USE OF PHYSIOLOGICAL MECHANISMS IN THE ELECTRICAL CONTROL OF PARALYZED EXTREMITIES

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External electrical control of human extremities with impaired voluntary control has become a field of research where engineers and physiologists meet in collaboration. The goal is to find out electronic and physiological possibilities to replace the insufficient or lost voluntary control by an artifical, external one.

Paralyzed extremity is a medical term denoting loss or impairment of voluntary muscular power of a limb. There exist several forms of paralysis, depending on the site and mechanism of lesion of the nervous system or muscle. For the understanding of essential differences between single forms it is necessary to explain at least elementary scheme of nervous pathways of the human motor system.

The pathway over which nerve impulses governing volitional movement must pass consists of two main components, an upper and a lower. The upper originates in the brain hemispheres, where the bodies of the so-called giant pyramidal cells are situated. Their long nerve fibres descend and terminate in the brain stem and in the spinal cord. This is the upper motor neurone, which through a synaptic connection communicates with the lower motor neuron. The cell body of the lower motor neuron is located in the anterior horns of grey matter, which extend through the whole length of the spinal cord. The nerve fibers which originate from the spinal motor neuron cells emerge from the spinal cord in the ventral roots and are distributed to the muscles of the trunk, neck and limbs by the spinal nerves. Now it can be realized that palsy of a human limb may be due to lesions at two different levels:

at the level of the upper motor neurone, i. e. from the cerebral corfex to the anterior horns of the spinal cord;
or, at the level of the lower motor neuron, or better, motor unit, that is, to the lesion of the grey matter of the spinal cord, of peripheral nerves, neuromuscular junction or of muscle fibers.

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In clinical medicine it is spoken in terms of upper and lower motor palsy. From the electrophysiological point of view, the difference between the two is essential. In the case of the lower motor palsy, electrical excitability of the nervous structures below the level of lesion is lost, while the affected muscles undergo progressive atrophy. This is the reason why electrical control of the paralyzed muscle after a lesion of the lower motor neurone is impossible.

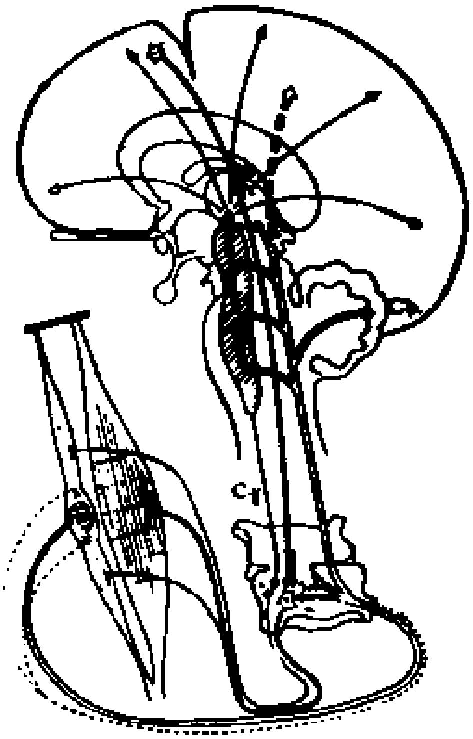


Figure 1. Palliard schematic drawing showing peripherial alpha and gamma motor systems (lower motor neurones) and central part of alpha and gamma systems (upper motor neurones). Extrapyramidal system is intentionally neglected

The state of electrical excitability remains, on the other hand, preserved after the lesion of the upper motor neuron. The failure is in the nervous pathway responsible for governing the lower motor neuron. However, apart from the decreased muscular strength in performing voluntary movements, there exists alteration of muscle tone. According to clinical experiences with hemiparetic and paraparetic patients the exaggerated muscle tone and impairment of reciprocal innervation constitute a much greater obstacle to voluntary movement than does the actual weakness of the affected muscles.

Figure 2 shows a polymyographic recording of motor action potentials in various muscles of both lower limbs of a paraparetic patient.

The patient was asked to extend his left lower limb. Immediately after the beginning of movement an activity of the antagonistic muscle groups arises which freezes the started movement. Furthermore, few seconds later a spontaneous contraction, i. e., a spasm of the same muscles occurs. In addition, an increased activity of muscle groups of the contralateral limb can be seen soon after the beginning of voluntary movement on the left side. This observation demonstrates that an activation and interaction of two neurophysiological mechanisms must be involved during movement of a limb in normal conditions.

1. control of musculuar contraction and
2. control of muscle tone.

For these two different physiological mechanisms there are two different pathways in existence, i. e., the so-called large motor system, consisting of alpha motor neurons, and the gamma motor system, consisting of small-diameter gamma nerve fibers. Both exist in the upper and in the lower-motor pathways.

