An investigation of the inhibition of voluntary EMG activity by electrical stimulation of the same muscle

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Introduction

In Britain each year there are approximately 100,000 people who suffer their first ever stroke of which approximately two thirds will survive. Of all acute stroke patients starting rehabilitation, about half will have a marked impairment of function of one arm and only about 14 % of these will regain useful function.[1] A significant problem is spasticity, typically causing over-activity in the flexor muscle groups in the upper limb. While often some ability to make a voluntary grip remains, the ability to selectively activate extensor muscles to enable release of a grasp is frequently lost.

Electrical stimulation exercises of the wrist, finger and thumb extensors have been demonstrated to be beneficial in re-educating the ability to open the hand[2]. However the effect may only be short lived, lasting only for the immediate time after the exercise. It has been suggested that if a functional aspect can be added to these exercises, the retraining effect could be improved.   Kraft et al.[3] used residual EMG from the extensor muscles in the forearm to trigger an FES device, stimulating the same muscles. In a comparative study it was shown that hemiplegic subjects had a greater improvement in Fugl-Meyer score than those who received conventional electrical stimulation exercises. In Krafts device the electrical stimulation was only initiated by detection of EMG activity. Our study is to investigate the possibility of using EMG both to control the duration and intensity of the electrical stimulation.

The main problem in recording voluntary EMG from a stimulated muscle is the stimulation artefact, which is 10,000 times larger than the level of the desired signal. This artefact will saturate any standard EMG amplifier. Secondly, following the stimulation pulse is the compound action potential or M wave due to the synchronous firing of motor units and H reflex due to activation of the Ia afferents which is an order of magnitude greater than the voluntary EMG. Difficult problems are often best ignored, so in this case the artefact and M wave can be prevented from entering the system by disconnecting the front end amplifier for 15 – 20 ms following the stimulation pulse. In this period a sample and hold circuit is used to maintain the level of the signal to minimise variation due to the DC off set of the system. However, the instantaneous level of the signal from the front end amplifier is not the same at the end of the blanking period as at the beginning, which results in a step response followed by a decay curve due to the RC time constant of the band pass filter circuitry. This new artefact can again be removed by a second stage of blanking following the band pass and rectifier stages of the circuit. However, to reduce the time period of the artefact still further the lower end of the pass band has the relatively high frequency of 200Hz. This leaves sufficient signal to produce an EMG envelope, which is used to control the stimulator. The stimulator's output can be either driven directly by the envelope producing an output proportional to the EMG envelope or used to trigger a fixed amplitude output for a fixed or adaptive (started and stopped by EMG) time.

The circuit is realised in surface mount technology as a head amplifier (70mm x 35mm) with electrodes attached directly to the under side of the board.
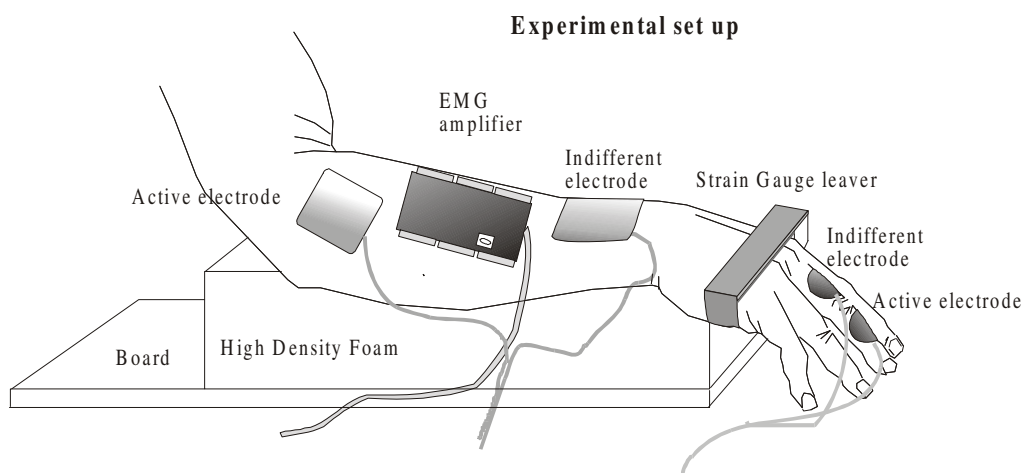
Initial experience

