMAN NEUROMUSCULAR CYBERNETIC SYSTEM WITH DISRUPTED INFRASPINAL FEEDBACK LOOPS: THE PHASIC AFFERENT PATHWAY FROM THE MUSCLE SPINDLE TO COMPUTER D HAS BEEN LOST
THE HUMAN NEUROMUSCULAR CYBERNETIC SYSTEM WITH DISRUPTED INTRASPINAL OR INFRASPINAL FEEDBACK LOOPS: THE EFFERENT ANDafferent PATHWAYS BETWEEN THE SUPRASPINAL STRUCTURES AND THE EXTRAfUSAL AND INTRAFUSAL MUSCLE FIBERS HAVE BEEN LOST
The mechanism for this adjustment has yet to be fully explained. It may be that the supraspinal structures learn to use other sensory stimuli that follow other afferent pathways as a substitute for the missing muscle spindle “body sense”, or it may simply learn to do without it. Whatever the mechanism, electromyometric feedback serves as an essential “stepping stone” in that process.

Early trail and error exploration for treatment procedures utilizing electromyometric feedback showed that it could not be imprecisely utilized if predictable and expedient results were to occur. Gradually, a theme of application evolved. Essentially this theme involved (1) facilitation of missing or weak myoelectric activity, (2) inhibition of myoelectric hyperactivity, (3) the simultaneous balancing of myoelectric activity from antagonistic muscles (or muscle groups), and (4) functional utilization of gained muscle control (function). This sequential theme had to be applied in the context of the natural neuromuscular developmental sequence (especially in the treatment of supraspinal lesion syndromes). When balancing myoelectric activity from antagonistic muscles, we discovered that both agonist and antagonist muscles had to be monitored simultaneously, with different electromyometers, and that the myoelectric activity had to be fed back to the patient through feedback nodes which could provide differential perception of each muscle. Therefore the selection of feedback modes representing each muscle is important to patient progress. Findings of earlier investigators (Schandler and Gringe, 1975) indicated that there is a hierarchy of subliminal (unconscious) “attention getting” of the various available modes and that tactile, auditory, and visual modes are decreasingly able to draw subliminal attention. We were able to show that these modes used together in combination are often more effective than when used alone and that the most “attention getting” modes should be used in combination to feed back myoelectric activity from the hardest muscles to control. For example, if a patient incurred damage to the afferent supraspinal pathways and eventually developed “spasticity” in her involved upper extremity flexors (dominated by hypertonic, hyper reflexive flexors of the hand, wrist, and elbow), it would be easier to learn to facilitate the extensors than to inhibit the flexors (in the balance phase). In such a case visual, auditory and tactile nodes should be combined to draw special attention to the inhibition of myoelectric activity from the antagonistic flexors while visual and low volume auditory feedback should be used to feed back from agonistic extensor muscles. If, on the other hand, a patient has incurred damage to efferent and afferent supraspinal pathways, and is therefore suffering from a structural flaccidity, the central problem is facilitation of agonist activity. In such a case visual, auditory and tactile feedback should be combined to feed back agonist myoelectric activity, while only the visual node should prove adequate for feedback from the antagonist. If a single muscle is being monitored during the facilitory or inhibitory phases, one electromyometer is effective. In the balance phase, two electromyometers should be used as illustrated in Figure 15.

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2 Inhibition is a voluntary function and must not be confused with an inability to contract. A muscle must be able to contract before it can be inhibited.

3 Patients who have neuromuscularly “plateaued” after two to five years of more traditional physical therapy, have made immediate progress through appropriate applications of electromyometric feedback.
Complicating the treatment program is the fact that previously “learned” programming is not obliterated or erased when supraspinal pathways are disrupted. Computer D still houses the programs of selective inhibition that permitted fine muscle coordination and the performance of habitual skills. The problem is that Computer D can still affect
efferent motor activity through the pathway to the Substation Complex, even without the lost pathways. Computer D may use this ability to try to implement previous programming, but the implementation is uncoordinated and the Substation Complex (red nucleus) simply responds by facilitating biased motor activity (the synergistic patterns) preventing the period of trial and error gross motor attempts so necessary for reprogramming.

