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STUDIES ON NEUROMUSCULAR DYSFUNCTION, XIII: NEW CONCEPTS AND TECHNIQUES OF NEUROMUSCULAR REEDUCATION FOR PARALYSIS

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In patients with paralysis, we are usually dealing with a permanent disability as a residual of disease or injury of a part of the neuromuscular mechanism. In poliomyelitis, for example, the virus infection invades the spinal cord and permanently destroys some of the motor neurons. These neurons degenerate and the muscle fibers they innervate undergo atrophy.

The objectives in treatment of any type of paralysis are to: (1) Obtain the fullest possible recovery of motor function, and (2) Reduce the disability to a minimum.

After a sufficient trial of treatment for recovery of motor function, it may be evident that recovery is insufficient and serious disability will remain. Further practical gain in reduction of disability may then be achieved in some cases through appliances or surgery.

Recovery of motor function occurs spontaneously in certain paralytic conditions. In acute poliomyelitis, for example, as the infection subsides, motor recovery occurs from decrease in inflammation and edema and from restoration of function in neurons which were not destroyed by the virus. In hemiplegia from cerebral hemorrhage or thrombosis, spontaneous improvement in motor function occurs after recovery from the shock of the injury, and from subsidence of the edema and inflammation. In addition, however, spontaneous motor restoration appears to result from a process of compensation by which extrapyramidal pathways take over function from the damaged corticospinal tract.[1]

FACTORS INFLUENCING TREATMENT OF PARALYSIS IN POLIOMYELITIS

Everyone agrees that a destroyed motor neuron in the anterior horn of the spinal cord is lost permanently, since no regeneration of nerve cells is possible after birth. The muscle fibers which were innervated by that motor neuron undergo atrophy and degeneration and eventually disappear to be replaced by fat or fibrous tissue. No therapy directed at the denervated muscle fibers or the destroyed anterior cell will have any effect in restoring voluntary motion. Spontaneous recovery from the virus infection of poliomyelitis renders motor nerve cells, which were damaged but not destroyed, again potentially capable of function. In fact, in our present state of knowledge of poliomyelitis, it is unfortunately true that the whole course of this neurogenic virus infection is beyond the control or influence of the physician. He can neither prevent the disease, nor prevent or influence the virus attack on the spinal cord and

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its permanent destructive residuals; nor, as far as we know, directly influence or control the process of recovery in the infected nerve cells. What, then, is the rationale of applying treatment to restore motor function in poliomyelitis if the process is occurring spontaneously without treatment and if there is no known method of influencing the subsidence of the spinal cord pathology? In fact, Sherman[2] came to the conclusion that treatment did not contribute to recovery of motor function in poliomyelitis, since the results were identical in the treated and untreated groups. This result can best be explained by the ineffectiveness of the particular treatment program applied in these cases. In contrast, almost all other specialists believe that treatment has a very important place in recovery of motor function in poliomyelitis.

Effective treatment is essential to restore function more completely in poliomyelitis even though spontaneous return of motor function is associated with the recovery of the spinal cord from the virus invasion. Treatment can also greatly accelerate motor recovery and render the utilization of the residual muscular power more efficient. The rationale of treatment for restoration of motor function in poliomyelitis is based on the following factors:

1. *Hypertrophy.* Muscular power can be increased by hypertrophy of the muscle fibers. It is well known that heavy work causes muscular hypertrophy associated with marked increase in power and endurance. In poliomyelitis, the innervated muscle fibers can be strengthened as much as possible by hypertrophy, thereby reducing the power deficit of the muscle resulting from loss of some of its muscle fibers through denervation.

2. *Preventing Disuse.* During the long period of spontaneous recovery, the patient without treatment would usually be extremely disinclined to use his paralyzed neuromuscular system. Being unable to walk, the actual daily exercise engaged in by the affected lower extremities would be negligible. For this reason, a secondary drain on motor function is established, which, superimposed on the original organic pathology, renders motor recovery more difficult and may even cause further progressive loss of function. The loss from disuse is not only in atrophy of the inactive muscle fibers, and greater weakness and fatigability of muscles, but also in severe decline in function of the complex mechanisms in the central nervous system for initiation, control and coordination of voluntary motion.

The voluntary motor system is not static but is a highly dynamic mechanism continuously undergoing improvement or decline in function depending on the quantity of activity in the various patterns of motion. Continued activity is essential to maintain the power, range of motion, endurance and coordination of the neuromuscular mechanisms. Also, an adequate level of daily activity is required for each specific motion and group of muscles, since activity in one neuromuscular pattern that excites one group of muscles will contribute to preventing decline of function from disuse in that specific muscle group only, but will have no influence on the process of disuse in another muscle group. Just as complete inactivity of a particular muscle produces loss of function and disuse atrophy more rapidly than mere restriction of activity,

so motor function is improved and the muscle hypertrophies more rapidly the more intensive the daily activity of the muscle.

3. *Development of correct motor patterns.* Patients attempting by themselves to regain the ability to perform complex practical activities in spite of paralysis often run into difficulties because of the development of abnormal patterns of motion which become habitual. The nervous system definitely functions on the principle of taking the path of least resistance. If the hip flexors are very weak and fatigue rapidly, the patient in attempting to walk will develop a pattern of elevation of the pelvis, using the quadratus lumborum, instead of flexing the hip. This is because the synaptic resistance is less in the voluntary motor pathway to the less affected quadratus than to the more paralyzed iliopsoas muscle and the nerve impulses selectively traverse the more available central pathway. As this abnormal pattern becomes habitual, walking will induce no activity in the hip flexors, since hip flexion is excluded from the pattern. The weak hip flexors become weaker and eventually may be completely paralyzed from disuse. Even if gradual spontaneous recovery of the innervation of the hip flexors from subsidence of spinal pathology would have occurred, the abnormal habit pattern may have become so strongly entrenched by that time that the hip flexors will still be excluded from the gait pattern and therefore continue to undergo loss of function from inactivity.

In addition to allowing substitution of stronger for weaker muscles and thereby producing disuse atrophy in the weaker group, abnormal habit patterns have other undesirable consequences. They may progressively strengthen one group of muscles through overuse and weaken another group through disuse, thus increasing muscle imbalance and producing progressive deformity. This is particularly serious in the complex trunk muscles, where progressive deformity of the spine may continue over a period of years associated with progressive muscle imbalance from abnormal habit patterns. It is difficult to explain the frequently observed gradual progression of deformity, weakness and muscle imbalance as a primary effect of polimyelitis, since it is well known that progression of neuromuscular involvement from the virus attack never occurs after the acute stage. Gradual progression of deformity is easily understood, however, as a secondary phenomenon resulting from abnormal habit patterns.

Training of new patterns of motion and new skills is dependent on formation of new functional pathways in the central nervous system, with resistance at the synapses in these pathways decreasing progressively through repetitive activity. Training of a motor skill requires that all of the essential motions which are combined in the new central pathway be carried out in the same pattern many times. Development of new functional pathways is involved, not only in training of motor skills but in the similar process of learning in general and in conditioned reflexes.