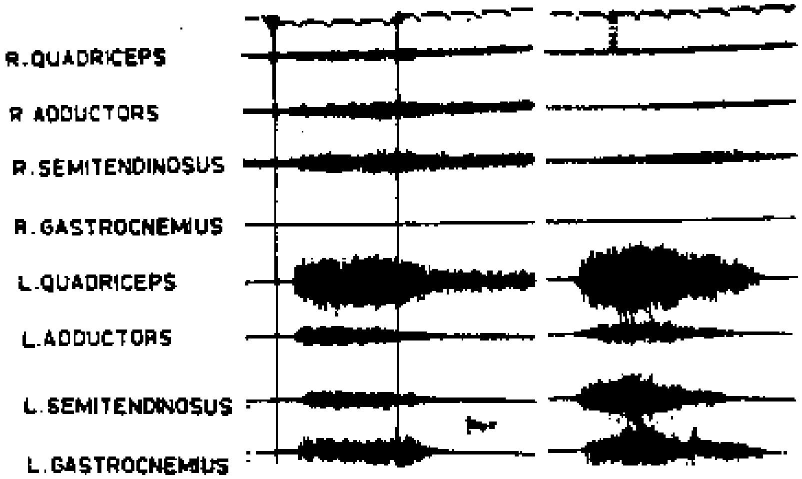


Figure 2. Electromyograph of a patient trying to extend the left lower limb. The movement is attempted between the vertical lines. In the second part of picture, a spasm is recorded a few seconds later

Before further considerations of the behavior of these mechanisms during electrical stimulation of paralyzed limbs, it will be of interest to see how far the practical application of functional electrical stimulation has advanced.

There have been obtained some encouraging results in the functional electrical stimulation of the peroneal nerve for correction of walk in peroneal palsy due to upper motor neuron involvement[1,2] and some were reported to this Symposium by our research group[3]. Some preliminary results of the functional electrical stimulation of extensors of the wrist in the patients with drop wrist have been reported by Long[4] and a similar project has been started in our research group.

Figure 3 shows several phases of an electrically controlled movement of the hand evoked by simultaneous stimulation of the median and ulnar nerves in a hemiparetic patient.

Figure 3. Phases of an electrically controlled movement of the hand evoked by simultaneous stimulation of the median and ulnar nerves in a hemiparetic patient

In all of these examples the same physiological mechanism is used. This is the preserved electrical excitability of the motor nerve fibers supplying paretic or paralyzed muscles. It is depolarization in these fibers that brings about muscular contraction following an electrical stimulus.

Once muscular contraction has bean elicited, the next essential demand is to perform movements of different speed and of wide variability of responses, which is meant by the physiological term »plasticity of function.«

We may succeed in varying speed of the movement to a certain degreee by changing the strength of the stimuli applied to a nerve trunk. The range of these variations, however, is, as may be expected, very poor since the thresholds of single motor nerve fibers for electrical stimulus do not differ much and since all of them respond according to all-or-nothing principle. An additional limitation is that there exist no great differences between the thresholds of the motor and sensory nerve fibers. In other words, there are physiological limitations for the use of the nerve trunk for gradation of muscle contraction, which is normally accomplished by recruitment of varying numbers of motor units at the level of the spinal cord.

From this consideration of the morphological and physiological characteristics of the lower motor pathway and of the consequent limitations of our present concept of functional electrical stimulation (FES) emerges an important fact which we should like to emphasize on this occasion when the external control of human extremities is discussed.

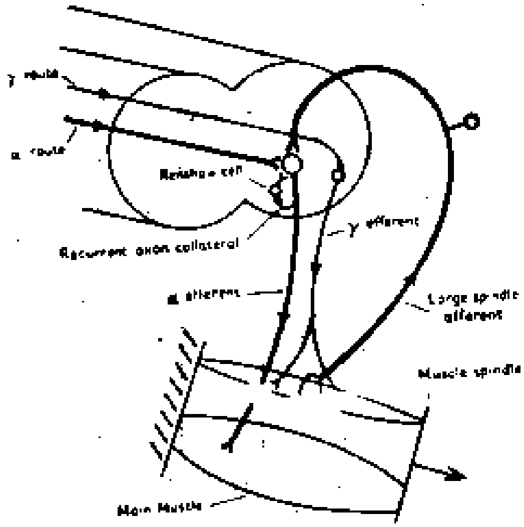


Figure 4. Granit schematic drawing showing the main skeletal muscle. Muscle spindle receptor is inserted in parallel with muscle fibers. This is a sensitive receptor measuring the length of the main muscle. Its sensitivity is adjusted by the thin gamma (upper) motor neurone. The output of the spindle is transmitted to the thick alpha (lower) motor neurone

This is the fact that it is not the upper but the lower motor neuron that controls the muscle. For the lower motor neuron does not receive the complete information necessary to perform a movement from the upper motor neuron; essential elements are added to this information at the spinal level by the sensory neurons, interneurons, Renshaw cells, etc. In other words, the upper motor neuron could, in a certain sense, be regarded as an on/off control system, which puts into action or stops a complicated mechanism of automatic regulatory system, called in neurophysiology spinal reflex integration mechanisms.