Over-control of antagonistic muscle activity (usually over-active synergists) beyond normal limits is a necessary element in inhibiting Computer D interference (Computer D is simply unaware that disruptions of feedback loops has occurred). This over-control, at myoelectric levels, well under visible contraction levels, allows the necessary trial and error period to take place.

Reprogramming is difficult even for “normal” individuals. For example, when a highly trained technician must change a single step in a sequential manual-task routine that prolonged repetition of Computer D programming has been so reinforced that it has become habitual and can be performed “without thinking”, it will take more time and energy to relearn the altered routine than it would to learn a new one. Indeed, the time and energy cost of learning the altered sequence might be so great that it might be less expensive to hire a new technician to learn it. One can easily see how almost unimaginably difficult the task is for a person who must relearn all the sequential routines (primary and accessory muscle coordination) associated with every daily motion.

Developmental and Synergistic Patterns

Special consideration must be given here to the problem of “patterns” which so often appear in disrupted feedback loop syndromes. There are two distinct pattern groups.

Group one is made up of the developmental reflex patterns, most often seen in “spastic” and athetoid post CVA and cerebral palsy syndrome patients (see Table 1). Neurologically, the developmental reflex patterns represent a failure on the part of Computer D (the cerebellum and vestibular nuclei) to balance reflex responses to specific sensory input from movements of the head and body with activity from Computer B and Computer C (see Table 2). Some of these developmental reflexes appear at birth or shortly thereafter, and they may represent a genetic plan to force the initiation of muscle toning, as well as compelling the central nervous system to begin its “programming” process. The new human is motivated to overcome these reflexes by increasing mobility to relieve the discomfort of the “fixed” positions these reflexes involve, but in over coming one reflex another reflex will appear, following a progression of reflexes. This sequential process provides a “push in the right direction” for the developing neuromuscular system. It provides a stage for trial and error Computer D programming, leading to rolling, sitting up, crawling, creeping, standing, walking and complex arm and hand maneuvers. Without this system, neuromuscular development would be tremendously delayed or not occur at all.

Group two are the synergistic patterns that arise from the natural tendency of certain
supraspinal structures (the red nucleus, for example) to affect the extrafusal and intrafusal innervation by **biasing toward facilitation of flexion of the upper extremities and extension of the lower extremities, or extension of the upper extremities and flexion of the lower extremities** (see Table 5). Although pathways from **Computer D** are still available that can affect efferent activity from **Computers B and C** (through the **Substation Complex**), apparently **Computer D** is incapable (unassisted) of coordinating **Computer B and C** output with muscle spindle activity without direct input into B and C. Essentially **Computer D** is unable to implement “learned” motor programs unless all previously constant factors are present. Those constant factors include pathway interplay between **Computer D** and the other supraspinal structures, and they in turn with each other. Thus, **Computer D** is unable to exert its “learned” inhibitory influences on **developmental** or **synergistic** reflex patterns affecting the involved musculature.

For **Computer D** to once again be able to exert itself as a **coordinating-inhibitory** influence, the system must go through another **trial and error** learning period for motor programs to be built, based on an entirely new set of constants. **Reprogramming** will naturally take place as the system attempts to accommodate to **functional deficits**. If an accessory feedback loop is not provided by electromyometry, the new constants will be the **developmental** or **synergistic** reflex patterns. There is, therefore, a time limit for rehabilitation, since reinforcement of the new programs will make them more **habitual**, as time goes on, making them increasingly **harder to “unlearn”**. Electromyometric feedback offers an opportunity to provide a **temporary substitute** for the lost pathways between structures, **making the pattern constants into variables subject to change**. The system can take advantage of the remaining pathways by having **Computer D** learn to coordinate all influences on motor pathways to produce desired responses mirrored by electromyometric feedback. Thus, a stage is set for a **trial and error** period allowing **Computer D** to “reprogram” with a new set of constants that do not include the lost pathways. If guided carefully, this reprogramming potential enables the system to once again work through and conquer the **developmental** or **synergetic reflex patterns**, and achieve **balanced muscular function** (this process is detailed in the following chapter).

