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EFFECTS OF ELECTRICAL STIMULATION ON SPINAL SPASTICITY

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ABSTRACT. **Seven spinal cord injured (SCI) patients with clinical signs of knee-joint spasticity were tested with the Wartenberg pendulum test and an electrogoniometer. All patients were subjected to four channel rhytmical electrical stimulation of the knee muscles for three consecutive days. In five patients some improvement of spasticity was achieved. No increase of spasticity was observed in any patient. Combining results from two separate but similar studies it is contended that about one-half of randomly selected SCI patients with knee joint spasticity might benefit by electrical stimulation.**

Key words: Spinal cord injury, spasticity, electrical stimulation.

The use of electrical stimulation for relieving spasticity can be traced back to Duchenne in 1871 (14). However, even today there does not seem to exist a well documented rationale for electrical stimulation of spastic extremities. Therefore this treatment procedure is either ignored (3) or discussed in broad terms. Thus Schriber (18) states that for spinal cord injured (SCI) patients "electrical stimulation is of little therapeutic value and may even make spasticity worse". Other authors report mostly about beneficial effects but the specific techniques and stimulation sites do not seem to be of primary importance (1, 4, 6–11, 13–15, 17–19, 23–25). One of the present authors (P. Hufford) encountered after electrical stimulation of a paraplegic patient the occurrence of dysreflexia. Patients who might develop dysreflexia should therefore be carefully monitored (blood pressure etc.).

On a normal neuromuscular system electrical stimulation exerts little influence in changing the excitation—inhibition equilibrium. In systems with a deficit in supraspinal volitional excitation (paresis), stimulation can improve volitional control (5, 16, 22). In spastic extremities which lack adequate inhibition, stimulation might act inhibitory. Thus quite specific and selective effects are achieved through rather generalized, non specific stimulation. A hypothesis attempting to explain these phenomena has been proposed recently (21) but experimental proof is still lacking.

METHOD

Four channels of cyclic stimulation has been applied to the flexors and extensors of both knee joints in seven patients with spinal cord lesions and clinical signs of spasticity in the knee joint.

During the first half period of activity (5 sec) stimulation activated the flexor of one leg and the extensor of the other leg. In the second half period the remaining two extensors and flexors were activated. It was postulated that such a stimulation sequence would produce reciprocal movements similar to the ones in gait and might favorably influence the neural reorganization at the spinal cord.

Stimulation currents were obtained from two dual channels Respond (*R*) units produced by Medtronic, Inc. The stimulators were triggered from an external clock circuit which enabled a variable cycling rate. The typical period of the cycle was 10 sec. Thus, for 5 sec the ipsilateral quadriceps and the contralateral hamstrings were stimulated and the next 5 sec the contralateral quadriceps and the ipsilateral hamstrings received stimulation. The stimulation parameters included compensated monophasic square pulses at a rate of 30 pulses per second with a 300 μsec pulse duration. The current amplitude was set to approximate 100 mA with the rise time of the pulse train set to 2 sec.

Stimulation was applied through carbon rubber electrodes 5×10 cm in size. Between the electrodes and the skin a conductive electrode jelly (Spectra 360) was applied. The electrodes were placed over motor points of the quadriceps and hamstrings muscles.

A typical experiment for each patient lasted five days and was always performed in the afternoon. On the first day (usually Monday) the patient was positioned in a semiprone position on a table so that the legs could freely swing about the knee joint (Fig. 1). Electrodes were attached to one leg only (usually the left one). In addition to the two pairs of stimulation electrodes a neutral ground

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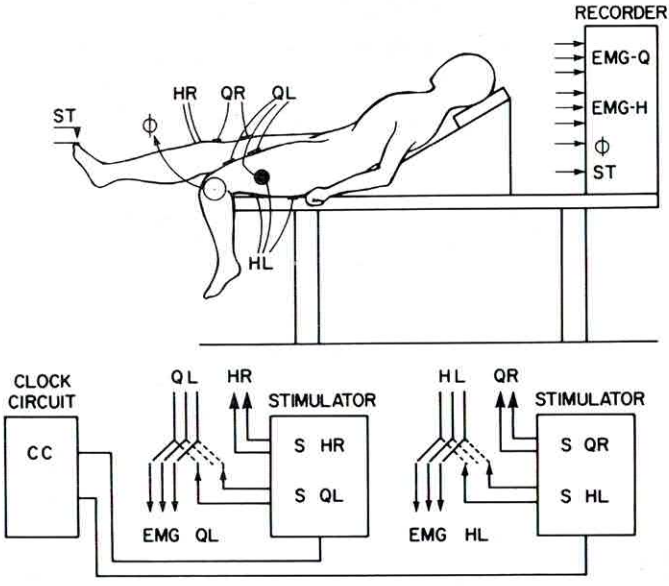

