Excessive Reflexes in Spinal Cord Injury Triggered by Electrical Stimulation

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Abstract

Interaction of electrocutaneous stimulation with an impaired human motor control system may result in unstable reflex loops causing excessive spastic reactions.

These contractions are usually excluded from analysis since the presence of spasm is one of the criteria commonly applied for discarding a contraction. They may, however, provide interesting information on the nature of spasticity. The dorsiflexor muscles of four SCI subjects were activated by means of surface electrical stimulation and the isometric ankle moment was measured. Short bursts of constant stimulation frequency at seven different frequencies (8, 12, 16, 20, 25, 33, 50 Hz) triggered spastic reactions in all subjects. The onset times of spastic activity during an electrically elicited contraction shortened with increased stimulation frequency. A stimulation burst may also have a spasticity reduction effect on a subsequent burst, indicating potential short term therapeutic effects of stimulation on spasticity in isometric conditions.

Keywords: Surface electrical stimulation, spasticity, reflexes, isometric contractions, spinal cord injury, human dorsiflexor muscles.

Introduction

Electrical stimulation has been proposed for many years as a promising rehabilitation technique to artificially activate muscles which are not anymore under voluntary control following spinal cord injuries.

One of the possible applications of functional electrical stimulation is the restoration of mobility (Franken et al., 1994; Kilgore et al., 1997; Veltink & Donaldson, 1998; Taylor et al., 1999). Despite considerable research effort devoted to the implementation of such technique into useful systems, FES has not been accepted as a clinical aid to restore mobility.

Among many problems researchers have been trying to solve, ranging from the lack of selectivity of excitation to the non-linearity and time-dependence of skeletal muscle's properties (Prochazka, 1993), spastic hyperreflexia has been an important factor limiting the success of FES (Kralj & Bajd, 1989). In a retrospective study, Barolat and Mainam (1987) 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 (Schmit et al., 2000).

Electrical stimulation of peripheral nerves interferes with the physiological control system. During electrically elicited contractions, such interference is manifested as a minor contribution to muscle activation for able bodied subjects due to the combined effect of orhodromic sensory and antidromic motor excitation of peripheral nerves, resulting in H reflexes and F waves (Mela, 2001), or as powerful muscle contractions in impaired control systems associated with SCI.

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

Subject	Age (years)	Lesion level	Spontaneous spasm	Lesion duration	ASIA class*
1	30	T5	present	12 months	A
2	59	T1	hardly any	6 months	B
3	49	T9/T10	severe	9 years	A
4	35	T5/T6	present	10 years	A

* ASIA (American Spinal Injury Association (Maynard et al., 1997) score is used to classified the completeness of the lesion: A, sensory and motor complete; B: sensory incomplete, motor complete).
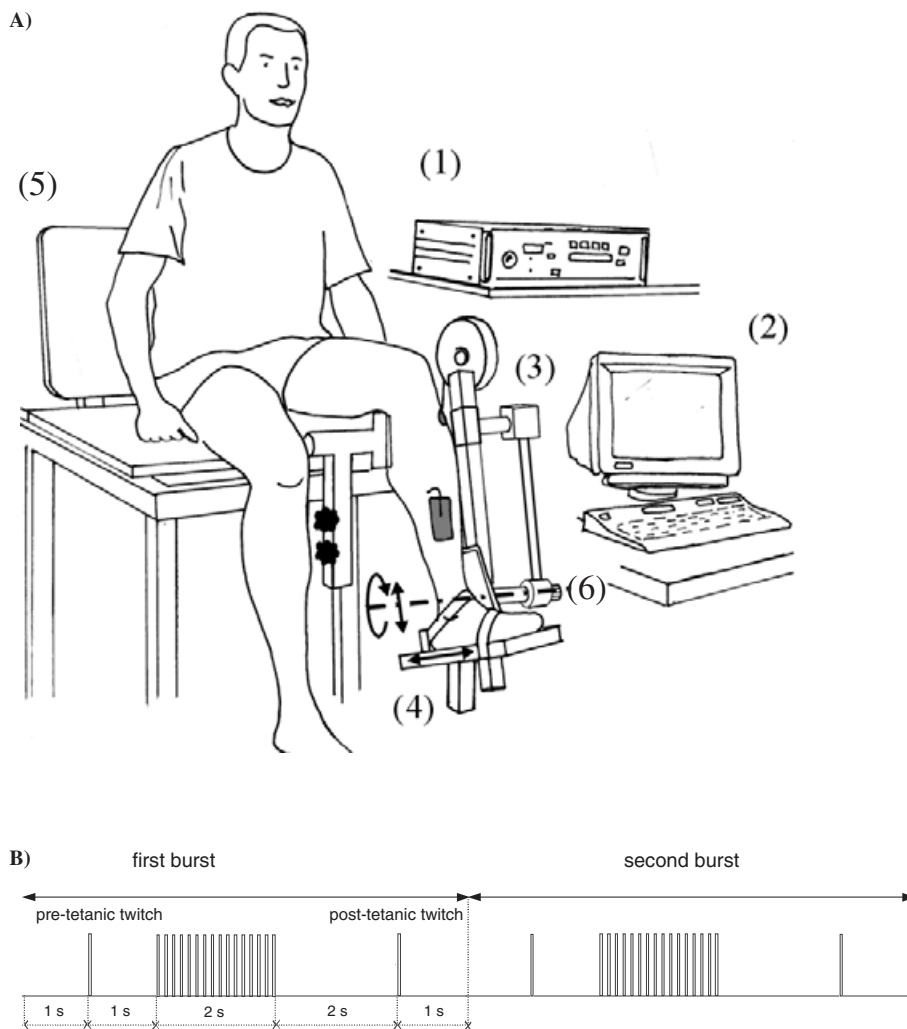