Successful repetition of a pattern of motion renders its subsequent performance progressively less difficult and requires less concentration until the pattern becomes automatic and habitual and can be carried out with great

ease. As the resistance in the specific synapses decreases, the tendency for overflow to excite muscles not essential to the smooth performance of the motor pattern also decreases, and the motion becomes progressively more sharply defined and better coordinated. Strongly developed habit patterns can only be changed by great conscious effort and persistence in retraining. However, it has been possible with intensive treatment to develop new habit patterns, for example in walking, which superseded very strongly developed habit patterns of thirty or forty years' duration. Once a habit pattern has been strongly developed and is used a good deal, it becomes a permanent part of the individual. Similarly, once compensatory pathways are developed sufficiently to restore motor function in patients with paralysis and the substitute pathways are used in daily activity, the recovery of function becomes permanent.

4. *Fatigue.* Another factor which encourages abnormal habit patterns is fatigue. A fairly normal pattern of use of the muscles in the lower extremities in walking may be present for the first few steps. As the patient continues, fatigue sets in and he finds it more difficult to include the weaker muscles in the total pattern. With further fatigue, the weaker muscles are excluded from the pattern and substitution is established. If the patient is allowed to continue to walk as far as he can without reference to the detailed gait pattern, he soon develops abnormal habit patterns of substitution of stronger muscles and exclusion of weaker ones. The abnormal patterns set up a cycle of disuse, with progressive loss of power and endurance and atrophy of the excluded weaker muscles. In this way, for example, in a patient with returning function in the anterior tibial muscle, uncontrolled attempts at ambulation associated with fatigue can set up an abnormal habit pattern excluding the anterior tibial muscle and this muscle may actually decline in function from inactivity.

This phenomenon may be the basis of the widely held view that fatigue is extremely harmful to paralyzed muscles in poliomyelitis. It must be pointed out, however, that it is the abnormal motor habit pattern, with exclusion of the weaker muscles and consequent disuse, that is responsible for the decline in function and not directly the fatigue itself. Fatigue in the weaker muscles encourages development of the abnormal pattern. But the actual basis of decline of function of the muscles is inactivity from exclusion from the habitual pattern rather than overactivity and fatigue. Careful observation in a large series of cases has failed to bring out any ill effects of fatigue of individual motions in treatment. On the contrary, the effect of fatigue of individual motions has been definitely beneficial, resulting in increased power, range of motion and endurance.

The exact mechanism of neuromuscular fatigue is not understood. It is known, however, that in voluntary motion, fatigue occurs first and to the greatest extent at synapses in the central nervous system. Fatigue also occurs at the myoneural junction and the last and least affected by fatigue is the muscle itself. The process is rapidly reversible and a brief rest restores function

to the original level. In poliomyelitis, during the acute stage of developing paralysis, neuromuscular activity and fatigue may be harmful to virus infected neurons. After the acute stage is over and the virus infection has subsided, there is no possibility of further damage to neurons by fatigue since the axon and presumably the nerve cell body are relatively insensitive to fatigue compared to the synapses. Furthermore, extensive experience with a large number of cases has demonstrated conclusively in a practical way, that fatigue of individual motions in treatment is beneficial and there has been no evidence in any case that fatigue of individual motions has proved deterimental even a few weeks after the onset of poliomyelitis. Demonstration of the fact that fatigue of paralyzed muscles is not harmful but highly beneficial is important in allowing more intensive treatment with acceleration of recovery of motor function.

5. *Dormant motor neurons.* With new techniques of neuromuscular re-education which excite the motor centers very strongly, it has been possible to demonstrate dormant anterior horn cells in almost every case of poliomyelitis studied, including chronic as well as acute and subacute cases. The demonstration of dormant motor neurons is particularly striking in so-called zero muscles. In these muscles, no contraction at all occurred on attempted voluntary motion. Not only did these muscles contract unmistakably but also produced voluntary motion when the motor centers were strongly stimulated by summation of methods of facilitation such as mass movement patterns, proprioceptive stimulation and rhythmic stabilization. (These techniques will be discussed in detail later in this paper.) The fact that a significant voluntary contraction occurred in these zero muscles indicated that the anterior horn cells were not all completely destroyed but that some of the motor neurons were alive and potentially capable of function but could not be excited by ordinary stimulation. Even after the lapse of years, these cells may still be dormant, since the greatest effort to contract these muscles voluntarily failed to elicit any response. Only by means of strong stimulation of the motor centers through new techniques of neuromuscular reeducation can these dormant motor neurons be excited to discharge and produce contraction of the muscle fibers they innervate. Treatment over a period of time restored function in the dormant cells so that voluntary motion can later be initiated without facilitation.

The fact that dormant anterior horn cells can be demonstrated routinely in cases of poliomyelitis indicates that treatment is necessary to insure that all the motor neurons which have survived the virus attack should be capable of full function in voluntary motion. Only then can the full potentialities for recovery of motor function be achieved. Those anterior horn cells which were affected by the virus but not killed may require stronger stimulation at the synapses to produce excitation and discharge of impulses down to the muscle fibers. More significant is the fact that the internuncial neurons in the spinal cord are usually attacked severely by the virus. Impulses for voluntary motion normally do not pass directly from the corticospinal tract to the motor neuron,

but must always traverse one or more internuncial neurons before reaching the anterior horn cells. Wrecking of the internuncial switchboard by the virus can thereby prevent excitation for voluntary motion from reaching some of the surviving anterior horn cells, rendering these neurons dormant.[3] Because of the multiplicity of interconnections of the internuncial neurons, very strong excitation can reach the dormant motor neurons by a roundabout devious route and have sufficient energy to discharge these motor neurons. After the same devious internuncial pathway has been traversed a number of times, synaptic resistance decreases and then voluntary effort without facilitation or special technique is capable of exciting the dormant neurons. Once these dormant motor neurons are available for excitation through voluntary effort, their associated muscle fibers can be developed to hypertrophy.

6. *Muscle spasm.* Muscle spasm, with its associated tenderness and pain, constitutes a deterrent and disturbing influence on recovery of voluntary motion in poliomyelitis. The muscle shortening leads to contracture, limitation of joint motion and deformity. The muscle spasm and pain limit the range of motion of the antagonist muscles; but spasm also sets up proprioceptive reflexes which inhibit the voluntary contraction of the antagonist.

It is important therefore to eliminate muscle spasm and insure full range of passive and active motion and to develop voluntary power, range of motion and endurance unhampered by muscle spasm and pain. Muscle spasm is treated in a variety of ways: by application of heat in various forms, use of drugs such as curare, neostigmine, myanesin, priscol, and the like, and by stretching. It must be pointed out that intensive treatment for recovery from paralysis is a very effective method for relaxation of muscle spasm and relief from the associated pain. The latter method has the advantage of combating muscle spasm and paralysis simultaneously.

