Spinal Cord Injury and Electric Stimulation: Types and Characteristics of Spasticity

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Abstract
Spasticity manifests itself in manifold features. In spinal cord injury (SCI) patients it can appear with different peculiarities. We studied the spasticity of 17 SCI patients in relation to their responses to therapeutic electrical stimulation (TES). We distinguished 3 types of spinal cord spasticity. The first and most frequent type (11 out of 17) is clinically characterized by clonic contractions of the antigravitatory muscles in the lower limbs (mostly quadriceps and soleus muscles), sometimes associated with tonic or clonic spasms in the trunk in the cervical level lesions; this kind of contractions are usually remarkably reduced (10 out of 11) by TES applied on quadriceps and hamstring muscles and on the peroneal nerve. The second type was observed in 1 of the 17 patients and it consists of strong tonic extensor spasm of trunk and lower limbs. This form of true spasticity is very similar to the hemiplegic spasticity and is presented by patients in which the lesions affected also, in some extent, the supra-tentorial nervous formations (viral lesions). It benefits greatly from TES of antagonistic muscles. The third form was observed in 2 cervical patients and is typified by severe spasms, mostly tonic, prevalently in flexion in the lower limbs and extension in the trunk; this form is not sensitive at all to TES. The reduction of contractions and spasticity in the first two types was usually, but not always, of short term. A consequence of this study is that we have to re-consider what we call spasticity in SCI patients.

Key words: paraplegia, SCI, spasticity, electrical stimulation, FES, TES.

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An epidemiologic study by Maynard and coll. [11] on spasticity in traumatic SCI patients showed that 67% of 96 subjects developed spasticity during hospitalization and 37% had to be submitted to antispastic pharmacological medication. At the one-year follow-up the percentages were changed respectively to 78% and 49%. Kawamura et al. [8] observed the spasms of quadriplegic patients and classified 3 types of spasticity in the upper and lower limbs (tonic flexor, tonic extensor and clonic spasms) and 2 types in the trunk (clonic and tonic spasms). Furthermore they classified 4 patterns of spasticity in the four limbs: flexor spasms in upper limbs associated with extensor spasms in the lower limbs; extensor in upper and flexor in lower limbs; extensor in the 4 limbs; flexor in the 4 limbs. Literature regarding the effect of functional/therapeutic electrical stimulation on spasms originating from SCI shows variable results, likely because of the difficulties to measure spasticity and the different methods of stimulation used. Most of the clinicians and researchers refer to a relaxing effect, though mostly of short term [3, 4, 5, 7, 13, 14, 15, 16, 17].

Thus, we have to face the fact that what we generally call spasticity in SCI patients manifests with different features and, in addition, some clonic contractions can occur both in hypertonic and in hypotonic or normotonic muscles. This implies the existence of different pathophysiological mechanisms and makes the spastic contractions respond in different ways to the same therapies, in particular to electric stimulation.

Starting from this point of view, we have reviewed the clinical documentation of 17 SCI patients treated with TES in the last three years, both for orthotic (application of RGO and the like) and therapeutic purposes. The only selection criterion was the availability of complete documentation. Although no statistical conclusion can be drawn from this study, a novel approach to the identification and description of spasticity in SCI patients is presented.
SCI spasticity and TES

Material and Methods

The clinical documentation of 17 patients affected with spinal cord lesions was examined. The age of patients ranged from 21 to 65, average 37y. The origins of the lesions were traumatic in 13 and viral in 4 patients. The levels of lesions were: 8 cervical, 8 thoracic and 1 lumbar. As for completeness of paralysis, 11 were Frankel A, 2 Frankel B, 3 Frankel C and 1 Frankel D (Tab. II).

During treatment with TES all antispastic pharmacological medications were interrupted and severity of spasticity was evaluated according to a scale based on the visible force, duration and frequency of spasms, as reported by the patients and by the nursing personnel. The observations were made in a lapse of 6 hours between 9 a.m. and 3 p.m., when various activities are more concentrated, from rehabilitation activities to eating and resting. The obtained scale is shown in Table I.