Fig. 1. Measurement of spasticity and stimulation of SCI patients. *QR, QL*: signals to or from right and left quadriceps. *HR, HL:* signals to or from right and left hamstrings. *EMG-QL, EMG-HL:* myoelectric activity of the left quadriceps and hamstrings. Φ: angle of left knee joint. *ST:* start signal. *SHR, SQL, SHL, SQR:* stimulation pulses for right and left hamstrings and quadriceps. *CC:* clock circuit.

electrode was attached to the leg laterally and was placed symmetrical to the stimulation electrodes. In order to obtain a better estimation of the gross overall myoelectric activity in the stimulated muscles, the stimulation electrodes were also used as recording electrodes for EMG. To the same leg which had the electrodes, a knee goniometer was fastened. The leg was passively lifted to full extension when the starting switch ST was closed. Signals from the electrodes, as well as the goniometer signal Φ and the start signal ST were recorded (Honeywell visicorder). The extended leg was suddenly released by the experimenter. Since the patients were asked to be completely relaxed the following swinging movement of the lower limb was determined only by the tonic level of the knee muscles. The EMG's and the goniograms as well as the start of movement (when the switch ST opened) were recorded, thus giving a graphic display of Wartenbergs spasticity test (2, 26). This procedure was repeated three times. To assess the variability of the patient's spasticity, the patient was asked to relax for 20 min. Thereafter the spasticity test was performed three times again. No other procedures were performed on the first day. Thus the first day's experiment intended to obtain a general idea about the level and characteristics of the patient's spasticity.

On the second day stimulation electrodes were also attached to the other leg. With signals QL and HL from the left quadriceps and hamstring connected to the EMG recorder spasticity was tested at the beginning of the experiment. Then the patient was asked to relax for 20 min when spasticity was tested again. After this relaxation period the stimulation treatment was introduced. Electrodes QL, HL, QR and HR were connected with the stimulators as described in the former paragraphs and stimulation was applied for 30 min. The stimulators were then disconnected and QL and HL were again connected to record myoelectric activity. Spasticity was tested, and once again after the patient was inactive for another 20 min.

The same procedure was repeated for the following two days. The patient was asked to keep the electrodes in place for the next few days. On the last day of the program (usually Friday) the patient was not stimulated any more but only tested for any possible long-term (carry over) effect of the treatment. Spasticity was tested at the beginning of the experiment and after 20 min of relaxation. The procedure was thus identical to the one on the first day.

Spasticity may be affected by various physiological (e.g. bladder, bowel, body position) and psychological factors. Therefore care had to be taken to reduce the variability of the results by keeping the experimental conditions as uniform as possible.

RESULTS

Regarding the effect of electrical stimulation on spasticity the patients could be categorized in three groups.

Patients 1 and 2

All the tests pre and post stimulation as well as pre and post relaxation showed with minor variations the hypotonic pattern of Fig. 2. However, both patients claimed quadriceps spasms specially in the morning and at night but such spasms were never observed during the tests. Stimulation produced smooth flexion–extension movements which might

be useful for functional purposes (12, 20) but their spasms remained unaffected by electrical stimulation.

Patients 3, 4 and 5

In these patients mostly the pattern of Fig. 2 was recorded during the program but subjective reports claimed reduced or weaker spasms during the rest of the day and at night. Patient 3 had less difficulties during transfers from bed to wheel-chair or other activities of daily living. Patient 4 had weaker spasms overnight and some increased volitional knee extension could be measured. Patient 5 stated that his spasms were markedly reduced the night after electrical stimulation in magnitude and frequency and he could sleep through the night uninterrupted by spasms which routinely woke him up before when he was turned in bed.

Patients 6 and 7

Both patients had strong hypertonia in the quadriceps which could be reduced by electrical stimulation. Patient 6 was tested for spontaneous spasticity for two consecutive days and exhibited strong extensor spasticity. In spite of variations in the response none of the records showed an oscillatory movement. The first day of stimulation produced a large reduction in spasticity. For the first time the limb oscillated but again became more stiff after 20 min of inactivity. The following day spasticity was the same as before stimulation but was somewhat reduced after stimulation and even more reduced after relaxation. Thus in this patient inactivity increased spasticity on one day and decreased it on another. On the third day of stimulation spasticity was again reduced but much less than on the first day. Follow-up tests on the next day produced about the same spasticity as on the day before.

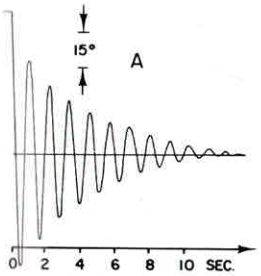

Fig. 2. Hypotonic pattern of knee joint movement in patients 1–5. The vertical axis displays angular movement in knee joint.