The system was tried by three individuals who have had a stroke. It was found possible to detect EMG in the wrist extensors of all three subjects. However the effort of producing wrist extension had the effect of increasing the spastic tone in the flexor muscles of the hand, sometimes resulting in clawing of the fingers when the extensors were stimulated. This effect was greatest when proportional control was used, as it required the greatest effort by the user. All three subjects were able to use the system to open their hand to acquire objects such as a door handle or large objects such as food cans. Two of the three subjects used the device daily at home. Both reported that their hand felt more relaxed and that they felt more aware of their affected arm than before. However, no significant changes in hand function were recorded using the Jebsen Test.

There was some evidence that users of the EMG systems could learn to relax their muscles after some practice suggesting that using the device may help train self control of spasticity. Additionally, stretch reflexes induced in flexor muscles, being velocity dependent, could be reduced by using a slow rise in the stimulation amplitude to the extensor muscle. This was only possible when the device was triggered rather than proportionally controlled.

Further investigations

It was also observed that the stimulation had an inhibiting effect on the voluntary EMG in normal subjects, effectively reducing the gain of the system as the contraction strength rose. To further investigate the effect, the following system was devised (figure 1). The arm rests on a high density foam support. A strain gauge instrumented lever is placed over the knuckles of the test subject to measure the wrist torque produced. A digital display gives visual feed back of the wrist torque produced. Following opto-isolation, filtered and rectified EMG, wrist torque, and the stimulation synchronisation signal were sampled at 1620Hz and digitised using a PICO ADC11 10 bit A to D module on the parallel port of a PC. The data was read into a Microsoft Excel spreadsheet using a macro written in Visual Basic. A second macro was written to average the data over 26 stimulation pulses. The forearm extensors were stimulated using 300μs pulses at a frequency of 4.6Hz and current amplitudes of up to 80mA. This slow stimulation frequency was chosen because it was observed that the inhibition effect lasts longer that the interpulse interval when a more standard 20Hz frequency was used, in fact in excess of 100ms. 50 x 50 mm Blue Pals stimulation electrodes were used (Axelgaard). The stimulator output stage was a voltage driven transformer. Stimulation current was measured using a Philips PM9355 current probe and a Gould oscilloscope type 1425.
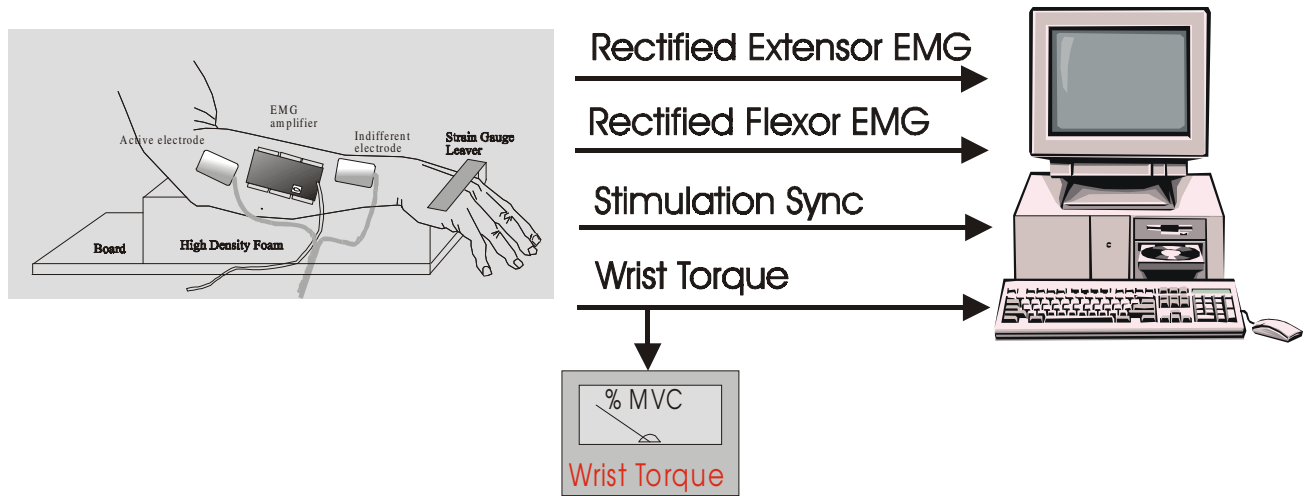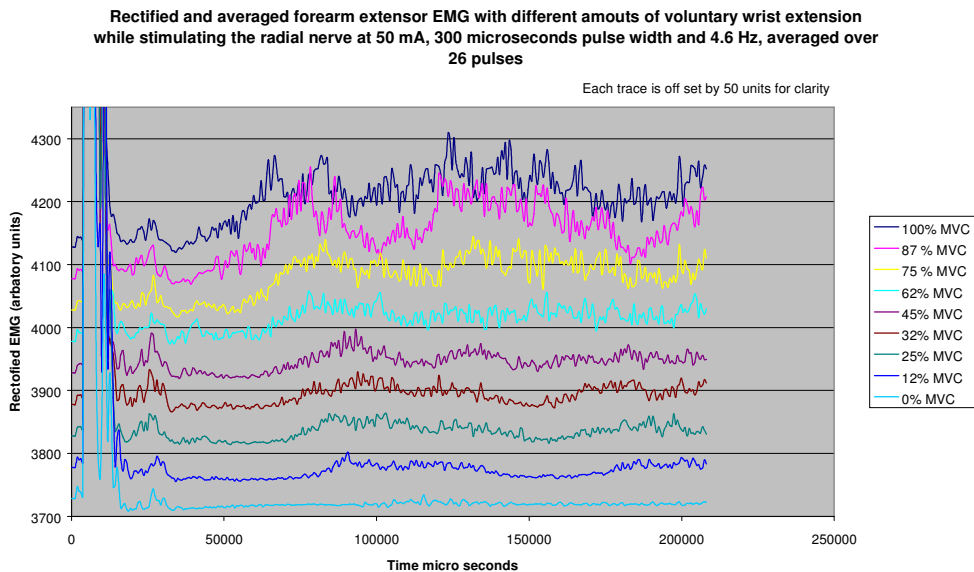
Figure 1