Fig. 1. Schematic representation of the experimental set-up and the stimulation train used during this study.
A) The set-up consisted of a custom designed bench (5) with an adjustable foot plate (4), a force transducer (3), a constant current stimulator (1), a potentiometric goniometer (6), an AD converter and a PC (2).
B) Two second continuous bursts were given at different constant frequencies (8, 12, 16, 20, 25, 33, 50 Hz). Each burst was repeated twice for each frequency without any recovery time allowed between repetitions. The bursts were preceded as well as followed by a twitch.

Such contractions are usually excluded from analysis, as the presence of spastic reactions is a common criterion for discarding contractions.

The objective of this study was to analyze the excessive spastic reactions triggered by electrical stimulation, 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.

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 (Maynard et al., 1997) 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

A detailed description of the set-up can be found in (Mela et al., 2001). Briefly: the subject was seated on a custom designed bench (Fig. 1) and safely secured to it. This was a necessary precaution to be taken, as electrical stimulation may trigger forceful spastic reactions involving multiple joints and affecting the balance of the subject. 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 ankle angle was measured by means of a potentiometric goniometer with respect to the reference position in which the sole of the foot was perpendicular to the tibia. The moment around the axis of the ankle was determined by multiplying the force reading of a strain gauge force transducer

by the distance of the transducer from 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).

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 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 used amplitudes 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 case, stimulation was applied in silent periods between spontaneous contractions.

Results

First contraction

All four subjects exhibited excessive responses after the onset of the applied electrical stimuli (e.g., Fig. 2). 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 Figure 2. The onset of the spasms as a function of the stimulation frequency is reported in Figure 3a in terms of time elapsed since the onset of the stimulation as well as in terms of number of pulses delivered from the onset of the stimulation (Fig. 3b) for each subject. Note the reversal of the sign of the slopes.
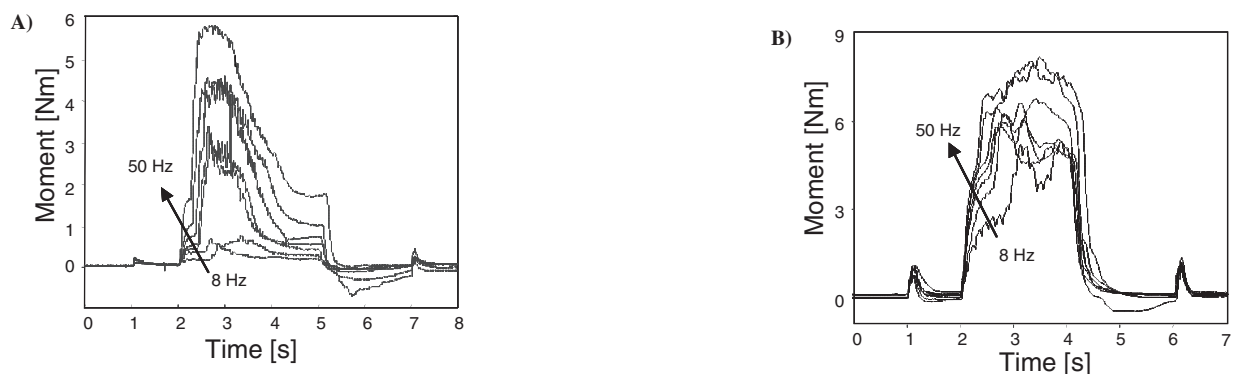