The parameters of electrostimulation used were: trains of impulses of 600 μs at 30 Hz, lasting 5 s with a duty-cycle 1/4, for 20 min per muscle group, 2-3 times a day, 5 days a week. The duration of the treatment varied from patient to patient from 1 to 12 months. The trains were delivered via surface electrodes. The target muscle groups were the ones usually reported in the literature to be stimulated. In particular we treated the quadriceps and hamstrings muscles, the dorsiflexor and eversors of foot, the latter usually by peroneal nerve stimulation, in order to activate the whole muscle group; gluteal, abdominal and lumbar muscles when required.

Most of the patients were alternatively stimulated on the quadriceps of one side and the hamstrings of the other side to give the sequence of walking.

Patient n° 8 (see Table II), with a high tonic extensor spasticity in his trunk and lower limbs, was stimulated on hamstrings muscles. This strategy was adopted in order to inhibit, by reciprocal inhibition, the spastic quadriceps muscles; in addition, the peroneal nerves of both sides were stimulated just to obtain a relaxation of quadriceps and soleus muscles by the withdrawal reflex.

Results

The results are summarized in Table II, that describes the patients and their type of contractions, besides spasticity severity before and after TES treatment.

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age</th>
<th>Level of injury/ Frankel grade</th>
<th>Cause*</th>
<th>Time from injury (y)</th>
<th>Type of contractions</th>
<th>**Severity of spasms Before TES After TES</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>43</td>
<td>C4/C</td>
<td>T</td>
<td>1</td>
<td>Clonic extensors, lower limbs</td>
<td>M L</td>
</tr>
<tr>
<td>2</td>
<td>65</td>
<td>C5/B</td>
<td>T</td>
<td>0.4</td>
<td>Tonic flexors, upper limbs</td>
<td>M L</td>
</tr>
<tr>
<td>3</td>
<td>23</td>
<td>C5/C</td>
<td>T</td>
<td>6</td>
<td>Clonic extensors, lower limbs</td>
<td>M M</td>
</tr>
<tr>
<td>4</td>
<td>33</td>
<td>C5/A</td>
<td>T</td>
<td>4</td>
<td>Tonic flexors and extensors</td>
<td>H H</td>
</tr>
<tr>
<td>5</td>
<td>53</td>
<td>C6/A</td>
<td>V</td>
<td>5</td>
<td>Tonic, mainly flexors</td>
<td>H H</td>
</tr>
<tr>
<td>6</td>
<td>63</td>
<td>C6/A</td>
<td>T</td>
<td>3.5</td>
<td>Clonic extensors and flexors</td>
<td>M A</td>
</tr>
<tr>
<td>7</td>
<td>24</td>
<td>C6/B</td>
<td>T</td>
<td>1</td>
<td>Clonic extensors and flexors</td>
<td>M A</td>
</tr>
<tr>
<td>8</td>
<td>21</td>
<td>C7/C</td>
<td>V</td>
<td>3</td>
<td>Clonic extensors, lower limbs</td>
<td>H L</td>
</tr>
<tr>
<td>9</td>
<td>21</td>
<td>T4/A</td>
<td>T</td>
<td>3</td>
<td>Clonic extensors and flexors</td>
<td>L A</td>
</tr>
<tr>
<td>10</td>
<td>29</td>
<td>T5/A</td>
<td>T</td>
<td>1</td>
<td>Clonic extensors and flexors</td>
<td>M A</td>
</tr>
<tr>
<td>11</td>
<td>39</td>
<td>T5/A</td>
<td>T</td>
<td>13.5</td>
<td>No contractions</td>
<td>A A</td>
</tr>
<tr>
<td>12</td>
<td>58</td>
<td>T6/A</td>
<td>V</td>
<td>3.5</td>
<td>Clonic extensors and flexors</td>
<td>M L</td>
</tr>
<tr>
<td>13</td>
<td>46</td>
<td>T7/A</td>
<td>T</td>
<td>6</td>
<td>Clonic extensors and flexors</td>
<td>M L</td>
</tr>
<tr>
<td>14</td>
<td>40</td>
<td>T8/A</td>
<td>T</td>
<td>1</td>
<td>Clonic extensors and flexors</td>
<td>M L</td>
</tr>
<tr>
<td>15</td>
<td>30</td>
<td>T9/A</td>
<td>T</td>
<td>1</td>
<td>Clonic extensors and flexors</td>
<td>M L</td>
</tr>
<tr>
<td>16</td>
<td>35</td>
<td>T10/A</td>
<td>T</td>
<td>0.5</td>
<td>No contractions</td>
<td>A A</td>
</tr>
<tr>
<td>17</td>
<td>45</td>
<td>L2/D</td>
<td>V</td>
<td>0.3</td>
<td>No contractions</td>
<td>A A</td>
</tr>
</tbody>
</table>
SCI spasticity and TES