Experimental set up

Active electrode

EMG amplifier

Indifferent electrode

Strain Gauge leaver

Indifferent electrode

Active electrode

Board

High Density Foam

Figure 2
Data Acquisition System

Method.

The effect on voluntary EMG was examined in two ways. Firstly a constant stimulation current was used while varying the degree of voluntary contraction. The subject was asked to extend their wrist and fingers while observing the displayed torque output. The stimulation was then applied for 6 seconds at a predetermined comfortable level. The amount of voluntary effort was increased in equal steps until the maximum voluntary contraction (MVC) was reached. The second method examined the effect of varying the stimulation amplitude while maintaining a constant voluntary effort. The subject was asked to maintain a wrist torque of while the stimulation was increased in 10mA steps.

Finally, an attempt was made to isolate sensory effects of the stimulation from the direct motor effects. It was not found to be possible to find a stimulation site, near that used for the forearm extensors, that did not produce some muscle movement. Electrodes were therefore placed on the ring finger to stimulate the digital nerve. Forearm EMG was recorded as before.

Figure 3

Rectified and averaged forearm extensor EMG with different amouts of voluntary wrist extension while stimulating the radial nerve at 50 mA, 300 microseconds pulse width and 4.6 Hz, averaged over 26 pulses

Results

Extension effects

Figure 3 shows the effect of varying the degree of voluntary effort while stimulating at 50mA (peak current). The subject is a 41 year old male with normal neurology. In this example blanking of only 4ms has been used. The stimulation pulse occurs at 0 on the time scale. After the stimulation artefact and M wave there is a reflex thought to be due to the H reflex, i.e. stimulation of the Ia afferent, which results in excitation of the α motor neurones. The H reflex increases as the voluntary effort increases, indicating that it is facilitated by descending voluntary command. This is followed by a "silent" period, which appears to shorten in duration as the voluntary effort is increased. The silent period has two distinct periods separated by a period of voluntary EMG that may also include a reflex response, just after the end of the each silent period.

