REDUCTION OF SPASTICITY BY ELECTRICAL STIMULATION - A CLINIC APPROACH

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ABSTRACT

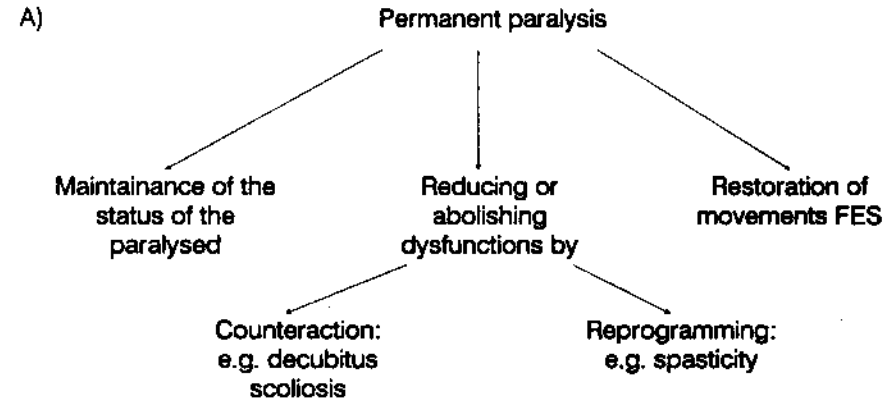
During the last 30 years more and more attempts have been made to influence spasticity by electrical stimulation. One approach started from observations gathered during the application of Functional Electrical Stimulation, FES, using mainly stimulation frequencies between 20-40 Hz. Another procedure developed be Hufschmidt of a neurological point of view uses multichannel stimulation at low frequencies of 0.5-1 Hz, arranging the stimulation sites from central to peripheral. He reported in general a good diminuation of spasticity, but little success in complete spinal cord injured patients.

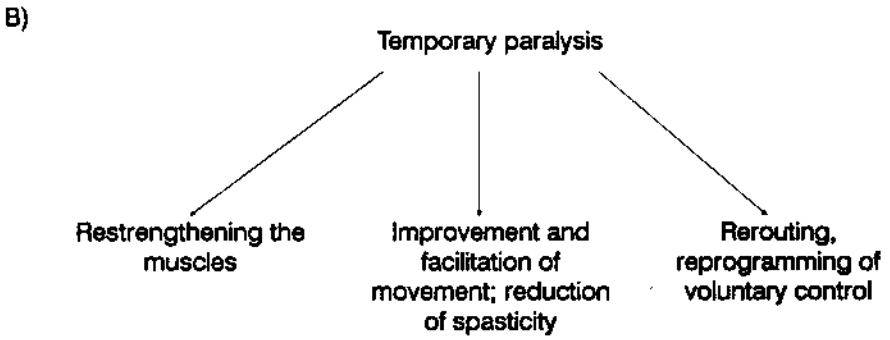
Summing up these observations we conducted a clinical survey study varying the parameters systematically, as stimulus pulse frequency and amplitude, sites of stimulation and their combination in multichannel arrangements, duration and repetition of sessions. We found that treating severe spasticity in most of the cases the spasticity might be diminished substantially when starting out with low frequencies and selecting the stimulation sites carefully. In the course of the treatment stimulus frequency and amplitude could be raises permitting the transition to FES and consolidating the antispastic effect. It has still to be proven whether this is resulting in a permanent reduction of spasticity or not. The results and their implications are discussed.

INTRODUCTION

Applying Functional Electrical Stimulation, FES, to spinal cord patients one is often confronted with a more or less pronounced spasticity these patients are suffering from. The question we have been mostly confronted with in the clinical routine ,besides the application of FES, was the one for the possibilities of deminuation of spasticity. In general after almost 30 years experience in the field of FES and the progressive inclusion of electrical stimulation in the medical therapy one may divide the spectrum of applications a paralysed patient may benefit from into three main areas:

The most prominent side effect of the normal training procedure or the FES induced use of paralysed muscles is maintainance of a nearly normal status. Moderate degrees of spasticity are often reduced during the course of FES application whereas patients with severe spasticity are excluded at all from the FES program.