It is also evident, of course, that proper positioning of the patient and extremities is required in cases with severe and extensive paralysis in order to prevent development of contractures.

PRINCIPLES OF THERAPY OF PARALYSIS DUE TO POLIOMYELITIS

Without a basic understanding of the physiology of the neuromuscular system, one cannot be in a position to evaluate the effectiveness of different types of treatment for achieving maximum recovery of motor function. Of the greatest importance in this connection is the concept of the "motor unit." It is generally recognized that muscles do not normally contract by themselves but are invariably excited through their nerve supply. The unit of neuromuscular function is not the single muscle fiber but is the motor unit which consists of the motor neuron with its axon branching and terminating in myoneural junctions and transmitting excitation to over one hundred muscle fibers. The most fundamental characteristic of the functioning of the motor unit is that it acts on the "all-or-none" principle. This means that the motor unit, if excited, invariably brings about maximal contraction of all of its muscle fibers; in other words, if the anterior horn cell is sufficiently excited

to discharge impulses, it can only produce maximal contraction of the muscle fibers it innervates. Excitation, then, is either sufficient to stimulate the anterior horn cell and produce the greatest possible activation of the motor unit or it fails to excite the motor unit at all. It must be emphasized that the muscle fibers do not have a determining influence in this process; in other words, once the excitation of the anterior horn cell is sufficient to produce the discharge of a nerve impulse, the associated muscle fibers have no choice but to contract maximally.

There are only two variable factors in the function of the motor unit:

(1) Frequency of discharge. A low frequency of discharge produces a simple twitch contraction or an incomplete or partial tetanus, while a higher frequency of discharge produces a complete tetanic contraction of the muscle fibers. Actual power of contraction of the motor unit is somewhat greater at a high frequency than at a low frequency.

(2) Condition of the various parts of the motor unit at the moment of excitation. Particularly significant in this connection is fatigue.

The quantity of activity in a whole muscle depends primarily upon the percentage of motor units being excited. When a weak contraction occurs in the muscle, only a small percentage of the motor units are active. As the strength of contraction of the muscle increases, more anterior horn cells are being stimulated and a greater percentage of the muscle fibers contracts. A maximal contraction involves activation of all of the motor units of the muscle. In voluntary motion, the stronger the effort and the resulting muscular contraction, the greater the proportion of active motor units in the peripheral mechanism and, presumably, the greater the proportion of active units also in the central pathway. A second less important factor in determining the strength of contraction of a muscle is variation in the frequency of nerve impulses. A stronger contraction is usually associated with somewhat higher frequency and a smoother tetanic contraction of muscle fibers.

The generally accepted plan of neuromuscular reeducation in treatment of paralysis of various types at the present time, is based on a sequence starting with passive motion; then, gradually over a period of time going through assisted motion, free motion and finally, resisted motion.[4] This system of neuromuscular reeducation apparently grew out of the impression that overactivity and fatigue of paralyzed muscles is harmful and will increase the paralysis. It has already been pointed out that this attitude is erroneous and that fatigue of individual motions is not harmful but beneficial.

This system, which places great emphasis on avoiding excessive activity and with most of the time spent in passive and assistive exercise, is relatively ineffective in the therapy of paralysis. The reason for this is obvious if one considers the physiology of the motor unit. In passive motion there is no activity of the motor units at all. In assistive motion only a tiny percentage of all of the motor units of the muscle are active in a single effort. The motor units that are active, even in assistive motion, are functioning maximally because of the "all-or-none" principle. Since it is the activity of the motor

unit which can lead to such beneficial effects as muscular hypertrophy and overcoming disuse, what conceivable advantage can there be in keeping the vast majority of the motor units inactive in such a therapeutic program? A number of investigators, working empirically in comparing the effectiveness of resistive exercise employing weights with the usual passive and assistive physical therapy program, have found consistently that resistive exercise is much more effective in restoration of function in paralysis.[5, 6, 7, 8] The reason for this difference is that even with such a simple approach as the use of weights for resistance, a much larger proportion of the motor units are excited with each voluntary effort and the total amount of activity in the motor units in a given period of therapy time is greatly increased.

From this discussion, it is obvious that the objective of an effective program of neuromuscular reeducation for paralysis must be the activation of as high a proportion of the motor units of the muscle as possible with each single voluntary effort. In other words, the whole therapeutic program must attempt to make each muscular contraction maximal. By exciting all or almost all of the motor units of the muscle, each exercise will have the greatest possible therapeutic effectiveness. This is true for all of the factors listed previously in the discussion of recovery of motor function in paralysis.

Let us consider these factors individually:

1. *Hypertrophy.* It has been pointed out previously that muscle power can be significantly enhanced by hypertrophy of the remaining innervated muscle fibers. It is evident that hypertrophy will result more effectively when most of the motor units are active with each effort as compared with excitation of only a small proportion of the motor units in a single exercise (as in assistive motion).

2. *Preventing disuse.* Maximal excitation of the muscle involving all of the motor units is obviously more effective in preventing disuse. In addition to preventing disuse more effectively in the motor units, maximal excitation of voluntary motion will also be more effective in preventing disuse in the central mechanisms.

3. *Development of correct motor patterns.* Correct patterns of motion require supervised training in complex activities; however, acceleration of recovery of motor function in paralyzed motions will make it easier to incorporate these motions in a correct total pattern. This can be brought about more effectively by therapy for the individual motions based on maximal activation of the voluntary motion. In addition, it is possible by use of resistive techniques of therapy to carry out training more effectively in formation of habit patterns for complex activities, such as sitting balance or standing balance, by maximal excitation of the pattern of motion. Such resistive techniques for maximal activation of the motor pattern can also be applied in training of essential skills in occupational therapy.

4. *Fatigue.* Recovery of endurance as well as power can be accelerated by use of therapeutic techniques involving maximal excitation of the motor units in individual voluntary motions. Furthermore, with more rapid recovery of

endurance, the problems created by fatigue in training of correct motor habit patterns are also decreased.

5. *Dormant motor neurons.* It has been pointed out previously that dormant motor neurons cannot be excited initially even by the maximal voluntary effort of the patient. Superstimulation of central mechanisms is essential and these methods will be discussed later.

6. *Muscle spasm.* Relaxation of muscle spasm and decrease in tenderness and pain with greater passive and active range of motion can be brought about with the aid of therapeutic techniques of active exercise. Two methods are available to produce this relaxation: (a) Immediately after contraction of a muscle in voluntary motion, particularly maximal isometric contraction from stretch, relaxation of muscle spasm supervenes. In this technique, voluntary excitation is followed by voluntary inhibition which is capable of inhibiting the involuntary reflex muscle spasm. The greater the excitation, the greater the subsequent inhibition of the motor neuron, and resistive therapy with maximal facilitation of excitation will be much more effective than assistive motion in producing relaxation of muscle spasm. As the inhibitory pathway is developed through repeated maximal activation, voluntary relaxation gradually eliminates the muscle spasm and its sequelae. (b) Maximal voluntary contraction of the antagonist muscle, particularly in the shortened range of motion, results in reciprocal relaxation of the agonist with relaxation of muscle spasm. This pathway for reciprocal inhibition can also be developed through repeated maximal excitation and is effective in relaxation of muscle spasm, in increasing passive and active range of motion, and decreasing the associated pain and tenderness. Both direct and reciprocal inhibition are stimulated more effectively in diagonal mass movement patterns than in simple straight motions.