A brief consideration of certain spinal reflex mechanisms participating in the motor control of human extremities will show how the fact referred to above applies to the problems of FES, and will point out the present imperfections of this method.

Figure 4 is a schematic drawing by Ragnar Granit, showing a main skeletal muscle, the motor of biocybernetics. A muscle receptor is inserted in parallel position. This is a sensitive instrument measuring the length of the main muscle. Its sensitivity is adjusted by the thin gamma upper motor neuron. The results of measurement are reported to the thick alpha lower motor neuron. So these are two control mechanisms of the lower motor neuron: the upper alpha motor neuron and the peripheral sensory neuron of the muscle receptor. That is, however, not all. The lower motor neuron is, in addition, influenced by other receptors, e. g., pain receptors. This influence is not exerted directly but through an interneuron. So far we have described three of several different control mechanisms which influence the lower motor neuron. It won't be necessary to mention them all. In principle, we can separate the nervous pathways influencing the lower motor neuron from periphery into two categories:

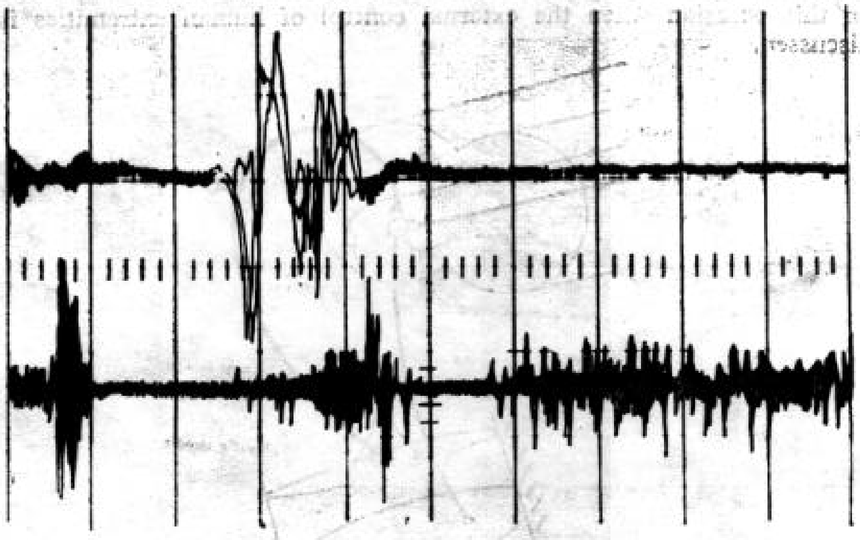


Figure 5. Electromyogram of ankle jerk in a paraplegic patient recorded from tricep surae muscle. Top channel: three jerks superimposed. Lower channel: single jerk. Horizontal scale, upper record: 10 msec/div, lower record: 50 msec/div. Vertical scale: 2 mV/div

1. Those conveying impulses from the periphery of the human body to the spinal motor neuron through a single neuron, i. e., the monosynaptic pathways, and

2. those consisting of a sensory neuron and interneurons, i. e., the polysynaptic pathways.

To summarize, there are three different mechanisms influencing the function of the lower motor neuron:

1. Impulses arriving via the upper motor neuron (both alpha and gamma);
2. Impulses arriving from periphery via the monosynaptic pathway;
3. Impulses arriving from periphery via the polysynaptic pathway.

It will be realized that the behavior and function of these mechanisms in the partly or completely isolated human spinal cord must not be overlooked in FES of paralyzed limbs. We shall refer to some results of our research into the human spinal cord in normal and pathological conditions which was carried out at our Department in collaboration with Peter Nathan[5,6,7].

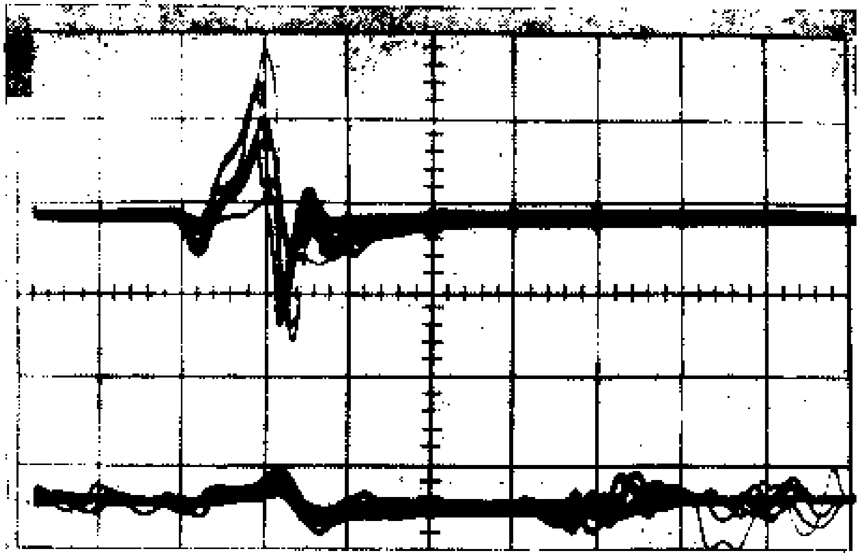


Figure 6. Electromyogram of knee jerk in a paraplegic patient. Top channel: vastus lateralis. Lower channel: gracilis. Five jerks superimposed. Horizontal scale: 10 msec/div. Vertical scale: 0.2 mV/div