In general spasticity may develope - and very often does - in all patients suffering from injuries or diseases of the motor sensory system (except the motoneurons in the spinal cord). The spasticity may be of different significance to the patient:

1) It may bother him not at all or not very much and, as a positive side effect, keep the muscles affected at a certain training state;
2) It may (even) support the execution of some functions, e.g. an extensor spasticity of the lower limb may allow the patient to stand for a shorter time span and change from one location to the other.
3) It may progressively reduce not only the range of mobility but the total quality of life so far at his command.

Patients belonging to the third category have to undergo special antispastic treatment as a pharmacological one, the Voita-procedure, or kryotherapie. All of the therapeutic approaches to the problem have their shortcomings and failures. Therefore already some time ago the electrical stimulation was employed for the reduction of spasticity (12, 13). As a side-effect of the application of FES often the reduction of spasticity was observed (7, 14, 19), but it was not possible to deduce a specific procedure. Hufschmidt published in 1968/69 two papers dealing with the Electrothe-

rapy of Spasticity (9, 10). He reported that in cases of spasticity being a consequence of different diseases a preferable multichannel-stimulation with low pulse frequencies, about 0.5 Hz, is very sucessful in reducing the spasticity with the exception of complete SCI. He has apparently the broadest clinical experience with a large number of patients treated. Unfortunately a more detailed and precise evaluation of his results is missing.

After switching off the stimulation often the spasticity releaving effect was persisting for hours to even days. This action is called the "carry over effect". More systematic clinical evaluations showed the same results (1, 3, 6, 17, 18, 19), although an antispastic action of the stimulation was to observe more regularly. The frequencies applied to relieve spasticity ranged of 20 - 50 Hz for muscular stimulation and up to 100 Hz for the stimulation of the afferent nervous pathways. However, if at all only in moderate cases of spasticity the stimulation was found to be successful (Bajd, personal communication).

Our first experience we had with a group of 40 patients with pronounced spasticity primarily being stimulated for the purpose of FES. From the very beginning we used a frequency of 20 Hz. Half of the patients showed no effect with respect to spasticity. The other half had a considerable deminuation, sometimes after a long period of stimulation.

Since three years we devote in cooperation with the Rehabilitation Hospital Karlsbad-Langensteinbach, Foundation Rehabilitation-Heidelberg, more of our activities with electrical stimulation to the rehabilitation of paralysed patients in general. In this connection we have been more often confronted with patients suffering from severe spasticity. Therefore we started a more systematic attempt to reduce spasticity by electrical stimulation.

PROCEDURE

The factors causing spastic patterns at the level of the spinal cord and perhaps also changes in the neuronal organization of the spinal cord and the upper motorsensoric centers are still unknown. The spastic patterns arising, enhancing, and persisting after the complete or partial loss of upper motor control are similar to those of polysynaptic reflexes.

Summing up the results obtained in animal experiments and human evaluations preferential spastic related pathways in the interneuron pool of the spinal cord have to be developed. In case of incomplete paralysis stemming from lesions in the brain this pool in the spinal cord has to be involved too in the process besides the affected higher neuronal structures (4, 5). Direct evidence for spasticity related structural changes concerning synaptic contact or axonal sprouting as well as direct muscular influences was missing (3, 16).

Assuming that spasticity is basically caused by changes in the functional organization of the spinal cord, the observed relief in spasticity and the carry over effect after by the stimulation may be related to a modification of the program of the neuronal network. In this case more specific selceted stimulation patterns and sites might be more effective than the ones used so far. In addition it might be possible to change the stimulation patterns and sites being successful in the beginning as the treatment

progresses, thus preparing the spastic muscle groups for functional use and changing the functional state of the neuronal pool of the spinal cord more persistant. This concept was systematically examined.

TESTING ROUTINE

Initially the antagonistic muscle groups are often chosen as sites of antispastic stimulation, following the principle of antagonistic suppression. Baid et. al. (1) have been successful stimulating the pertinent dermatoms. Hufschmidt (9, 10) stimulated initialle the spastic muscles themselves. Later he changed the regime into a coordinated multichannel stimulation of antagonistically working muscle groups at low frequencies, 0.5 to 1 Hz, and with specific time delays between the channels.