Fig. 2.   Effect of stimulation frequency on the onset of excessive spastic reactions.
A) Experimental moment-time traces for subject 4 at different frequencies.
B) Moment-time traces for subject 2. Note the dependence of the reflex onset on the stimulation frequency, higher stimulation frequency triggering the spasm earlier. The arrows indicate the stimulation frequency increasing from 8 to 50 Hz.
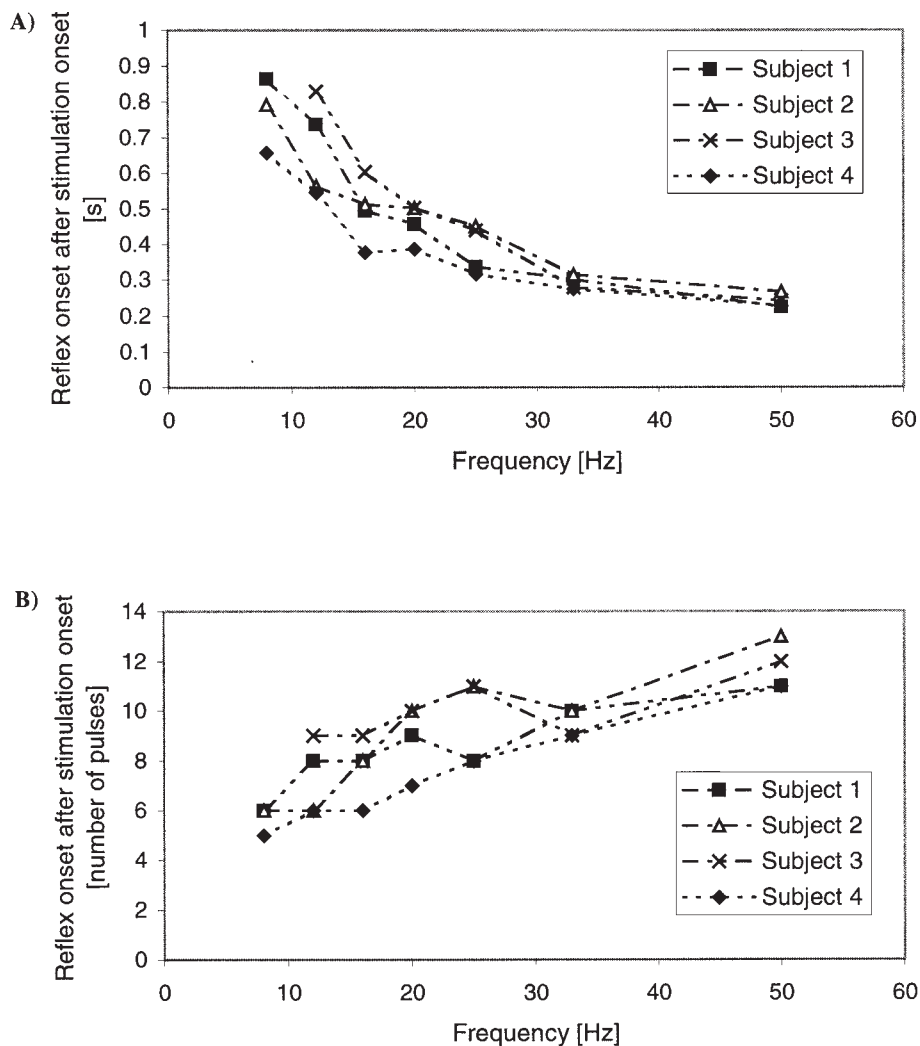
A)

B)

Fig. 3.   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.