From the table we can see that among the 17 patients studied: i. 3 patients (No. 11, 16, 17) did not present any spastic contractions; ii, the cervical level is the greatest producer of spasticity; iii. 3 cervical patients showed the most serious form of contractions.

One of the patients without contractions (No. 11) was paraplegic for more than 13 years and, though his lower limbs muscles were very atrophic, he still assumed antispastic drugs. Ten of the 11 patients with prevailing clonic spasms gained considerable benefits from TES. Patient No. 8 obtained a substantial decrease of his high tonic extensor spasticity, followed by unmasking of voluntary flexion and extension of the knee. Three had no improvement by TES: one of them (No. 3) had tonic and clonic extensor spasms in the lower limbs and trunk of moderate intensity; patient No. 4 had tonic and clonic extensor spasms in his lower limbs and trunk of high intensity; patient No. 5 had highly severe tonic flexor spasticity in his lower limbs and extensor spasms in the trunk.

As it can be seen in Table II, the presented 17 SCI patients exhibited 3 types of contractions.

Eleven patients (No. 1, 2, 3, 6, 7, 9, 10, 12, 13, 14, 15) had mainly clonic contractions of their antigravitational muscles, usually quadriceps in lying position and soleus in sitting position; patient No.1, C4-C traumatic, had also some tonic flexor spasms in the upper limbs; 10 of these subjects gained substantial benefits from TES, with reduction or cessation of the spasms: 1 (No.3) had no visible result.

Two (No. 4 and 5) had very severe prevalently tonic (but also clonic) contractions, No. 4 mainly extensor, No. 5 mainly flexor; the spasms could be elicited even by the slightest stimuli; these patients had no improvement by TES, that, on the contrary, was a powerful stimulus triggering the spasms.

One (No. 8) showed a strong tonic extensor spastic contraction in the lower limbs and trunk in any position; sometimes, when put in sitting position, clonic contractions of the soleus muscle started; the patient, C7-C of viral origin, obtained a remarkable relaxation of spasticity by the electrostimulation of hamstrings and gluteal muscles, as antagonists of the quadriceps, and of peroneal nerve with an intensity sufficient to evoke the withdrawal reflex such to break the extensor pattern.

It should be mentioned that in a parallel electrophysiological study of ours conducted on the same topic we have observed that some SCI patient with clonic contractions can suffer a worsening by electrical stimulation, which was not the case in the 17 patients of the present study.

Concerning the duration of the positive results after TES, the clinical experience drawn from this work confirms, in the majority of cases, the other Authors’ findings [4, 7, 15], i.e. a short-term effect, not lasting more than 24 hours.