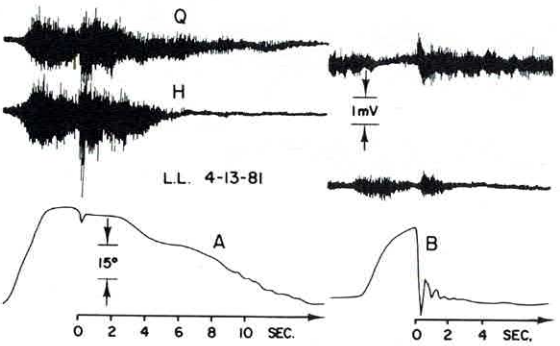

Fig. 3a. (A) Spasticity at start of test. (B) After 20 min of inactivity, lowest trace displays joint movement.

Subjectively the patient and his relatives claimed marked improvement. They observed less spasms and of decreased intensity during the evening and at night after electrical stimulation.

On initial testing the spasticity of patient 7 is shown in Fig. 3*a-A*: 20 min of inactivity reduced spasticity to a large degree (Fig. 3*a-B*). A typical record of the results of stimulation is shown in Fig. 3*b*. The pattern changed from a strong spasm prior to stimulation to reciprocal muscular activity in both antagonists following stimulation. Since this patient had also traces of volitional control we tested the effect of mutual facilitation of voluntary effort and electrical stimulation. The results are shown in Fig. 3*c*. Having volitional control of the knee extensor, only the hamstrings were stimulated in this experiment. The record shows that while either stimulation or voluntary effort could produce some trace of movement, their combination resulted in a large smoothly controlled movement (22) which could be potentially useful for functional purposes (12, 20, 27). Subjectively, the patient reported a relief of spasms for about six hours. He also claimed improved voluntary control of his legs.

A review of patient data with abbreviated results of electrical stimulation are shown in Table I.

DISCUSSION

Data of the seven patients show a large variability of spasticity in SCI patients. It is partly due to this variability that authors arrive at different conclusions regarding the effects of electrical stimu-

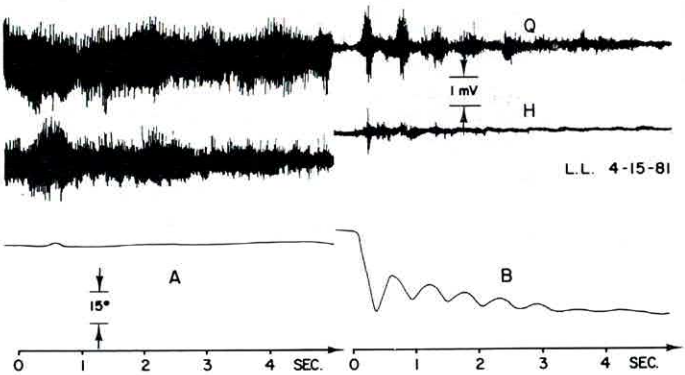

Fig. 3b. (A) Prestimulation spasticity. (B) Spasticity after 30 min of stimulation, lowest trace displays joint movement.

lation on a spastic SCI patient. In spite of a small patient population which has been analyzed in our study we have been fortunate to have patients with quite different patterns of spasticity behavior. In an attempt to categorize patients, the following grouping was observed:

1. Patients with occasional spasms but usually flaccid limb. Electrical stimulation did not change their spasticity but they might be candidates for other functional stimulation programs such as muscle re-education, standing, walking attempts, etc. (12, 20, 27) (Patients 1 and 2).

2. Patients with occasional spasms and usually flaccid limbs where objectively no change in spasticity could be measured, still they subjectively claimed a reduction in the number and intensity of spasms due to electrical stimulation (Patients 3, 4 and 5).

3. Patients where objectively and subjectively an improvement in spasticity and volitional control was observed (Patients 6 and 7).

Extreme care should be taken before a consistent reduction in spasticity is ascribed to electrical stimulation. The spontaneous variations in spasticity of some patients are so great that only after several days of careful measurements and observations reliable statements regarding the effectiveness of electrical stimulation should be made. The same, of course, holds true also for ascribing negative effects to electrical stimulation. Thus, for example. Bowman et al. (4) state that in three out of ten patients spasticity was increased due to electrical stimulation. A second look at their data shows that the same level of increased spasticity which resulted due to siumulation has been observed also days before when stimulation has not yet been applied. We feel that a beneficial effect of stimulation should be claimed only in cases where more relaxation was obtained than due to any spontaneous variation in the past days. Conversely, a harmful effect of stimulation should be claimed only when spasticity after stimulation was greater than at any time in the past days. Neither our data nor Bowman's report show such an impairment. Combining our and Bowman's patients we may thus conclude that electrical stimulation reduces spasticity in perhaps one half of the patients with SCI. In the rest of the patients the effects of electrical stimulation fall within the limits of spontaneous variations.