In treatment of poliomyelitis and peripheral nerve injuries, electrical stimulation of the muscles is used a good deal as a method of treatment. This method is particularly valuable in peripheral nerve lesions since direct stimulation of the denervated muscle can help to prevent atrophy during the period when the motor nerve fibers are regenerating. In poliomyelitis, however, electrical stimulation has only limited usefulness. While electrical stimulation of the muscle directly, and of the nerve, can produce contraction, unlike in paralysis from peripheral nerve injury there is no real value in preventing atrophy of permanently denervated muscle fibers. The therapeutic effect therefore is limited to the innervated muscle fibers in poliomyelitis. Stimulation of *all* of the remaining innervated muscle fibers by electrical stimulation from each stimulus cannot be accomplished since maximal stimulation of the nerve or muscle would be too painful. For this reason, the intensity of electrical stimulation must be decreased to a tolerable level which results in excitation of only a small proportion of the innervated muscle fibers. If, through specialized techniques of neuromuscular reeducation, voluntary motion could activate more of the muscle fibers than can electrical stimulation, the former technique would be preferred. Furthermore, it should be pointed out that electrical stimulation of the nerve produces excitation only in the nerve, myoneural

junction and the muscle, whereas voluntary motion activates, in addition, the central mechanisms and the anterior horn cells, which is another advantage favoring neuromuscular reeducation. The stimulation of the central mechanism for voluntary motion is particularly important in developing pathways for excitation of severely paralyzed muscles and for starting innervation of dormant anterior horn cells. As has been pointed out, only repeated activation of the central mechanisms can decrease synaptic resistance and thereby make it easier to innervate the severely involved muscle voluntarily. With the availability of highly effective methods for facilitation of voluntary motion resulting in maximal excitation of the remaining anterior horn cells, electrical stimulation in the treatment of poliomyelitis will play a less important role.

FACTORS INFLUENCING TREATMENT OF UPPER MOTOR NEURON PARALYSIS

Up to now the discussion has been confined to recovery of function in one particular type of paralysis, namely poliomyelitis. In patients with lesions of the corticospinal tract, the peripheral motor unit mechanism is entirely intact, yet paralysis may be severe and some muscles may not respond at all to the attempt at voluntary motion. In patients with lesions of the corticospinal tract there is, to a limited extent, spontaneous recovery of function; for example, in thrombosis or hemorrhage of the internal capsule, paralysis of the opposite extremities may be complete at first but there is gradual recovery over a period of time based on recovery from shock, subsidence of edema and ischemia in adjacent areas and, particularly, compensation through the taking over of function of voluntary motion by extrapyramidal pathways. It is well established that this process of compensation occurs, since in a monkey after complete removal of the motor cortex on one side, there is a great deal of spontaneous recovery of function despite complete anatomical destruction of the affected corticospinal tract. This recovery of function has been shown to result from the taking over of the function of voluntary motion by extrapyramidal mechanisms.[1]

Essentially, upper motor neuron paralysis is related to the presence of large numbers of dormant anterior horn cells which are intact anatomically and are potentially capable of function, but which do not receive sufficient excitation from higher centers to cause them to bring about contraction of the muscle fibers they innervate. The severity of the paralysis in specific voluntary motions depends on the proportion of the anterior horn cells which are dormant; and zero muscles are those in which all of the anterior horn cells are incapable of excitation by voluntary effort. On the other hand, the same motor units can and do discharge in response to other types of stimulation, particularly on the reflex level.

Secondary factors enter into the problem of upper motor neuron paralysis: (1) Prolonged disuse with its associated loss of function and muscular atrophy; (2) Development of abnormal habit patterns of motion based on substitution of the stronger motions and exclusion of the weaker motions; (3) Spasticity caused by the corticospinal lesion, and which is due to the

loss of inhibition of stretch, and other proprioceptive reflexes, interferes greatly with the efficiency of the remaining voluntary motion in the paralyzed patient. Clonus is a related disturbing factor on the same basis as spasticity. Disuse and abnormal habit patterns and spasticity may combine to produce contractures and deformities. Painful limitation of motion from contractures may result in still further secondary loss of motor function.

It has been possible in a large series of cases to demonstrate conclusively that the process of compensation, by which other central motor mechanisms take over function for the damaged areas, can be greatly accelerated and vastly extended through training. The development of these new substitute pathways for voluntary motion is dependent on application of new techniques of neuromuscular reeducation which can stimulate the various motor centers very strongly and thereby produce sufficient excitation at the synapses of the anterior horn cells to discharge the dormant motor units. As in the treatment of paralysis from poliomyelitis, the objective is maximal stimulation of the central mechanisms to excite the greatest possible number of motor units to activity, that is, the objective is maximal voluntary motion with each effort. As the new substitute pathways become more highly developed and the synaptic resistance in these functional pathways is reduced progressively through repeated usage, the conscious effort required to produce motion is progressively reduced. Once the substitute pathways are sufficiently developed so that they can be excited easily in voluntary effort in complex habitual patterns of motion, the restoration of motor function is permanent, particularly if these habitual patterns of motion are used in everyday practical activities.

The techniques of maximal excitation of central motor mechanisms which are capable of restoring motor function in upper motor neuron paralysis, can also reverse the secondary influences which interfere with voluntary motion. These techniques are effective in overcoming secondary disuse from enforced inactivity and exclusion of certain motions in abnormal habit patterns. It was indeed a surprise to learn that anterior horn cells which had not functioned in voluntary motion for as long as 40 years and had been lying dormant during all of that time as a result of severe upper motor neuron paralysis, still had the potentialities of function and in spite of this prolonged disuse could be reactivated and could again function efficiently in voluntary motion. This was even true of dormant anterior horn cells which had never functioned throughout the life of the individual (in our series as long as 48 years), because the injury to the corticospinal tracts occurred before or at birth. Similarly, the atrophied muscle fibers innervated by such dormant motor neurons could not only be restored to useful function in voluntary motion but could undergo hypertrophy with resumption of activity. These specialized techniques of neuromuscular reeducation, besides developing voluntary excitation of previously dormant motor units, were also very effective in bringing about voluntary inhibition of spasticity and clonus. In fact, in many cases the relaxation of spasticity resulting from application of these new methods of neuromuscular reeducation was greater and more lasting than the relaxation of spasticity

produced by any of the drugs which have been tried for this purpose. Furthermore, with repetition of these techniques in daily therapy, new pathways for central inhibition of spasticity have been developed which eventually eliminated the spasticity through restoration of central inhibition. In other words, whereas a drug which blocks the myoneural junction has merely a temporary effect in relaxing spasticity with little or no lasting benefit, the relaxation of spasticity through development of pathways for central inhibition has both an immediate and a lasting effect. Besides, the relaxation of spasticity is accompanied by facilitation of voluntary motion whereas many drugs, while they decrease spasticity, unfortunately also result in depression of neuromuscular function with weakness and fatigue of voluntary motion. There is, of course, no contraindication to the combination of drug therapy and neuromuscular reeducation for relaxation of spasticity.