Figure 5 shows the activity of the main muscle fibers evoked by stimulation of the muscle receptor — the instrument measuring the length of the main skeletal muscle. The tendon of the triceps surae (i. e., calf muscle) in a paraplegic patient was tapped by a reflex hammer, which made the muscle stretch. Stretch activated the muscle receptor, and the muscle receptor activated the spinal motor neuron; the muscle contracted and returned to the original position. There can be seen a synchronized discharge of motor units in the top channel, starting 30 msec. following the tap. The lower channel shows the same measurement with a slower beam (50 msec/cm), to demonstrate later events. Following the first synchronized, i. e., monosynaptic discharge, several bursts of activity occur that reflect the activation of the polysynaptic, interneuronic system.

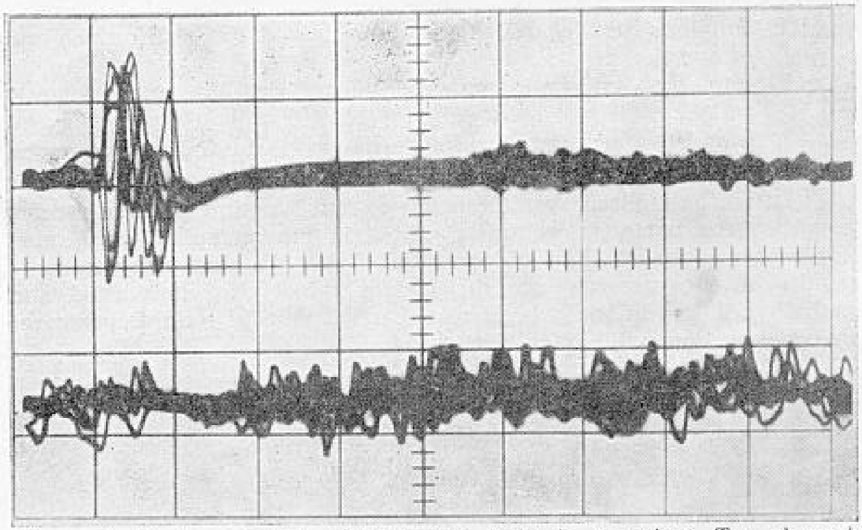


Figure 7. Electromyogram of hamstring jerks in a patient. Top channel: vastus lateralis. Lower channel: semitendinosus. Five jerks superimposed. Horizontal scale: 20 msec/div. Vertical scale, upper record: 0.3 mV/div, lower record: 1 mV/div

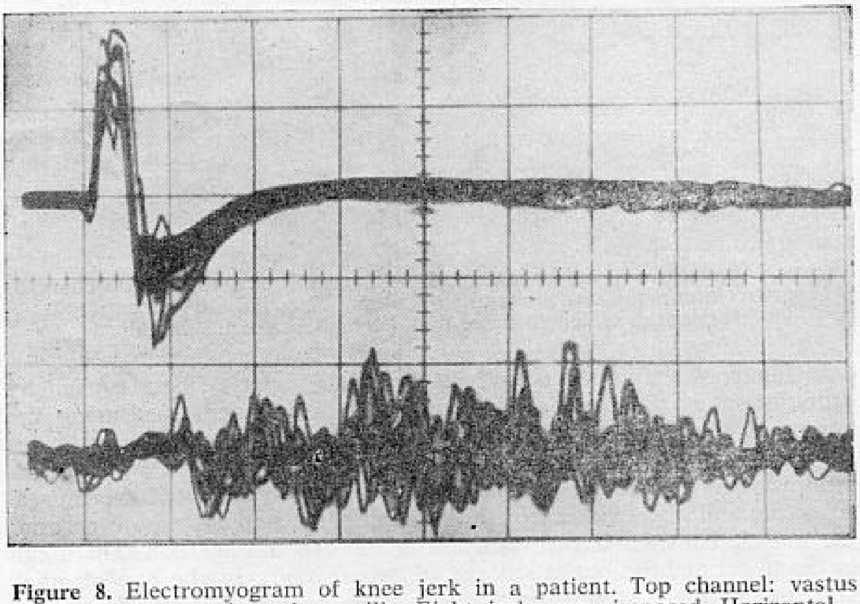


Figure 8. Electromyogram of knee jerk in a patient. Top channel: vastus lateralis. Lower channel: gracilis. Eight jerks superimposed. Horizontal scale: 20 msec/div. Vertical scale: 0.3 mV/div

When a lower motor neuron is activated, it spreads an inhibitory influence through an oligoneuronic pathway on the lower motor neurons supplying the antagonistic muscle groups. This can be seen in Figures 6, 7 and 8.