Testing our patients with varying grades and different etiology of spasticity we started off with the stimulation of the direct antagonistic muscles. We proceeded in activating more functional antagonistic muscle groups and finally incorporated functionally agonistic - antagonistic muscles in the motoric control of an entire region. Those parameters were evaluated which turned out to be effective in reducing and/or amplifying spasticity. The efficiency was assessed by the intensity and duration of spasticity after stimulation, its threshold for response, its intensity in resting position and by changes of joint mobility. In order to avoid every possible spasticity eliciting influences, the patient had to be positioned very carefully during the test procedure. The stimulation source used, was a 8-channel, battery operated stimulator with constant current output, rectangular carge compensated pulses up to 1000 sec pulse duration and 100 mA pulse amplitude. The stimulation was employed via surface electrodes.

The following parameters have been modified:

- *pulse frequency 0.1 to 100 Hz;*
- *pulse amplitude up to 100 mA;*
- *pulse width 20 to 500 µsec;*
- *stimulation site: antagonistic and perhaps agonistic muscle groups to the spastic muscles as well as the contralateraly synergetic muscles;*
- *the corrseponding dermatoms or their functionally related areas, respectively;*
- *combination of stimulation sites;*
- *duration of application per session;*
- *stimulation pattern: continuous or intermittent (with rampwise switching on and off) at varying intervals;*
- *repetition of sessions per day, also with regard to the "carry over" effect.*

At first the direct and the nearby antagonists of the spastic muscle groups are stimulated, if possible on both sides of the body. For each of the selected stimulation sites stimulation frequency and amplitude are varied individually and in combination until a spastic movement has started in order to seperate the range of reducing from the range of increasing spasticity.

According to the results of these tests the determined stimulation parameters frequency and amplitude, if possible with a sufficient distance to the limiting values, are used to ascertain the best position for the electrodes. Then their effectiveness is tested in continuous stimulation and is observed in different combinations of the stimulation sites.

Every 10 minutes it is checked wheter a spastic movement can be triggered. In the case of an improvement stimulation is continued until no further improvement can be obtained in the last 10 minute period.

It is important to observe expecially adverse effects of the stimulation. If such effects occur the stimulation parameters and sites are changed.

In case of fatigue of the untrained muscles the stimulation has to be interrupted for an adequate resting period and the muscles have to be trained at first. A stimulation leading to exhaustion of the muscle should definitely be avoided.

The testing procedure is repeated with varying electrode combinations until a satisfying result is obtained or the procedure is terminated if any improvement is missing. In the latter case, provided spasticity is not intensified, the procedure may be repeated for a number of days as sometimes positive effects may occur.

In case of instantaneous relief the duration of the carry over effect is evaluated and the effectiveness of a repetitive stimulation is determined.

As a next step it is investigated whether an intermittent stimulation pattern of 5-10 sec on/off cycles with a slow linear rise and decay of the stimulus is more effective than continuous stimulation.

Finally it is checked whether several stimulation sessions per day give a better effect than only one.

Obviously there aren't any universal rules for the selection of the above listed stimulation parameters. But from our observations and from the experiences of other groups the following instructions for diminishing spasticity by electrical stimulation may be extracted:

- *in many cases the stimulation of the dermatom areas associated to antagonistic muscles is successful, sometimes the direct stimulation of the spastic muscles turns out to be more effective, e.g. in the case of extensor spasticity of the thigh the stimulation of the m. quadriceps possibly in combination with stimulation of the glutei muscles may be effective whereas the stimulation of the antagonists (ischiocrual muscles) fails;*
- *extreme low frequencies may be inefficient compared to those of about 2 Hz; higher frequencies may tend to elicit spasticity;*
- *in the case of brain damage or incomplete SCI lower frequencies of about 0.5 Hz might act better;*
- *stimulation sessions of long duration - longer than 60 minutes - seem to be less effective than shorter ones (30-60 minutes);*
- *the spasticity reducing effect of electrostimulation may be highly dependent on the number of stimulation sessions per day, e.g. in one of our patients, a serve spasticity eliciting effect of stimulation was*

observed when repeatedly applied after cessation of the carry over effect (ca. 3 hours), whereas a single session per day was successful. In another case of severe spasticity of the flexor and adductor muscles of the hip a stimulation programme of 30 minutes, repeated every 2 hours during night, was so effective that daytime stimulation could be reduced to a single session;