Regarding the duration of relaxation our data agree with former reports that the effect—if it is found—lasts up to 24 hours. Patients who could reliably benefit from electrical stimulation should thus be given stimulators for home use. In our study five patients thought that such stimulators would be helpful to them and expressed their willingness to apply electrical stimulation to themselves in a home environment if such stimulators were available. An alternative is of course offered by spinal cord stimulation where the the stimulator is surgically implanted and electrodes are placed

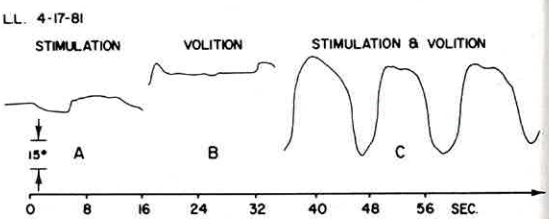

Fig. 3c. Movements produced by (A) hamstring stimulation, (B) voluntary effort, (C) combination of hamstring stimulation and voluntary effort. Vertical axis displays joint movement.

Table I. *General patient data and effects of electrical stimulation*

No.	Initials	Sex	Age	Lesion	Accident	Months post injury	Effects of electrical stimulation
1	F. G.	M	28	C6 compl	MVA[a]	10	None
2	S. P.	M	28	T5 compl	MVA	14	None
3	J. P.	M	36	T5 compl	MVA	6	Reduced spasms (subjective observ.)
4	J. H.	M	39	C7 incompl	GSW[b]	5	Reduced spasms (subjective observ.)
5	C. L.	M	22	T4 compl	MVA	4	Reduced spasms (subjective observ.)
6	C. C.	M	45	C5 incompl	MVA	11	Reduced spasticity, impr. volunt. movements
7	L. L.	M	19	T4 incompl	GSW	8	Reduced spasticity, functional movement with electr. stim.

[a] Motor vehicle accident.
[b] Gun shot wound.

over the dura of the dorsal column. We believe however that surface stimulation should be used whenever possible since it is easier, less expensive and can be discontinued at any time. Only in cases when long-term use of stimulation offers functional benefits to the patient, when skin electrodes prove to be uncomfortable and spasticity affects several joints (8) spinal cord stimulation might prove to be the method of choice. Unfortunately, no well documented study exists as yet comparing the effects of surface stimulation versus spinal cord stimulation on spasticity in SCI patients. Such a study would be of great clinical value since it is quite possible that the basic neurophysiological mechanisms in both techniques are the same (21).

Comparing the four-channel stimulation used in our experiments to other techniques we are not able to answer the question if a more aggressive electrical stimulation regimen is advantageous to methods applied until now. Since there is enough evidence that most reported techniques do not have long lasting therapeutic effects (longer than one day), various stimulation regimens and stimulation parameters (frequency, pulse width) could be tested on the same patient over a period of several days in order to assess the simplest and most efficient treatment.

Due to the small number of patients it is risky to speculate about the differences between patients in the three categories. It seems however that patients with flaccid paralysis and occasional spasms benefit less from electrical stimulation than patients with tonic spasticity like patients 6 and 7. Perhaps electrical stimulation is more effective in patients who need a continuous additional signal input in order to improve the excitation—inhibition imbalance of the spinal cord or some residual supraspinal structures. It is for example interesting to note that both patients in whom no effect of electrical stimulation was observed (no. 1 and no. 2) had complete lesions while both successful cases (no. 6 and no. 7) had incomplete lesions. The remaining three patients with non-measurable but subjective improvements had either complete (no. 3 and no. 5) or incomplete (no. 4) lesions. To assess a possible correlation between the type of lesion and the efficacy of electrical stimulation would present an interesting neurophysiological investigation.

CONCLUSIONS

1. On randomly selected SCI patients with spasticity in the knee joint about one half might benefit from electrical stimulation with surface electrodes. In the rest of the patients the effects of stimulation are inconclusive but there are no documented reports of increase in spasticity due to stimulation.

2. Even if electrical stimulation does not improve spasticity it is worthwhile to look at other potential benefits which stimulation might offer to the patient like improved voluntary control and muscle training for functional purposes.

3. Further studies are required to assess the comparative efficacy of different treatment regimens with electrical stimulation including spinal cord stimulation.

4. Since surface electrical stimulation is a harmless, non-invasive procedure its value for treatment of spasticity should be tested on a long-term basis either at home or in a clinical environment.

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