PRINCIPLES OF THERAPY OF UPPER MOTOR NEURON PARALYSIS

The mechanisms in the central nervous system which direct and control the activity of the motor units are much more complex and variable in function than the "final common pathway." There is also extreme gradation in complexity from the simple reflex arc to skilled voluntary motion. It is evident that one must understand the central mechanisms for voluntary motion which control the firing of the anterior horn cells since these processes are the most important and fundamental for neuromuscular reeducation. The characteristic physiology of the central motor mechanisms is very different from the simple "all-or-none" character of the peripheral motor unit mechanism.

Voluntary motion is often conceived of in terms of impulses starting in the motor cortex, passing down through the corticospinal tract, and, after crossing to the opposite side, passing across several synaptic junctions in the internuncial switchboard to excite the synapses on the anterior horn cells sufficiently to stimulate these neurons to discharge and produce contraction in the associated muscle fibers. This conception is definitely an oversimplification and abstraction. Even simple elementary voluntary motion such as flexion of the elbow involves much more complex mechanisms at various levels of the brain and spinal cord, including both motor and sensory systems. For example, not only is the corticospinal system active in such a simple voluntary motion, but impulses also pass from the cerebral cortical areas through the pontine nuclei to the cortex of the opposite cerebellar hemisphere and through the cerebellar nuclei to relay back to the cerebral cortex and also through the rubrospinal tract to discharge impulses down to the anterior horn cells. This large and complex motor mechanism is responsible for coordination of voluntary motion. With a deficiency in its function, instead of a smooth voluntary contraction, there would be tremor, dysmetria and other disabling defects in voluntary motion. Voluntary motion is also dependent on the proper functioning of postural mechanisms which influence muscle tonus and vary in complexity from the simple spinal two-neuron-arc stretch reflex, through more complex reflexes at the brain stem level concerned with righting and equilib-

rium to the higher center control of these mechanisms, largely of an inhibitory nature. An elementary voluntary motion probably also involves activity in the basal ganglia and is controlled by proprioceptive sensory impulses at the conscious as well as the reflex level. Furthermore, even a simple voluntary motion involves not only contraction of the prime mover but also reciprocal inhibition of the antagonist muscles and activity in fixator and synergic muscles. Besides the functioning of apparently unrelated parts of the nervous system such as those relating to special senses, various aspects of mental function and emotional expression obviously have an influence on voluntary motion. In other words, while particular motor mechanisms have a direct role in initiating and controlling voluntary motion, the function of the entire nervous system is involved in an interplay of influences that affect the process.

The interaction of different motor centers at various levels in voluntary motion should be clear enough from the fact that a similar interplay of centers is involved in even the simplest reflex activity. It has been known for a long time, for example, that the simplest known reflex, the two-neuron-arc knee jerk is influenced by various higher centers including the vestibular mechanism at the medullary level, by voluntary motion such as clenching the fist, and even by a mental process such as solving a problem in arithmetic. It is evident from this discussion that the simple reflex arc and the simple voluntary motor pathway are both oversimplified abstractions. In actual function, the various centers and pathways of the motor system all play a role and influence the process of voluntary motion in a complex, closely interrelated manner.

The characteristic functioning of the central motor mechanisms can be illustrated by the gradation in strength of voluntary contraction of a muscle from minimal to maximal contraction. The determining factor in the percentage of motor units excited is the level of excitation at the synaptic endings on the anterior horn cells. With slight voluntary effort to contract a muscle, the excitation at the synapses of the anterior horn cells is also slight and while excitation is built up at many anterior cells, at only a few does the level of excitation reach the threshold in order to discharge the motor unit. At other anterior horn cells, the excitation is below the threshold level and no response occurs. As voluntary effort is increased, excitation spreads to a larger number of anterior horn cells and while many motor units do not discharge because the excitation is below threshold, a greater number of motor units are excited. In free voluntary motion with gravity eliminated, no matter how strong the effort of voluntary contraction, a considerable percentage of the motor units cannot be excited because the synaptic excitation remains below threshold. With the addition of resistance, the excitation builds up to a higher level and a large proportion of the motor units are activated as synaptic excitation builds up to threshold level at many more anterior horn cells. Still greater excitation can be achieved through summation of several facilitating mechanisms so that more anterior horn cells discharge and a maximal response can be obtained. In fact, temporal and spatial summation have been shown to be

an extremely important factor in determining the quantity of excitation, both in reflexes and in voluntary motion.

Besides the quantitative gradation in excitation at the synapses of the motor neurons which is related to the strength of contraction of the muscle in voluntary motion, there are important inhibitory influences which greatly affect the quantity of excitation and the actual discharge of the motor units. These inhibitory influences come from activity in motor centers at various levels in the spinal cord and brain and converge on the final common pathway. Summation is also a significant factor in inhibition as well as in excitation. Aside from motor centers, inhibitory influences on voluntary motion can also arise from other central mechanisms. For example, it is well known that a new habit pattern of walking is easily disturbed by the person becoming aware that he is being watched. In this example, inhibitory influences from emotional centers decrease the level of excitation at essential synapses of the voluntary motor pattern. The quantity of excitation in the various motor centers is therefore dependent on the interplay of excitation and inhibition from a number of sources, on summation of excitation and summation of inhibition, and on the quantitative relationship of excitation and inhibition at the synapses of the motor neurons. Besides these factors, there are other variables which determine the quantity and character of activity in the motor centers, such as the state of fatigue in the specific synapses at the moment, and the chemical and ionic environment of the neurons such as supply of glucose and tension of oxygen and carbon dioxide.

SPECIAL TECHNIQUES

In working out a program of neuromuscular reeducation for treatment of paralysis, based on achieving maximal contraction of the paralyzed muscles with each effort, a number of techniques and fundamental mechanisms were discovered which are effective in facilitating voluntary muscular contraction. Not only can these facilitating mechanisms be applied individually, but summation of excitation from several mechanisms produces an even greater response. Therefore, in order to produce truly maximal voluntary motion, simultaneous application of a combination of facilitating techniques is used routinely.

Techniques for facilitation of voluntary motion influence primarily the motor centers and the quantity of excitation at the synapses of the motor neurons. Given an excitation at this level sufficient to discharge as many anterior horn cells as possible, the functioning of the peripheral motor unit mechanisms follows automatically and each motor unit functions "all-or-none." The activity in the muscle also, therefore, is an automatic result of the level of excitation built up at the synapses of the anterior horn cells.