To summarize, the task of correcting neuromuscular dysfunction is not a simple matter of facilitation or inhibition of muscular contractions. As indicated above, the central nervous system has a particular way of learning or allowing itself to be **programmed**. It has become clear (through our own trial and error over the years) that the central nervous system will not allow itself to be taught (programmed or reprogrammed) unless the teaching is consistent with its **preexisting design for learning**. The composite of supraspinal structures is restricted to learning the way it was built to learn.

**Treatment of Neuromuscular Dysfunction Without Disrupted Feedback Loops**

If none of the supraspinal or infraspinal pathways have been damaged but a neuromuscular dysfunction has developed as a result of psychological (psychogenic) influences over tonic gamma innervation of intrafusal muscle (Brodal, 1974), as may
occur in *neuromuscular defense mechanism syndromes*, then electromyometric feedback may be useful as a treatment modality.

As the “normal” human nervous system develops, it programs itself to perform tasks that increase its comfort and chances for survival by helping it relate to its environment. The “normal” nervous system learns to make the body walk, run, use its hands and numerous other neuromuscular skills, while learning to communicate (verbally and nonverbally) with the part of its environment which it is most dependent on for survival: *other human beings*. The neuromuscular skills (including communication) soon become so “automatic” they can be performed “without thinking”, or *unconsciously*. Examples of *unconscious* communication include *postures* associated with sitting, standing and walking, as well as head, shoulder, arm and hand *gestures*. Upper extremity, shoulder and head gestures become so habitually a part of conversation that it is not unusual to observe individuals gesturing while conversing on the telephone. Indeed, the body so *unconsciously* communicates emotions that an individual may be totally unaware of the muscle activity that reflects emotion. A person may consider herself “relaxed”, but may actually be *unconsciously* contracting her shoulder muscles enough (because of some *emotional strain*) to cause a *referred pain headache*. To alter such a situation requires the reprogramming of the *unconscious* muscular response programmed in *Computer D*.

Electromyometric feedback allows the patient to explore previously *unconscious* neuromuscular function by augmenting or adding to existing feedback loops with a *conscious sensory feedback loop* (see Figure 16). Neuromuscular dysfunction is demonstrated by electromyometry as a comparative myoelectric imbalance between muscles failing to act in a normal equitably reciprocal manner. Electromyometry provides an opportunity to explore and rebalance neuromuscular activity, through *trial and error*, thereby reprogramming *Computer D* to coordinate influences on *intrafusal tonic gamma innervation* to achieve a more “normal” condition. Experience has shown that the same *theme* of (1) *facilitation*, (2) *inhibition*, (3) *balance*, and (4) *function* operate in the treatment of psychogenic neuromuscular disorders (see *Psychogenic Neuromuscular Disorders*). The use of dual instrumentation is indispensable in the *balance phase of treatment*, although one instrument may be used in *facilitation, inhibition* and *function* phases. The selection of feedback modes is just as important to successful treatment of *psychogenic disorders* as it is in the treatment of disrupted feedback loop syndromes. It has been found that *inhibition* during the *balance phase* is much more difficult to learn than *facilitation*, and consequently, visual and auditory modes should usually feed back myoelectric activity from the antagonist (the muscle to be inhibited) and the visual node is usually sufficient to feed back from the muscle to be facilitated.

In effect, electromyometric feedback can add an extra feedback loop to supplement those that nature has already provided. This extra loop allows the computer system a chance to analyze specific “automatic” neuromuscular functions and to reprogram, by providing a means for *Computer D* inhibition (*over-inhibition* of *over active* musculature), setting the stage for *trial and error* exploration of efferent and afferent neuromuscular interplay.

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4 Talking or coordinated hand function while controlling myoelectric activity from a given muscle may serve to take the patient through the functional stage.
Figure 16

THE NORMAL HUMAN NEUROMUSCULAR CYBERNETIC SYSTEM SUPPLEMENTED WITH ELECTROMYOGRAPHER FEEDBACK