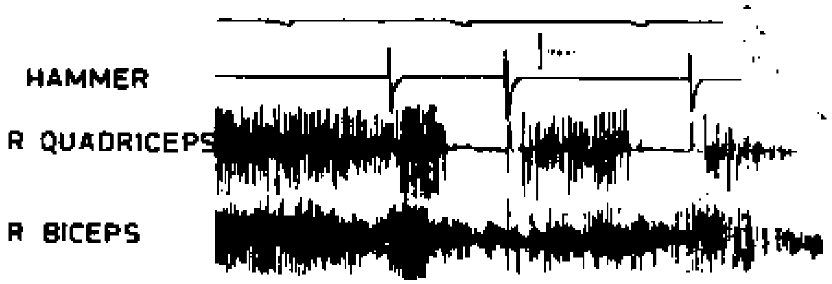


Figure 9. Electromyogram recorded on a 10 channel Schwarzer electroencephalograph. First channel: 1 cps clock. Second channel — event marker indicates tap of patellar tendon by reflex hammer to evoke knee jerk in paraplegic patient. Third and fourth channels show activity quadriceps and biceps muscles before and during knee jerk

Figure 9, on the other hand, demonstrates the activity of the interneuronic system, which can exert an inhibitory or excitatory influence on the motor neurons of the antagonistic muscle groups. Thus, there exists an additional complex spinal neuronic mechanism beside numerous regulatory mechanisms from periphery and from higher

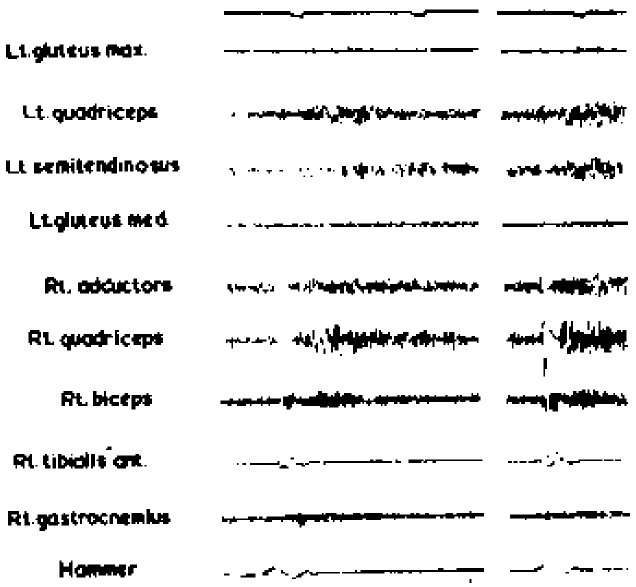


Figure 10. Electromyogram of right knee jerk in the paraplegic patient. First channel: 1 cps clock

levels influencing the lower motor neuron. That is the interneuronic system of the spinal cord, which is responsible for creating the final program of motor activity in accordance with the requests of the higher levels and with the information arriving from periphery. An additional characteristic of this spinal interneuronic system is its ability to influence the lower motor neurons at *different spinal levels.* It is demonstrated in Figure 10, which shows the influence of a stretch of muscle receptors in the quadriceps femoris on the ipsilateral and contralateral muscle groups of the lower extremities.

Thus, the interneuronic system is a functional structure which supersedes the segmental organization of the spinal cord and which is one of the conditions of complex and plastic movement of a limb. The remains of the segmental organization of the segmental organization of the central nervous system actually represent the limits of complex movement.

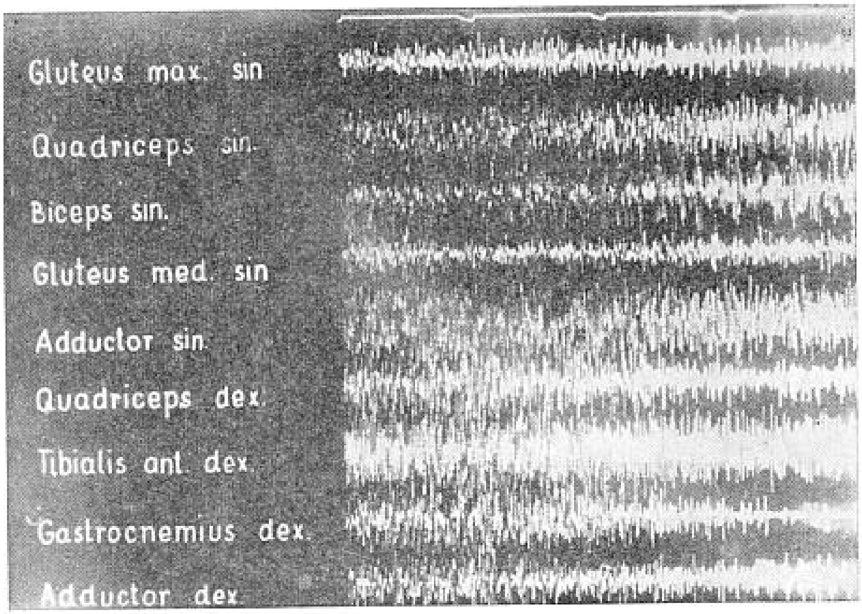


Figure 11. Electromyogram of a paraplegic patient recorded at the end of a spasm evoked by noxious stimulus