Figure 4 shows the effect of varying the stimulation intensity while maintain a constant force. As the current is increased the silent period becomes more pronounced and extends in duration. At higher levels a second period of EMG inhibition appears after approximately 50ms extending for a further 40ms.

Figure 4

Voluntary rectified forearm extensor EMG at 50% MVC with radial nerve stimulation currents of 0 to 50 mA. 4.6Hz stimulation frequency , average of 26 pulses
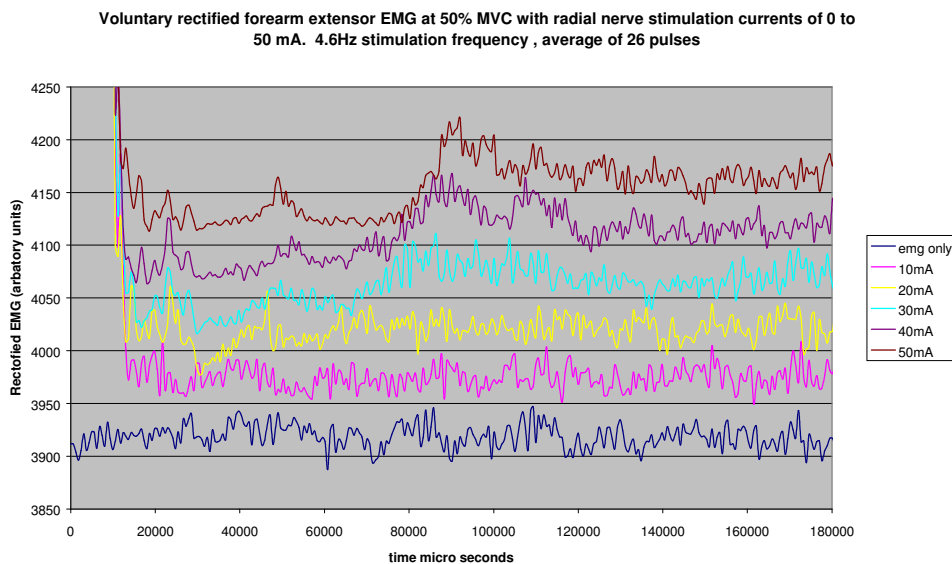
Figure 5 shows a comparison of the inhibition seen when stimulating the wrist extensors and that seen when stimulating the digital nerve. The subject here is a 43 year old female with normal neurology. In both cases a contraction of 50% MVC is produced. In the upper trace the forearm extensors are stimulated at 50 mA while in the lower trace the digital nerve is stimulated at 25mA. Despite this discrepancy, the digital nerve stimulation is perceived as more intense that the forearm extensor stimulation. As in the above cases, the voluntary EMG inhibition is between the H reflex and the second peak and then followed by a second period of inhibition. In the lower trace a similar second period of inhibition of equal duration is seen, delayed by 6ms in comparison to the upper trace.