The specific techniques of facilitation for maximal voluntary motion in neuromuscular reeducation include: (1) Maximal resistance. (2) Proprioceptive and other reflexes. (3) Mass movement patterns. (4) Reversal of antagonists. (5) Rhythmic stabilization.

1. *Maximal resistance.* When a voluntary motion is carried out against resistance, the voluntary effort of the patient to move is opposed by force

acting in the opposite direction. Maximal resistance for isotonic contraction is the application of an opposing force which is almost equal to, but slightly less than, the power of the motion, so that with the greatest possible exertion, the patient does succeed in carrying out the motion through as much range as possible. For maximal isometric contraction, the resistance applied is slightly greater than the power of the muscle, so that the muscle slowly lengthens as the patient holds a position as strongly as he can against the resistance of the therapist.

The greatest flexibility in application of resistive exercise in neuromuscular reeducation for paralysis is afforded by application of resistance by a physical therapist in individual treatment. The therapist can vary the resistance through the range of motion, if power of the voluntary motion varies in different parts of the range, exactly as needed to maintain a maximal contraction throughout the range. The therapist can also combine other facilitating techniques with resistance in order to obtain more effective treatment of severely paralyzed and zero muscles. The therapist can also assist the patient to increase active range of motion by taking the joint beyond his active range and having him make a maximal isometric contraction. The therapist also develops a close personal relationship with the patient whom she treats daily and can elicit greater effort, concentration and cooperation. The therapist can not only apply resistance for greater motor response in elementary motions, but can also accelerate training of essential patterns such as standing balance, sitting balance, and balance on crutches, by applying maximal resistance in these more complex patterns.

Maximal resistance can also be applied in other ways including carrying out of the motion against gravity if that particular resistance approximately equals the power of the paralyzed muscle. Similarly for more paralyzed muscles, motion with gravity eliminated may result in maximal contraction. Application of weights including dumbells or boots is another method of applying maximal resistance. Use of pulleys with varying weights can also be applied for this purpose. For resistive exercise for the fingers and thumb, a glove can be used with the resistance applied in the form of rubber bands of the correct tension to produce the maximal response. These techniques are more limited than application of resistive exercise in individual treatment by a physical therapist, but are valuable in supplementing the treatment and in treatment of muscles in which the paralysis is not extreme. These techniques can not be applied effectively for severely paralyzed or zero muscles. With these techniques the actual resistance changes as the motion proceeds. In abducting the arm with a dumbell, for example, the resistance is zero with the arm at the side in the sitting position and, as the arm is abducted, the resistance increases to 90 degrees of abduction. Similarly, the resistance changes as the motion proceeds in use of pulleys, because the angle of pull changes with the joint motion.

In our treatment program each patient is given an intensive prescribed gymnasium program for development of power and endurance in elementary

motions using gravity, weights, pulleys, and the like, for maximal resistance. In addition, resistance is applied in complex patterns of activity as, for example, in mat work where the patient is required to sit up, roll over, or do push ups. Similarly, restrictive techniques of therapeutic exercise are applied in complex activities in occupational therapy using friction in wood working and other crafts, working against weight in developing patterns for self care, such as feeding, and working against resistance in gardening.

The mechanism by which resistance increases the power of muscular contraction and the proportion of active motor units, is apparently related to tension in the muscle. When the muscle contracts against strong resistance, the tension in the muscle is increased and proprioceptive stimulation resulting from increased tension facilities the voluntary motor mechanism. Gellhorn[9] demonstrated in monkeys that the muscular contraction in response to stimulation of the motor cortex was greatly increased in that muscle through proprioceptive stimulation by the increased tension resulting from strong resistance or stretching. He also showed that subthreshold stimulation of the motor cortex can result in contraction of a stretched resisted muscle by summation of the weak cortical stimulus with stronger proprioceptive facilitation. Another less significant factor in the mechanism of facilitation through resistance is stimulation of pressure sensation at the point at which resistance is applied, as in manual resistance by the therapist. Even in isolated muscle, greater tension increases the response, but this is a relatively unimportant factor in voluntary motion.

2. *Proprioceptive and other reflexes.* Placing a paralyzed muscle under greater tension by stretching will facilitate the voluntary contraction of that muscle. The mechanism is similar to the proprioceptive facilitation described in the discussion on resistance. This technique is always applied in combination with maximal resistance. By placing the paralyzed muscle in the lengthened position and applying maximal resistance, the therapist can frequently succeed in obtaining voluntary contraction against resistance, even in zero muscles. In other words, the severely paralyzed muscle fails to respond at all in free or assisted motion in the neutral position, whereas the facilitation provided by proprioceptive stimulation is sufficient to discharge some of the motor units. Stretching of the muscle can sometimes be accomplished by a change in position of the body; for example, voluntary contraction of the paralyzed hamstrings for knee flexion is markedly facilitated in the sitting position when the hamstring muscles are stretched, as compared to contraction of the same muscles in the supine or prone position. Sudden stretching of a paralyzed muscle by initiating a stretch reflex and strongly stimulating the proprioceptors, may be more effective than gradual stretching in facilitating voluntary motion in severely paralyzed muscles. Facilitation from proprioceptive stimulation can also be summated with other techniques of facilitation for greater response.

Not only is stretching effective in facilitating contraction in the stretched muscle but stretch of another muscle which is part of the same mass move-

ment pattern can also produce proprioceptive facilitation of a paralyzed muscle. For example, voluntary contraction of the iliopsoas muscle can be facilitated, not only by stretching of the iliopsoas but also by stretch of the anterior tibial muscle which is associated with the iliopsoas in a total flexion pattern of the lower extremity. Gellhorn[9] also demonstrated this phenomenon in studies of electrical stimulation of the motor cortex in monkeys.

Other reflexes can also facilitate voluntary motion. An example of a postural reflex is the tonic neck reflex of Magnus in which rotation of the head results in extension of the upper extremity on the side to which the face is turned and flexion on the opposite side. This reflex, when exaggerated, can be used to facilitate maximal voluntary motion against resistance.

In patients with lesions of the corticospinal tract, the Von Becterew reflex which is a variant of the Babinski reflex, is hyperactive. The passive flexion of the big toe results in a mass flexion reflex involving all joints of the lower extremity. Moderate stimulation of the reflex will facilitate voluntary contraction of the flexor muscles of the hip, knee or ankle and still allow voluntary control of the motion with the ability to contract and relax the muscles at will. This is sometimes useful in initiating voluntary contraction in zero flexor muscles in the lower extremity in patients with upper motor neuron lesions. The after-discharge period of the reflex can also be used to facilitate voluntary contraction of the flexor muscles. After repetition of this procedure over a period of time, voluntary contraction of the flexor muscles can be initiated and carried out without the stimulation of the reflex.

By a similar technique, the gag reflex can be utilized to facilitate vountary contraction of the soft palate and pharyngeal muscles. Resistance cannot be applied in this technique. However, by weak stimulation of the gag reflex simultaneously with strong voluntary effort in contraction of the soft palate such as saying "Ah" loudly, it has been possible to develop through the facilitation of the reflex, voluntary contraction in completely or severely paralyzed palate and pharyngeal muscles.