Considering this complicated functional organization of the spinal cord, we can realize, how little of this richness can be substituted by our present FES. We know that it is not the best we can do to use the lower motor neuron alone. For stimulation in this very artificial way, we are avoiding the spinal cord and discarding valuable function which it could perform even when partly or completely deprived of the voluntary control of the brain.

The following question is then selfetplanatory: Why not change the site of stimulation? I don't think of stimulation of the cerebral motor structures; on the contrary, I suggest programmed electrical stimulation at the afferent (sensory) terminals of the spinal reflex mechanisms. Why shouldn't we make use of the preserved integration mechanisms of the partly or completely isolated spinal cord, though they represent a disadvantage for our present technique?

Figure 11 shows movement of extremities of a paraplegic patient, evoked by a noxious stimulus. The response is a complex one and chaotic as regards function. It is expected that this response could be changed into a functional one provided the way of afferent stimulation is changed, considering the physiological mechanisms of the isolated spinal cord.

A principal solution of the afferent functional electrical stimulation will be seen in the next figures. Figure 12 schematically shows the principle of the so-called H-reflex, i. e., the reflex response of the muscle following electrical stimulation of sensory nerve fibers alone. The H-reflex is thus an example of afferent electrical stimulation giving rise to a movement due to excitation of the lower motor neuron.

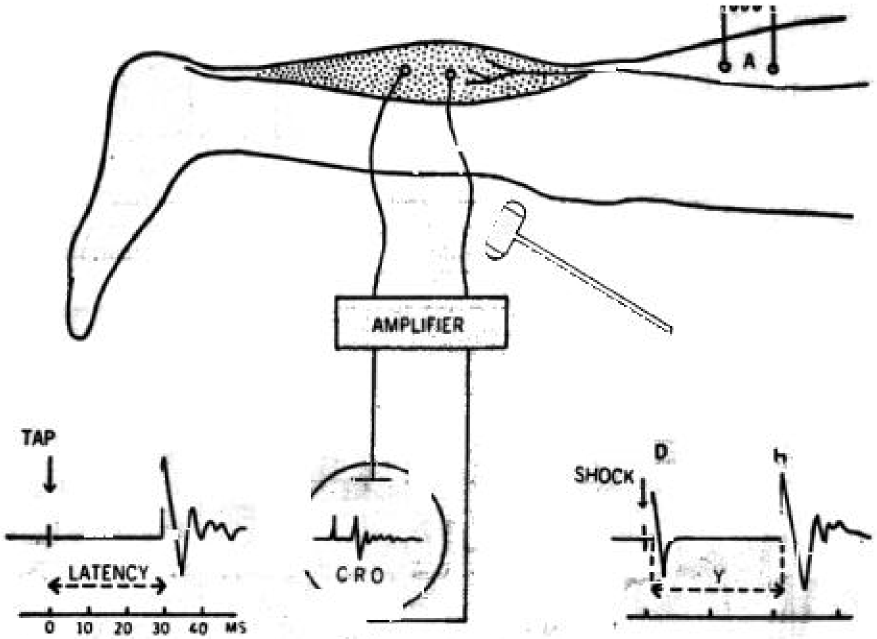


Figure 12. Schematic drawing showing the principle of the so-called H-reflex, i.e. the reflex response of the muscle following electrical stimulation of sensory nerve fibers

Figure 13 demonstrates a so-called H-wave (at a time base of 10 msec. per cm.) and a simulataneous mechanogram bellow (at a time base of 100 msec. per cm.). So it is evidence that an electrically controlled movement can be evoked in this way too. Figure 14 is even more illustrative. The top record is an H-wave, middle is an H-wave preceded by an M-wave, which is the result of the electrical stimulation of both sensory and motor nerve fibers; bottom record is a mechanogram showing both motor responses superimposed.