Figure 5

Forearm extensor voluntory EMG and wrist touque following stimulation of radial and digital nerves
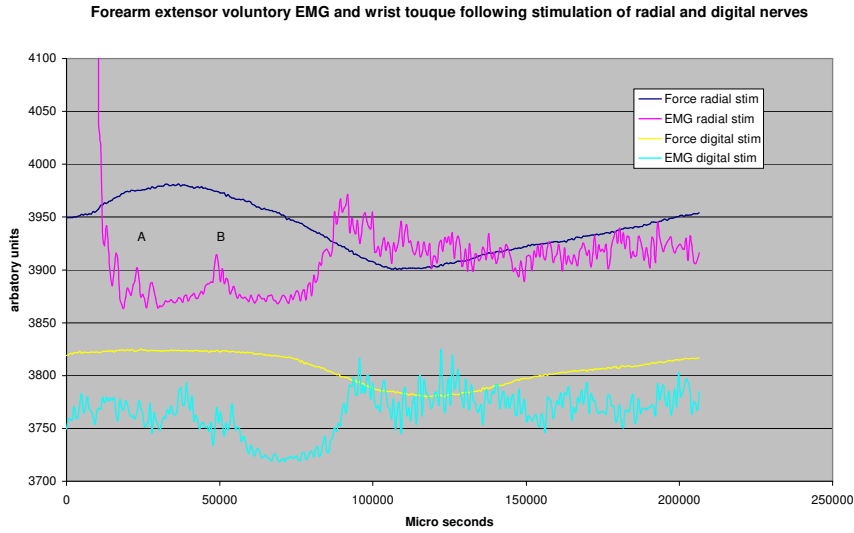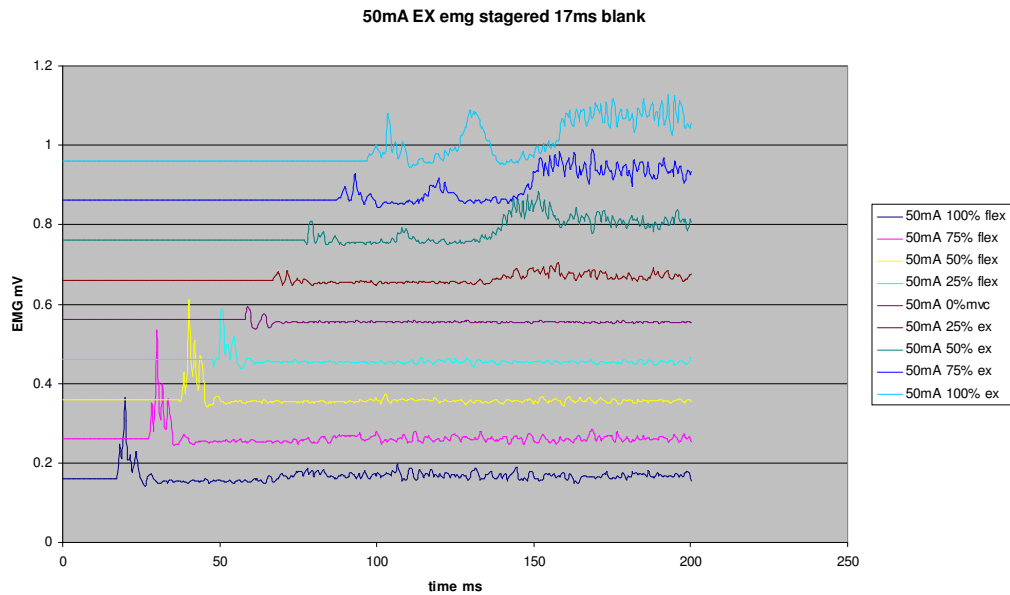
Figure 6 shows the effect of attempting a voluntary wrist and finger flexion while stimulating the extensors. The traces are off set by 10ms and the first 17ms are removed for clarity. The early reflex appears to be modulated by voluntary effort, increasing with either extension or flexion activity.

Figure 6

50mA EX emg stagered 17ms blank

Flexion Effects

Figure 7

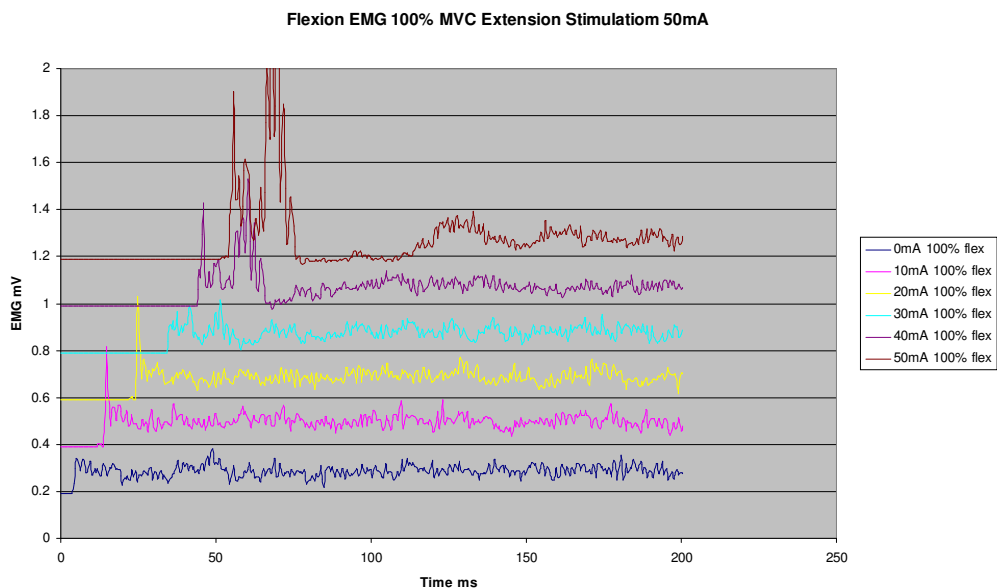**Flexion EMG 100% MVC Extension Stimulatiom 50mA**

