Reflex activity triggered by electrical stimulation in spinal cord injured persons

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Abstract

In spinal cord injured (SCI) persons, neuromuscular stimulation for activation of paralyzed muscles frequently results in the triggering of unwanted spastic contractions caused by the activation of hyper-sensitive reflex loops. Experimental investigations in four SCI persons showed that the onset of these spastic contractions decreased with stimulation frequency, but the number of stimulation pulses delivered before the spastic contraction started decreased with stimulation frequency. Furthermore, the extent of these spastic reactions appeared to be history-dependent: subsequent stimulation bursts resulted in less extensive spastic activity. These effects should be taken into account when stimulating muscles in SCI persons and deserve further investigation.

1. Introduction

Electrical stimulation has been proposed for many years as a promising rehabilitation technique to artificially activate muscles that are not anymore under voluntary control following spinal cord injuries. Thus, paralysed muscles can be activated to contribute to the restoration of lost motor functions (Functional Electrical Stimulation: FES) [1, 2].

Despite considerable research effort devoted to the implementation of such technique into useful systems, FES has not been commonly acceptable as a clinical aid to restore mobility. Among many problems researchers have been trying to solve, spastic hyperreflexia has been an important factor limiting the success of FES [1]. In a retrospective study, Barolat and Mainam [3] showed that 97% of the SCI individuals of their study experienced spasms, 72% exhibited spasms within six months after the injury with a peak in severity within 12 months. Even if narrow inclusion criteria are followed during the recruitment of SCI subjects for experimental investigations, the selected subjects will most likely express muscle spasm at different levels. The spastic activity may be manifested as severe short bursts of forceful muscle contractions or less forceful but frequent contractions. These contractions occur spontaneously, but are likely to be triggered by tactile or nociceptive afferents.

The objective of this study was to analyze the excessive spastic reactions triggered by electrical stimulation in SCI persons, specifically the influence of stimulation frequency on the onset of the spasm and the effect of previous activation on the size of the spastic reactions. This study has been published more extensively in the form of a journal paper [4].

2. Methods

Subjects

Four spinal cord injured subjects (1 female, 3 males) enrolled in the FES training program of the Roessingh Rehabilitation Centre (Enschede, The Netherlands) gave their written informed consent to participate in this study. A description of the subjects in terms of age, level, duration and ASIA class [5] of the lesion is given in Table 1. At the time of the experiments, none of the subjects trained regularly the tested muscle with electrical stimulation.

Set up

The set-up was described in detail by Mela et al. [6]. Briefly: the subject was seated on a custom designed bench and safely secured to it. The foot of the leg to be tested was tightly strapped to a foot plate which could be rotated around an axis, allowing the ankle to be positioned in any dorsal flexion or plantar flexion angle. The moment around the axis of the ankle was...
determined by multiplying the force reading of a strain
gauge force transducer by the distance from the
transducer to the axis of the ankle. Isometric ankle
moments were measured at a joint angle close to
optimum (i.e. the joint angle at which maximal moment
was elicited).

<table>
<thead>
<tr>
<th>Subject</th>
<th>Age (years)</th>
<th>Lesion level</th>
<th>Spontaneous spasms</th>
<th>Lesion duration (years)</th>
<th>ASIA class</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>30</td>
<td>T5</td>
<td>present</td>
<td>1</td>
<td>A</td>
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<tr>
<td>2</td>
<td>59</td>
<td>T1</td>
<td>hardly</td>
<td>0.5</td>
<td>B</td>
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<tr>
<td>3</td>
<td>49</td>
<td>T9/T10</td>
<td>severe</td>
<td>9</td>
<td>A</td>
</tr>
<tr>
<td>4</td>
<td>35</td>
<td>T5/T6</td>
<td>present</td>
<td>10</td>
<td>A</td>
</tr>
</tbody>
</table>

Table 1. Characteristics of SCI subjects with respect to level, classification, duration of the lesion and presence of spasm

Stimulation protocol
Two second duration constant frequency bursts at
seven different stimulation frequencies (8, 12, 16, 20,
25, 33, 50 Hz) were applied in a random order. Each
stimulation protocol was repeated twice without any
additional time allowed between repetitions, which
meant five seconds between consecutive bursts. Bursts
with different frequencies were separated by one minute
recovery time. Since subject 4 exhibited spasms little
longer than two seconds, the duration of the contractions
was extended to three seconds. Current amplitudes were
chosen as to be higher than the threshold value,
resulting in partial recruitment of the dorsiflexor
muscles. The amplitudes used were: 50, 45, 40, 55 mA
for subject 1 to 4 respectively. Subject 3 participated in
our study despite exhibiting recurrent spontaneous
spasms. In his particular case, stimulation was applied
in silent periods between spontaneous contractions.

3. Results
Frequency dependent onset of spastic activity
All four subjects exhibited excessive responses after
the onset of the applied electrical stimuli (e.g. fig.1). The
onset of the spasms was dependent on the
stimulation frequency of the burst, higher stimulation
frequencies triggering the reflex earlier, as indicated by
the arrows in fig.2. The onset of the spasms as a
function of the stimulation frequency is reported in
figure 2 in terms of time elapsed since the onset of the
stimulation (figure 2a) as well as in terms of number of
pulses delivered since the onset of the stimulation
(figure 2b) for each subject. Note the reversal of the sign
of the slopes.

It is concluded that at higher stimulation frequencies,
a higher number of pulses were delivered before the
spasm was triggered, although the onset time of the
spasm decreased.

Repeated contraction
The immediate repetition of the burst with the same
parameters had the effect of clearly reducing if not
eliminating the spastic reaction [4].

4. Discussion
Short bursts of electrocutaneous stimulation triggered
spastic reactions in all four subjects. The same protocol
with comparable current amplitudes, used for
experiments on able-bodied subjects [6] did not elicit
any exaggerated reaction. This is in accordance with
studies showing that the threshold for skin stimulus to
trigger reflex reactions is lower after SCI or stroke
compared to able-bodied individuals [7, 8].

Afferent activity can be generated by electrical
stimulation both by direct stimulation of afferents (e.g.
Ia afferents) and by sensory signals from spindles and
tendon sensors (proprioreceptors) upon muscle
contractions. These afferent signals may elicit reflexes
in able-bodied people, but can trigger spastic
contractions in SCI individuals. In an intact control
system the gains of the reflex loops are modulated by
central inputs. If such modulation is not optimal, the
reflex loop may become unstable, resulting in spastic
contractions that may continue even after stimulation
has stopped. The gain of the reflex loops may also be
influenced by increased sensitivity of the muscle
spindles due to inadequate γ-fiber activation by the
central nervous system.
Apparently, a cumulative effect in the spinal neural circuitry seems to determine the onset of the spastic activity. The onset time of the excessive spasms exhibited by the subjects of this study was clearly longer for lower stimulation frequencies and fewer stimulation pulses were required at those frequencies to trigger the spasm.

Figure 2: Effect of stimulation frequency on onset time of spastic contractions. Onset times for all subjects in terms of a) time from the beginning of the stimulation burst, and b) number of pulses from the onset of the burst [4].

References

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