Yet, some of our patients had a different history. Patient No. 10, 29y. old, T5-A traumatic, practised TES at home for about 4 months and became completely free from the contractions; then he stopped electrical stimulation and stayed without any spasm for about 4 weeks, after which the contractions slowly returned, so he began again TES. Patient No. 6, 63 y. old, C6-A traumatic, after 3 years and a half of complete paraplegia and partial tetraplegia, experienced TES at lower limbs for more than one year together with intensive physical exercise; in this way he acquired a very good trophism, many new motor abilities and voluntary contraction of quadriceps muscles with force 3 MRC (muscle force testing by Medical Research Council scale, score from 1 to 5). This man stopped electrical stimulation, but not physical exercises, about 2 years ago and to-day is still free from any kind of spasm or involuntary contractions.

Discussion

The observations on the 17 SCI patients above mentioned strongly suggest that what we generally call “spasticity” in SCI patients displays at least three different initial forms; in addition, the three forms of “spastic” contractions respond differently to electric stimulation.

The first form, and most frequent, shows generally clonic contractions, mostly in the quadriceps and in the soleus muscles, sometimes in the hamstrings muscles as well. They can appear in normotonic, in hypertonic and also in hypotonic and hypotrophic muscles, when hypotrophy is not too advanced. Indeed, it seems inappropriate to classify as spastic a hypotrophic and hypotonic muscle. Furthermore, the majority of these patients gain benefits from electric stimulation applied directly on these muscles: improvements of haematic and lymphatic circulation, muscle bulk, skin trophism, heart function, as described by several Authors [2, 6, 7, 9, 10, 12], together with a decrease or ceasing of the clonic contractions. We know that stimulating a spastic muscle means increasing, not decreasing spasticity. However, only maximal and prolonged electric stimulation can transiently exhaust a spastic muscle, due to local fatigue and possibly the Vedenski effect [1]. A neurophysiological study in progress by our group is confirming that many (but not all) of these patients do not show electrical signs of hyper-excitability of H-reflex nor other signs of spasticity. It thus appears more reasonable to ascribe their contractions to other origins. We feel that a possible explanation could be as it follows: the first contraction is reflex in nature (the positive support reaction and other primitive reflex reactions can re-appear in neurological patients such as hemiplegics and paraplegics) and caused, e.g., by strains when the patient is being brought to a standing position to try an orthosis on, or when changing position in the bed, or when is sitting down on a wheelchair. The first contraction is immediately followed by a fall of tone in the muscle implying a stretching movement, that provokes a stretch reflex causing again a contraction, then a fall and the cycle repeats itself. This seems more evident in the patients with more hypotonic and hypotrophic muscles, where the electro-training and strengthening of their muscles seem to reduce for a long the clonic contractions. Surely, wider and deeper neurophysiological studies on these subjects should be carried out.
The second kind of contractions is in our opinion true spasticity. It is localized in the antigravitatory muscles of the lower limbs and trunk, particularly in the quadriceps and calf muscles, and shows a strong tonic extensor contraction. Clonic contraction of the soleus muscle is sometime associated when the patient is put in a seated position. This form looks very like hemiplegic spasticity. Thus, in patient No. 8 we used the same strategy utilized in hemiplegia applying TES on the antagonistic muscles, as hamstrings, gluteal and peroneal nerve of both sides, in order to counteract spasticity of quadriceps and calf muscles and to evoke flexion of the lower limbs by the withdrawal reaction, in order to break the extensor spastic pattern.

The third form is a severe spasticity, arisen in 2 of our cervical patients and showing uncontrollable spasms of the trunk and legs, often also in the upper limbs, mostly tonic, one patient in flexion (the most severe, No. 5) and one prevalently in extension. This kind of spasticity cannot be treated by electric stimulation and the pharmacological medication can be ineffective as well.

Conclusion

As it was said in the introduction of this paper, the study was not designed for drawing statistic and general conclusions. This notwithstanding, from this study some strong suggestions arise: i, spasticity in SCI subjects manifests itself with different clinical features, likely implying different pathophysiological mechanisms; ii, in some patients the clonic contractions seem not to depend on spasticity, but on other causes; iii, each form of contractions needs a different approach, especially regarding TES. We believe that these findings present a challenge for both neurophysiologist and physiatrist communities.

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References