3. *Mass movement patterns.* Primitive patterns of motion in which combinations of motions are associated in a more complex pattern are present in infants. These patterns are used in normal activity, in work and sports, such as throwing a ball, kicking a football, chopping wood, using a shovel, or swimming. An example of such associated movements in a mass movement pattern is: extension, adduction, internal rotation of the shoulder, extension of the elbow, pronation and ulnar flexion of the wrist. This natural movement pattern is used in throwing a ball. An example of a mass movement pattern in the lower extremity is: flexion, adduction, external rotation of the hip, flexion of the knee, dorsiflexion, inversion of the ankle and extension of the toes. An example of a mass movement pattern of the neck and trunk is: flexion, lateral motion and rotation of the neck and upper trunk to the right, with extension, adduction, internal rotation of the left shoulder in a motion of rolling over to the right from the supine position.

Mass movement patterns are instinctive natural pathways in the central

nervous system and are not learned. The synaptic resistance in these pathways is small. From extensive observation, it is apparent that the fundamental mass movement patterns are all diagonal movements rather than straight movements. For example, in the hip, the natural patterns are: flexion, adduction, external rotation and their antagonists: extension, abduction, internal rotation; likewise, flexion, abduction, internal rotation and their antagonists: extension, adduction, external rotation of the hip. It appears likely that these patterns are the fundamental elementary motions and that straight motions such as flexion, extension, abduction and adduction are derived from the fundamental diagonal patterns. As an example, straight flexion of the hip would be derived from a combination of flexion adduction and flexion abduction of the hip.

Stimulation of the premotor cortex in man results in so-called adversive movements which are primitive diagonal mass movement patterns. Gellhorn[9] has shown in monkeys that stimulation of the motor cortex produces a primary contraction of the triceps and an associated secondary contraction of the flexor carpi. Such functional associations of synergic motions are parts of mass movement patterns. He has demonstrated that the motor points for these associated muscles are adjacent on the surface of the cortex. As has been pointed out previously, proprioceptive stimulation of a muscle in the mass movement pattern facilitates contraction of other component motions of the pattern.

From extensive observations on a large number of patients with neuromuscular disorders, it is apparent that even in patients with severe paralysis from various lesions as of the corticospinal tracts, anterior horn cells or cerebellum, the mass movement patterns are still intact and undisturbed. The patterns are also present in patients with Parkinson's disease. Of all types of paralysis studied, the only patients who showed disturbance and disruption of the natural mass movement patterns were those with athetosis from lesions in the basal ganglia. Since marked damage to both corticospinal tracts leaves the fundamental mass movement patterns intact despite very severe paralysis, and since the patterns only appear to be disturbed in patients with athetosis, it is reasonable to assume that the basal ganglia have a decisive influence in the mechanism of the mass movement patterns and the general impression that these patterns are extrapyramidal in nature is confirmed.

Definite facilitation of voluntary motion, resulting in a greater response against maximal resistance, is obtained in voluntary motion in a mass movement pattern as compared to elementary straight motions. If, for example, the anterior tibial muscle is severely paralyzed but hip flexion and adduction and knee flexion are less severely involved, it is found routinely that maximal contraction of the hip and knee muscles in the natural mass movement pattern of flexion, adduction, external rotation of the hip, flexion of the knee, and the like, results in overflow to the anterior tibial muscle which can then contract much more strongly against resistance. In other words, the quantity of energy discharged in a mass movement pattern will determine the excitation

built up at synapses of a severely paralyzed component motion of the pattern. This principle may be applied in a combination of mass movement patterns. For example, if the left lower extremity is much stronger than the right, combined asymmetrical mass movement flexion patterns of both lower extremities and of the lower trunk will produce a greater response in the more paralyzed muscles of the right lower extremity because the total energy in the pattern is greater than if only the right lower extremity pattern were excited. Similarly, if the upper extremities are relatively uninvolved compared to the lower extremities, flexion, adduction and internal rotation of the left shoulder against resistance will facilitate flexion and adduction of the paralyzed right hip muscles against resistance. Mass movement patterns of the upper extremities can be combined bilaterally symmetrically, as well as bilaterally asymmetrically, and similarly for the lower extremities. Facilitation of voluntary motion through mass movement patterns can also be summated with proprioception and other facilitating mechanisms for greater response. In addition to facilitation of voluntary motion, maximal stimulation of mass movement patterns against resistance results in marked and prolonged relaxation of spasticity, muscle spasm or Parkinsonian rigidity. The basis of this relaxation is apparently the strong activation of inhibitory mechanisms for muscle tonus through excitation of mass movement patterns. Furthermore, it has been found that training of mass movement patterns in patients with athetosis in which the patterns are disturbed, results in a marked decrease in involuntary motion and improvement in voluntary control of the affected motions.

4. *Reversal of antagonists.*

A. Quick reversal of antagonists. In this technique of facilitation of voluntary motion, the antagonistic motion is performed isotonically slowly against maximal resistance and then suddenly the motion is reversed and the agonist is contracted and assisted as rapidly as possible to the shortened position of the muscle, following which the agonist is contracted isometrically in the shortened position against maximal resistance. Quick reversal of antagonists is evident in normal activity such as chopping wood, the boxer's punch, the golf swing, the farmer using the scythe, or the football kick. This method of facilitation summates with mass movement patterns for greater response. It is also useful following immediately after rhythmic stabilization.

The fundamental mechanism of facilitation through quick reversal of antagonists was demonstrated years ago by Sherrington and termed "successive induction."[1] The maximal excitation of the antagonist is followed immediately by strong facilitation of excitation of the agonist. The facilitating effect of successive induction is demonstrable not only in voluntary motion but also in simple reflexes in a spinal animal; the stimulation of the flexion reflex strongly facilitates an antagonistic extension reflex in the same limb immediately afterward.

The specific technique of quick reversal of antagonists is particularly valuable in facilitating maximal contraction in the shortened range of motion and

is therefore effective in increasing active range of motion. It is also beneficial in increasing endurance. A major factor in cerebellar asynergia is deficiency in power and duration of isometric contraction. In patients with cerebellar involvement, therefore, quick reversal of antagonists is a very useful technique, since it results in marked facilitation of isometric contraction.

B. Slow reversal of antagonists. Slow reversal of antagonists is another technique based on successive induction. In this method the antagonist motion is carried out isometrically slowly against maximal resistance and immediately afterward the agonist is contracted slowly isotonically against maximal resistance. The maximal excitation of the antagonist facilitates the voluntary contraction of the agonist against resistance. In this method the facilitation of isotonic contraction is favored. This technique is valuable in conditions such as Parkinson's disease where the primary deficiency is initiation and performance of isotonic contraction. This method is used routinely with mass movement patterns and can be carried out immediately after rhythmic stabilization for greater response. Slow reversal of antagonists is useful in patients with spasticity in facilitating voluntary motion and decreasing spasticity, whereas quick reversal of antagonists would not be applicable because of the block presented by the spasticity in carrying a quick reversal through the range of motion. Slow reversal of antagonists is also useful in facilitating motion which is associated with pain from muscle spasm whereas quick reversal is contraindicated because of the danger that sudden stretching will aggravate the pain.