The H-wave, however, is an essentially segmental response, whereas we need excitation of the interneuronic system. Walking or other

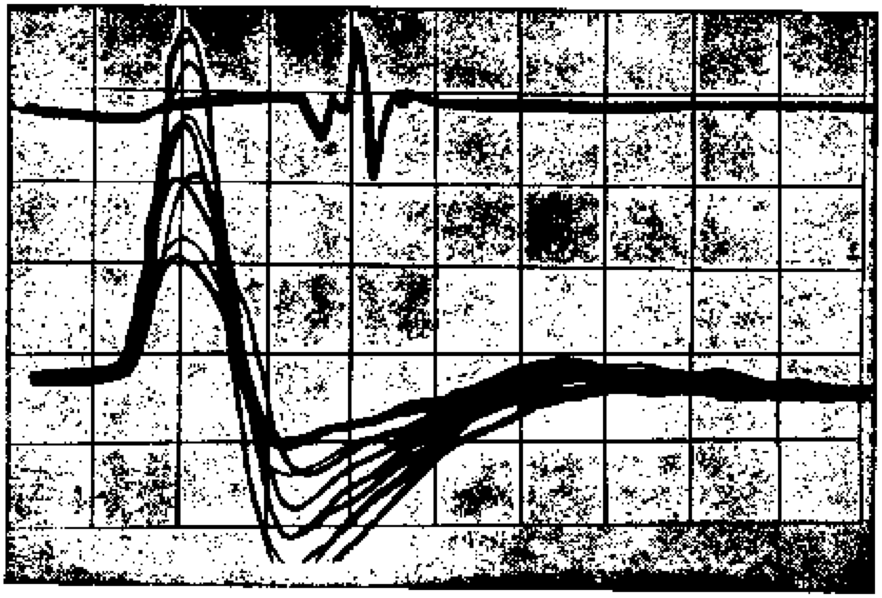
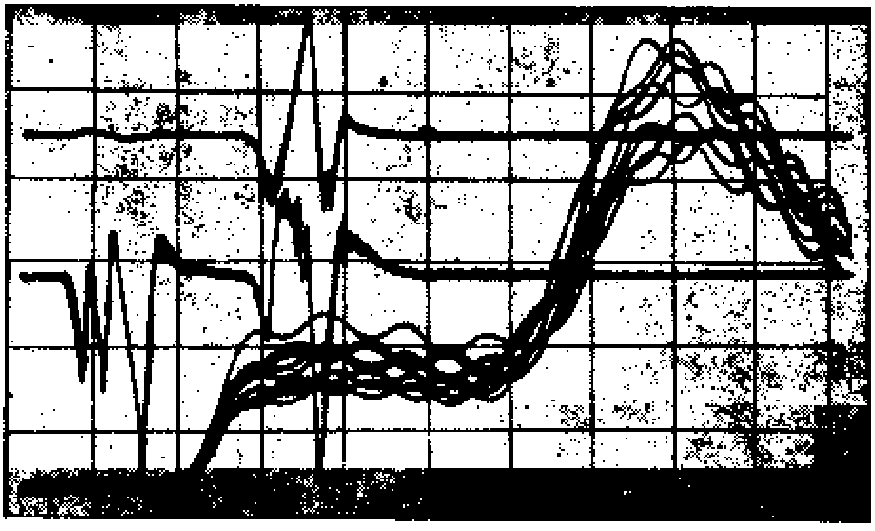


Figure 13. Electromyogram (top record) and electromechanogram (bottom record) of H-reflex of triceps surae. Horizontal scale, top channel: 10 msec/div, lower channel: 100 msec/div



Fidure 14. H-reflex (top record). M-wave and H-reflex (middle record). Electromechanogram of M and H-wave (bottom record). Horizontal scale, top record: 10 msec/div, middle record: 10 msec/div, bottom record: 100 msec/div

skillful performance cannot be imagined without a vital contribution of interneurons. But even that could be accomplished by the H-reflex alone in paraplegic patients, i. e., in conditions of the isolated spinal cord.

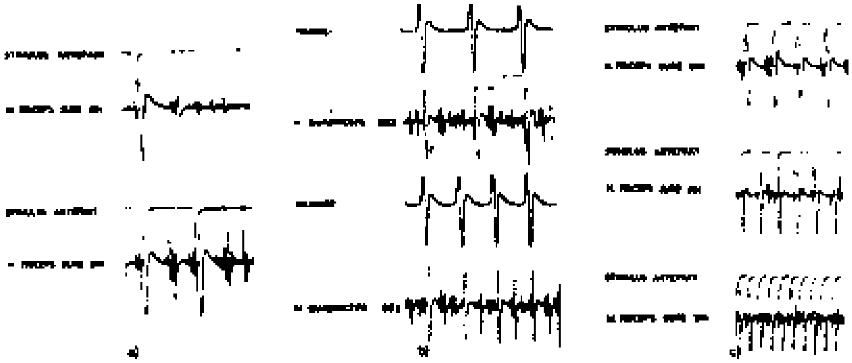


Figure 15. Electromyograms of H-reflex from triceps surae in a paraplegic pat'ent recorded on a 10 channel Schwarzer electroencephalograph. The frequency of stimulation is varied. In (b) a tap by reflex hammer on Achilis tendon is applied instead

Figure 15 shows how, by varying the frequency of sensory stimulation, an H-wave with a simultaneous excitation of the interneuronic system can be obtained. It can be seen how the latency time and amplitude of the polysynaptic spinal response is changing.