- *the stimulation site most effective may change with time. An 5 year old boy was suffering of severe cerebral palsy after a car accident with pronounced spasticity of his right arm. In the beginning stimulation of the left part of the neck and shoulder had a relaxing effect. Later stimulation of the right neck, shoulder and upper arm was more successful.*

ASSESSMENT OF SPASTICITY

Parameters for judging the level of spasticity and the antispastic effect of the stimulation are difficult to define. The spasticity has varying manifestations and often the effected areas are inaccesible for quantitative scaling. Basically one can differentiate between tonic and phasic spasticity. The EMG is used for grading spasticity. Baid et. al. (2) introduced a pendulum test which proved to be useful to measure spasticity of the muscles controlling the knee joint. However, trying to assess very pronounced spasticity, as in our case, which comprises several muscle groups the mentioned test procedures are not applicable: First of all one needs the results instantly during the course of the stimulation. This requires the comprehension of maybe very small improvements in oder to be able to judge whether this particular set of parameters might be useful or not. In addition the assessment is complicated due to the great variations the picture of the spasticity presents.

Therefore one has to establish his own set of parameters maybe together with a relative grading scale similar to the one used by the physiotherapist to assess the muscular status of a patient. We have found the following parameters to be useful for grading the spastic response to a manual trigger:

- *easiness to trigger off spasticity;*
- *speed and degree of contractions of phasic spasticity;*
- *force needed to overcome tonic spasticity;*
- *duration of the contractions;*
- *repetition of the contractions;*
- *spreading to other muscles (groups);*
- *subjective feeling of the patient, altough often of limited use;*
- *in addition to the assessment at longer time intervals: the duration of the carry over effect.*

CLINICAL PROCEDURE

Phase 1:

Determining the stimulation parameters resulting in a sufficient relaxation may require up to a week. During the following days the stimulation scheme is verified or altered depending on the progress of the success and the patient is trained in its application. In addition it has to be discussed with the patient in which way it is possible to integrate it in his daily living routine.

After being discharged from the clinic the patient has to return regularily for ambulant inspections, in the beginning at shorter time intervals. At each inspection it will be checked initially whether the patient is able to handle the stimulation procedure correctly by himself. Following the inspection and the testing of the effectiveness of the hitherto used stimulation scheme the parameters may be modified.

Phase 2:

If the patient is on antispastic medication, this medication is slowly reduced to zero after a sufficient level of reduction of spasticity has been achieved.

Phase 3:

The therapeutic methods utilized so far have a restrictive shortcoming. After their cessation the spasticity returns, at least in cases of severe spasticity.

The main goal of phase 3 is therefore the attemp to reduce the spastic excitability further and by doing so to reprogram the spastic organisation of the neuronal network.

This approach to antispastic therapy is founded on the following considerations: Assuming the validity of the hypothesis that spasticity is mainly caused by the developement of wrong pathways within the spinal cord, it is reasonable to extinguish these pathways - if ever possible -, in order to consolidate the therapeutic effect of the stimulation.

The procedure to do so may be subdiveded into two parts:

1) Raising the frequency and consecutively the amplitude at the stimulated sites thus lilfting the threshold of excitability. If it is possible to proceed to higher stimulation frequencies: These may produce now a better reduction than the lower once. This increase in frequency and amplitude is especially desiserable to strengthen the overstressed and weak antagonistic muscles, and to restore the muscular balance this way. The antagonists may be overstressed and weakend for two reasons. At first the pure overextension caused by the activity of the spastic muscle. This effect may be enhanced by an postural one caused by the spastic activity and/or as a consequence of the injury, e.g. the overstressing of the m. gluteus max. of a handicapped sitting in a wheelchair. Secondly an antagonistic reflex inhibition may occur, which weakens the muscle in addition. - This reflex inhibition may also be the reason for special training properties paralysed muscles sometimes prossess, which have already been discussed above: On the one side exists the group of muscles; which are trainable at a time course comparable to muscles of a not handicapped subject. On the other side the muscles which seem to be

weak already at their initial state and need a much longer training period, but develop finally the same degree of force. The initial weakness of the latter group might be due to an additional reflex inhibition and the longer training period is needed to overcome the antagonistic inhibition.