5. *Rhythmic stabilization.* In this technique, the patient attempts to hold a rigid position of a joint and the therapist alternately and rhythmically applies maximal resistance in one plane, first in one direction and then in the opposite direction in an attempt to move the joint. As an example, the patient holds the wrist rigid in the neutral position and the therapist alternately and rhythmically applies resistance to the radial extensor, then the ulnar flexor, then the radial extensor, and so on. The patient is alternating isometric contractions of the antagonist muscles and as the procedure is continued, the power of the isometrically contracting muscles increases, following which maximal isotonic contraction of the agonist is performed against resistance. This technique of facilitation is combined with mass movement patterns and proprioception for greater response. Also, as a rule, immediately after rhythmic stabilization, quick reversal of antagonists or slow reversal of antagonists is carried out, thereby further increasing the facilitating effect. Rhythmic stabilization not only strongly facilitates voluntary contraction of paralyzed muscles but also effectively inhibits spasticity, muscle spasm or rigidity.

This method also utilizes the principle of successive induction but instead of alternating isotonic contraction there is repeated alternation of the isometric contraction of the antagonist muscles. Patients with many different types of paralysis resulting from lesions of the corticospinal tracts, basal ganglia (including Parkinson's disease and athetosis) and lower motor neurons, responded remarkably well to this technique of facilitation of voluntary motion. On the

other hand, patients with even a slight degree of cerebellar involvement failed to show facilitation from this method or even failed to perform rhythmic stabilization successfully. Patients with combined lesions of other motor mechanisms and of the cerebellum also failed to respond to rhythmic stabilization. In fact, rhythmic stabilization is a sensitive test of cerebellar function. Since patients with various types of paralysis from lesions of different motor centers and pathways all were successful in rhythmic stabilization and showed facilitation from this method, while only patients with involvement of the cerebellum showed a deficiency in rhythmic stabilization, it became apparent that the cerebellum is probably an essential part of the central mechanism for rhythmic stabilization. In other words, just as the extrapyramidal system is activated through use of mass movement patterns, the cerebellar system is excited through rhythmic stabilization and acts as a facilitating mechanism.

NEUROLOGIC IMPLICATIONS

A careful study of a large number of patients with cerebellar disease from multiple sclerosis or familial cerebellar ataxia revealed that the asynergia and the disability correlated closely with the deficiency in carrying out rhythmic stabilization but failed to correlate at all with the degree of paralysis of the muscles. It soon became apparent that the intention tremor, hypotonia, dysmetria, rebound and marked fatigability in these cases were related fundamentally to a deficiency in power, range, and particularly endurance of isometric voluntary contraction of individual muscles. The inability to perform rhythmic stabilization by alternating isometric contraction of antagonists was related to a more basic deficiency of isometric contraction of each antagonist individually. Isotonic voluntary contraction was involved much less, if at all. This hypothesis has been tested by treating patients with the cerebellar syndrome by developing the power, range and duration of isometric contraction of the affected muscles. For this purpose, the quick reversal technique, combined with mass movement patterns against maximal resistance was particularly effective. It has been possible to demonstrate that this procedure improves and may in some cases completely eliminate the cerebellar syndrome and its attendant disability. As the deficiency in isometric contraction improved, the ability to perform rhythmic stabilization also improved and the whole syndrome of asynergia, including intention tremor, dysmetria, rebound, hypotonia and fatigability, was benefited concomitantly. It should be pointed out that this is the first effective therapy developed for the cerebellar syndrome and also the first time that isometric contraction has been implicated as the basic deficiency involved.[1, 10, 11] From these observations, it is reasonable to set up the hypothesis that the fundamental function of the cerebellar hemisphere is to facilitate voluntary isometric muscular contraction.

The function of the corticospinal mechanism appears to be initiation of voluntary motion and inhibition of spasticity. Spasticity is dependent on the stretch reflex and fascilitating postural mechanisms in the reticular formation and vestibular nuclei.[12] It has been pointed out earlier that the basic patterns

of voluntary motion are the mass movement patterns. The integrity of the neostriatum (caudate nucleus and putamen) appears to be essential for the proper functioning of the mass movement patterns which are disturbed in athetosis but not in any other type of paralysis. It appears therefore that normally the corticospinal system functions together with the extrapyramidal mechanism of the mass movement patterns in the performance of voluntary motion.

An analysis of Parkinson's disease suggests the possibility that the affected area in the basal ganglia, the paleostriatum (globus pallidus and substantia nigra), has the fundamental function of facilitating isotonic voluntary muscular contraction. It will be recalled that there is evidence that the cerebellum has the opposite effect of facilitating voluntary isometric contraction. The deficiency in Parkinson's disease, appears to be related to weakness, slowness, fatigability, lack of range and difficulty in initiating isotonic voluntary motion. Isometric contraction is carried out much more strongly and rhythmic stabilization is performed effectively. Improvement in voluntary isotonic contraction through application of mass movement patterns, rhythmic stabilization and slow reversal of antagonists, has significantly improved the disability and has been accompanied by striking improvement in rigidity in Parkinson's disease.

SUMMARY

1. A discussion of the rationale of treatment for restoration of motor function in poliomyelitis indicates the importance of the following factors: hypertrophy, preventing disuse, development of correct motor patterns, fatigue, dormant motor neurons and muscle spasm.
2. An analysis of the physiology of the peripheral neuromuscular mechanism or "motor units" indicates that it is essential to strive for maximal voluntary contraction of the paralyzed muscle for effective neuromuscular reeducation.
3. A discussion of the factors involved in treatment of upper motor neuron paralysis indicates that maximal activation of the voluntary motion is also an essential objective for effective treatment.
4. An analysis of the physiology of the central motor mechanisms involved in voluntary motion which control the excitation of the motor units shows the importance of understanding the complex inter-relationships of different centers at various levels. It is also apparent that the summation of facilitation in the motor centers is the most important factor in neuromuscular reeducation for treatment of paralysis.
5. The specific techniques for facilitation of voluntary motion which have been developed for more effective treatment of paralysis include: maximal resistance, proprioceptive and other reflexes, mass movement patterns, reversal of antagonists and rhythmic stabilization. These methods are routinely used in combination for summation of the facilitating effect.

6. On the basis of observation of a large number of patients with a variety of lesions of the central nervous system undergoing neuromuscular re-education, evidence has been accumulated to support the following hypotheses:
   a. The corticospinal system initiates voluntary motion.
   b. The cerebellar hemispheres facilitate voluntary isometric muscular contraction.
   c. The paleostriatum facilitates voluntary isotonic muscular contraction.
   d. The neostriatum is responsible for the integrity of mass movement patterns.

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