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 (e.g., Fig. 3a), if not eliminating, the spastic reaction (e.g., Fig. 4b and c). Subject 4 exhibited similar reflexes during the second burst, showing the same dependence on the stimulation frequency of the burst. The onset times were longer than those measured during the first burst. The resulting spastic contractions were less powerful and of shorter duration (Fig. 4a).

Subject 1 and 2 had moment-time traces that did not show

any sudden increase in the exerted moment such as to be attributed to a reflex (e.g., Fig. 4b, 2nd burst).

For subject 3, contrary to what was seen for the other three subjects, the reflex reaction resulted in spastic contractions continuing after the stimulation stopped. The repeated bursts could exhibit reflexes similar to those of the first burst, but delayed as for subject 4, absent as for subjects 1 and 2 or present as an additional moment component (Fig. 4c).

Figure 5 shows the ratio between the maximum moment during the spastic contraction and the moment just prior the onset of the reflex for all subjects except subject 3. In his case, the spastic contractions saturated the force sensor (e.g., Fig. 4c). The ratios refer to the first burst of each stimulation frequency. For subject 4 the moment ratios were calculated for the second bursts as well.
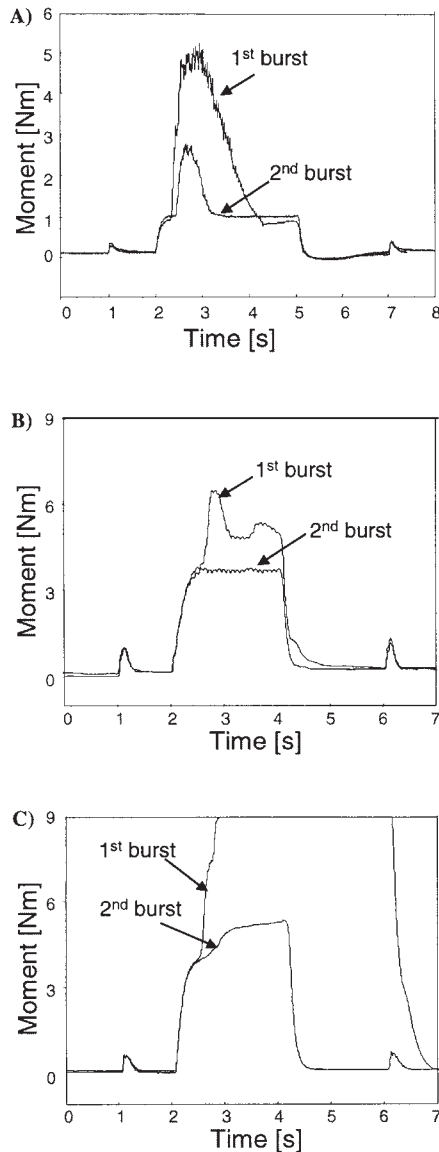
Fig. 4.   Effect of preceding stimulation on spastic reaction. Examples of repeated stimulation with burst at the same frequency and approximately 5 seconds allowed between bursts are given.
A) Moment-time traces for subject 4: the reflex was delayed and less powerful (stimulation frequency 25 Hz);
B) Moment-time traces for subject 1: the second burst did not exhibit reflexes (stimulation frequency 16 Hz);
C) Moment-time traces for subject 3: the second burst showed an additional force component (beginning at the arrow) due to a reflex which, unlikely the one triggered during the first burst, ended with the stimulation (stimulation frequency 50 Hz). The first burst for this subject gave always rise to an unstable reflex which continued also when stimulation had stopped. Subject 3 exhibited also second contractions with the patterns shown in this picture A) and B).

There is no clear evidence for a stimulation frequency effect on this ratio.

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 (Mela et al., 2001) 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 (Shahani & Young, 1971; Roby-Brami & Bussel, 1987; Nielsen et al., 1998).

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.

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. Apparently, an integration effect in the spinal circuitry is involved requiring a certain number of inputs within a given time to change the membrane potential as to increase the reflex loop gain. In an intact control system the gain of the reflex loop is modulated by central inputs. If such modulation is not optimal, the reflex loop may become unstable, resulting in spastic contractions that continue even after stimulation has stopped, as shown for subject 3.