2) Proceeding to a functional use with inclusion of the spastic muscles into the FES scheme. This phase is now aiming on the true reprogramming of the neuronal structures causing the spasticity and/or are blocking the voluntary control of the higher centers in incomplete lesions.

The stimulation of the spastic muscles with raising frequencies and amplitudes may turn out to be a true test for the level of suppression of the spastic patterns. The inability to stimulate sufficient force might be a limiting factor for some time, in the execution of functions. E.g. in the case of a severe spasticity of the mm. quadricepes femoris these might not be stimulated strong enough to enable the handicapped to stand up, because spasticity is elicited already at lower levels of stimulus amplitude.

In handicapped with incomplete paralysis the reprogramming may be supported besides by stimulation by biofeedback, inclusion in voluntary use, or abolishing of a blocking. (These effects may also be achieved in patients lacking spasticity.) The abolishing of a blocking may also be achieved by stimulation.

The relaxation of a severe spasticity which has already resulted in contractures offeres also the chance for the physiotherapist to conduct his treatment more successfully. The relaxing effect of the physiotherapy supports again the stimulation, thus the one amplifying the other.

We did not observe so far a contraindication of the combination of antispastic stimulation with another therapeutic procedures, but did not explore this area specifically. The reduction of the antispastic medication has already be dealt with above.

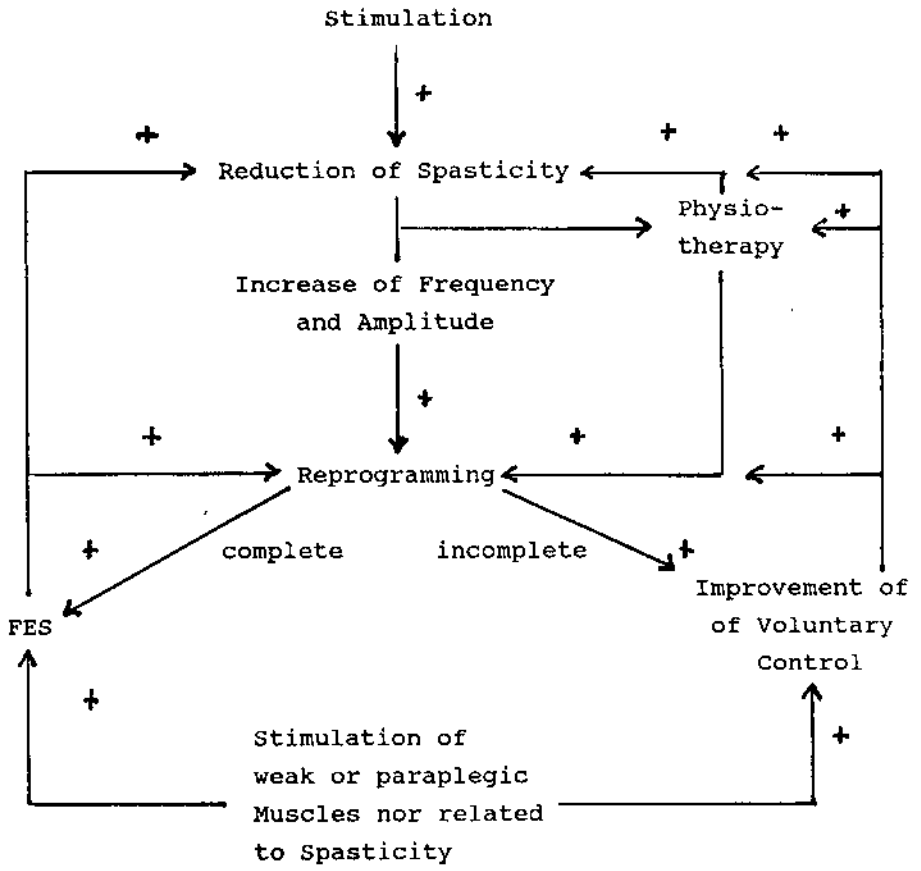
The application of antispastic stimulation of phase 3 requires also periodical routine check-ups in order to:

- *modify the stimulation program in acoordance with the improvements abtained;*
- *determine the time of proceeding to functional stimulation if intended;*
- *prevent potentially unfavourable futur developements.*

DISCUSSION

The purpose of this introductory study was to develop a methodological access to the treatment of spasticity on a broader scope to create a base from whereon quantitative clinical evaluations may be started. We treated so far about 30 patients of different etiologies primarily with antispastic stimulation. This group consisted mostly of spinal cord injured patients but includes also some with hemiplegia as well as infections and degenerative diseases as Coxsackie A virus or MS and Adenomyeolopathey.

In all but one incomplete patient the spasticity has been diminished at different grades depending upon the time of treatment, but at least to such an extent, that the spastic became managable again. In cases of very severe spasticity is may take about



one year before the spasticity is permanently reduced to such a level that it is of no more importance and two years that the stimulation has not to be executed daily.

Taking into account the results of other groups working in the field of FES it has to be realized that in most cases spasticity is influenced positively by electrical stimulation. Actually we cannot predict which specific treatment will be successful for the different types of spasticity. This may be due to lack of method in application. A wrongly chosen stimulation frequency could e.g. be the cause of a great number of failures. Especially in cases where the stimulation was applied at frequencies of 20 Hz and more there was always a certain number of unsuccessful trials (11). This is also the same in the case of the 40 patients with severe spasticity, who were stimulated for FES by us with a frequency of 20 Hz from the beginning. Half of this group showed no effect with respect to spasticity, while spasticity of the other half of the group diminished considerably at different stages partly only after a long time of stimulation. Hufschmidt seems to have the richest clinical experience, and he reports good success in stimulating spasticity with low frequencies (0.5 - 1 Hz) in all types of diseases except

spinal cord injuries. Unfortunately it is difficult to gather exact information from his publications about the number of cases successfully treated, the amount of reduction of spasticity achieved and the continuation of the success after suspending stimulation. Moreover, Hufschmidt as far as we are informed, did not proceed to higher frequencies to achieve functional use of the muscles. The still short duration of treatment and the inhomogenity of our own small group of patients does not yet allow final conclusions.

As far as the etiology of spasticity and the influence of stimulation on it is concerned there are, up to now, no reliable models. As discussed in (22, 23) a good working hypothesis is assuming that within the spinal cord preferential circuits along refley pathways are established if motor control and coordinating functions of the upper motor centres have been lost. These circuits tend to stabilize themselves on the basis of self-organizing effects of the interneural cell pool as maybe described by so called synergetic models by Haken (8).

Afferent pulse patterns as induced by the periodic stimulation sequences seem to disturb the newly established neural organisation and to inhibit it. The carry over effect may be founded on the same mechanism. In a addition the often multisegmental organisation of the spasticity in connection with the observation that these patterns may be changed by applying FES for a long time are pointing in the same direction.

If such a mechanism exists the functional reprogramming of the contraction pattern is the best way to cure spasticity permanently.

However, the question arises if a sophisticated stimulation pattern - an "intelligent afference" - could induce more effectively the desired reorganisation of the interneural network or even cause a distinct stimulus of the activation of certain functional strucutues. For the time being this is highly hypothetical.

There is possibly an indication of a potency of programming of rather simple movement patterns in the spinal cord. It was demonstrated that spinal cats despite their transsected spinal cord learned to perform coordinated movements like walking on a belt (15), and with Vojta therapy it is possible to elicit movements in completely paralyzed persons by pressing on defined cutaneous areas (20). The stimulation patterns being used so far allow only a vague idea of how to gain access to the nervous networks in the spinal cord and the higher sensormotor centers. In addition it is not yet known how much these higher centers really participate in the generation of spasticity. Judging from the spastic deminishing and the unblocking effects of stimulation it might very well be, that the pathways of the higher sensomotor centers are more likely to be dislinked from the spinal cord level in these disorders than actively participating in it.

However it becomes more and more evident, that electrical stimulation might be sucessfully used to diminish spasticity even in cases where other therapies fail. But the patient has to prove great engagement to carry the treatment through effectively.

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