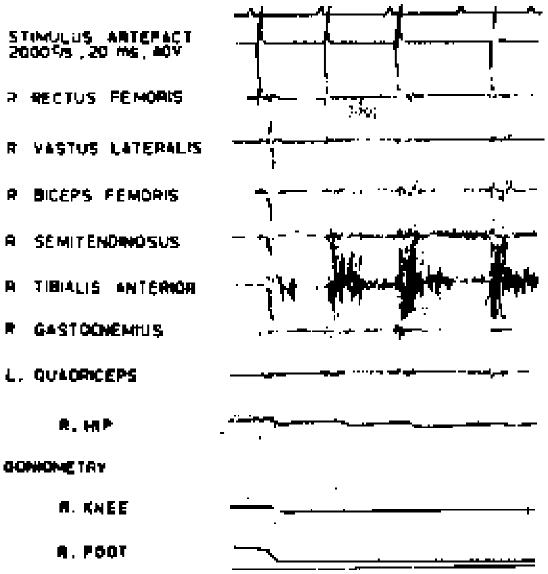


Figure 16. Electromyogram with goniometry of hip, knee and ankle joints after noxious electrical stimuli in a paraplegic patient recorded on 16 channel Kiser electroencephalograph

We can make use of other sensory or afferent pathways in addition. Figure 16 demonstrates a reflex response elicited in a paraplegic patient by an electrical stimulus, which excited only cutaneous sensory nerve endings.

Therefore, there was no direct or monosynaptic response. It would take too long to demonstrate how we could even change the sign of the response in polysynaptic electrical afferent stimulation, although it was performed at one and the same site.

Let us return to the practical application of the FES. It will be realized that we have made use of the afferent stimulation before though not consciously. It can be seen from Figure 17, which is a polyelectromyographic recording of a hemiparetic patient during walk, aided by the functional electronic peroneal brace. It is not difficult to make out how the activity of the peroneal muscle groups is facilitated already during the first electrical stimulation, which was directed only to the motor fibers of the paralyzed muscles. The reflex mechanisms involved here, however, are not the same as those in a paraplegic patient. It is control of peripheral facilitation which was made use of here*.

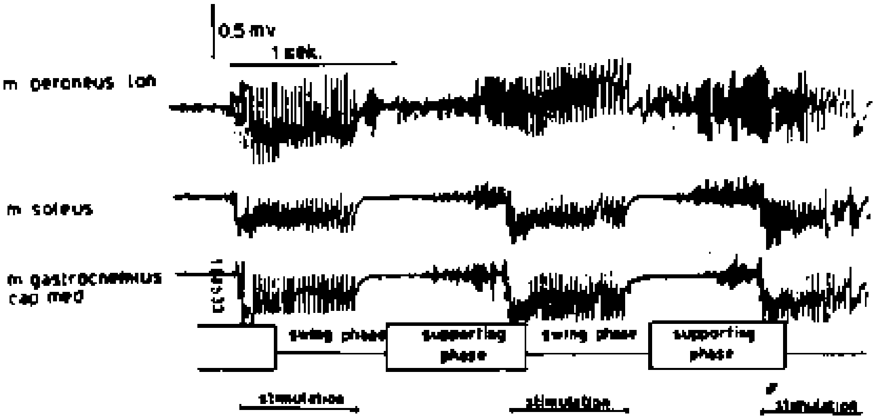


Figure 17. Electromyogram of a hemiparetic patient during walk aided by electrical stimulation, i.e. »functional electronic peroneal brace« recorded on a 10 channel Schwarzer electroencephalograph

To conclude, it must be borne in mind that in electrical control of paralyzed muscles we do not operate muscles only but influence the nervous system too. Considering that we do it, why do we then frequently oppose its mechanisms and why shouldn't we try to make the best use of them in our external control of human extremities?

Summary. An outline of neurophysiological mechanisms essential in volitional movement in normal man as well as their behavior following the upper motor neuron involvement is given.

The physiological limitations of the present technique of the external electrical control of paralyzed extremities are discussed. It is

emphasized that it is not the upper but the lower motor neuron which directly controls the muscle.

The neurophysiological mechanisms preserved in the conditions of the isolated spinal cord folowing a complete or incomplete transverse lesion are described. It is pointed out that it is the interneuronic system which, being interposed between the primary sensory neurons and the lower motor neuron, supersedes the primitive segmental organization of the spinal cord, enabling it to initiate complicated movement. In addition, it is responsible for the creating of the final program of the activation of motor units, in accordance with the demands arriving from the higher motor levels and with the information arriving from periphery.

In order to use these mechanisms rather than suppress them, in the external electrical control of paralyzed limb, a new way of electrical stimulation is proposed, i. e., a programmed afferent stimulation of the spinal reflex mechanisms, which would substitute for the upper motor neuron rather than for the lower motor neuron.

The advantages expected from such a technique would be functional plasticity of the movement integrated at the level of the spinal cord, which we have not succeeded in obtaining with the stimulation of the efferent pathways.

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