The gain of the reflex loop may also be influenced by increased sensitivity of the muscle spindles due to inadequate γ fiber activation by the central nervous system. It is unlikely that the stimulation applied would result in excitation of these fibers as they are relatively small sized and have, therefore, a high excitation threshold (McNeal, 1976; Veltink et al., 1988).

Latencies of triggered spasm in the order of several hundreds of milliseconds have been shown for flexion withdrawal reflexes elicited via stimulation of the common peroneal nerve (Granat et al., 1988). The same authors reported that stimulation at higher frequencies tended to reduce latencies. It is not clear whether the spastic contractions investigated in the current study involved flexion withdrawal reflexes elicited by excitation of the cutaneous afferent nerve fibers. Although some overlap of the diameter distribution cannot be excluded, the pain and touch nerve fibers associated with the withdrawal reflex have diameters smaller than the α motor neuron. They are, therefore, expected to be excited at stimulation currents higher than those applied in this study, which resulted in only partial recruitment of the dorsiflexor muscles.
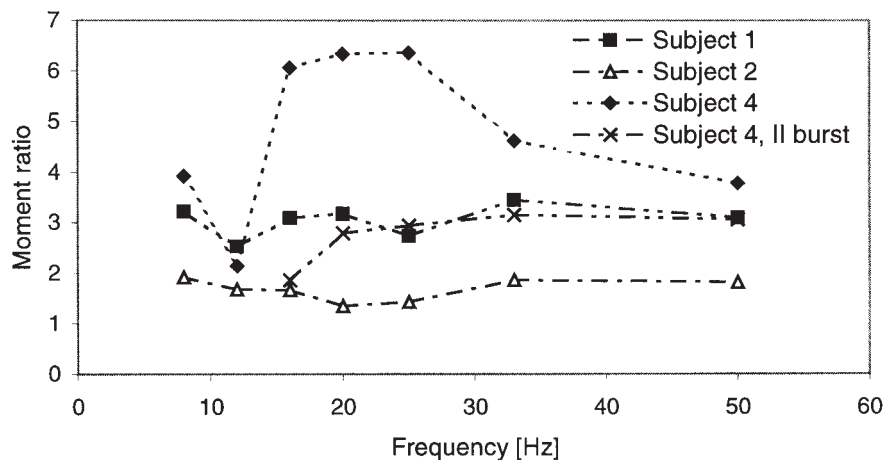
Fig. 5. Ratio between the maximal moment during reflex contractions and the moment at the onset of the reflexes as a function of stimulation frequency for each subject. For subject 4 the ratio was evaluated for the second burst as well. Subject 3 was not included because of saturation of the force recording.

The peak moment during the spastic contraction was two to six fold the level prior to the onset of such contractions. If stimulation was applied with functional aim to produce a desired moment, this would result in a much higher moment than the target one. However, for the present study, it must be considered that the moments produced by direct stimulation (prior to the reflex onset) were probably too low to be used functionally. Furthermore, the moment ratios have been evaluated on the first burst. During the following burst the spastic reactions were reduced or suppressed. Other authors (Stein et al., 1992) reported the presence of reflexes in experiments on the tibialis anterior muscle of complete SCI subjects. They observed that 2 second bursts at 40 Hz and supramaximal stimulation current gave rise to reflexes that could last several seconds. If the bursts were repeated every five seconds, the reflexes died out in the first few repetitions. In our study, the reflex occurred again after one minute allowed between repetitions with a different frequency, suggesting that the modulating effect of a previous stimulation burst during intermittent stimulation is on a short time scale.

Merletti and colleagues (1979) showed the therapeutic effect of FES in reducing spasticity in hemiparetic subjects if treated within six months after the lesion, before high spasticity had developed. The effect could be of limited duration in some cases, while it was long lasting and sometimes even permanent in other cases.

In SCI subjects, muscle contraction resulting from electrical stimulation is not limited to the effect of direct α-motor neuron excitation but is also influenced by reflexive contributions, which may range from small contribution as in able bodied subjects to excessive spastic contractions continuing after the stimulation has stopped. These findings indicate that the interaction with the physiological motor control system is essential to be taken into account when applying FES, as FES may even have a larger training effect than immediate orthotic effect (Ladouceur & Barbeau, 2000).

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