Figure 7 shows the EMG from the flexor muscles, stimulating with different stimuli while maintaining 100% voluntary contraction. The traces for each stimulation current are off set so the reflex activity at the start of each trace is not overlaid. A similar pattern to the extensors is seen with a prominent reflex occurring 15 to 25 ms after the stimulation pulse, which increases with stimulation intensity. This may be due to stimulation of Ib afferents from the golgi tendon organs in the extensor muscle, which excites the antagonist muscle via an inhibitory interneuron. There is a silent period following the reflex that becomes bi-phasic at higher stimulation levels.

Figure 8
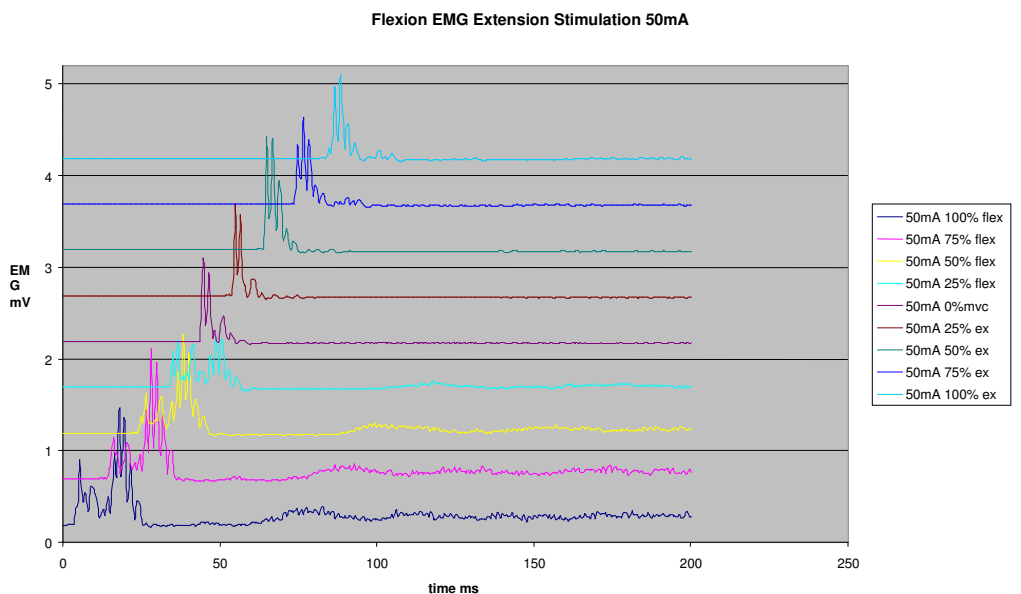
Flexion EMG Extension Stimulation 50mA

Figure 8, which is displayed at a lower signal gain, shows flexor EMG with and extension stimulation amplitude of 50mA with varying amounts of voluntary flexion an extension. In the extension phase, there is a very early reflex visible. In the flexion stage this reflex reduces with increasing voluntary flexion. A later reflex, possibly due to Ib excitation is seen to increase as voluntary extension is increased. The reflex is followed by a silent period, the first part of which may be due to Ia reciprocal inhibition.

Figure 9

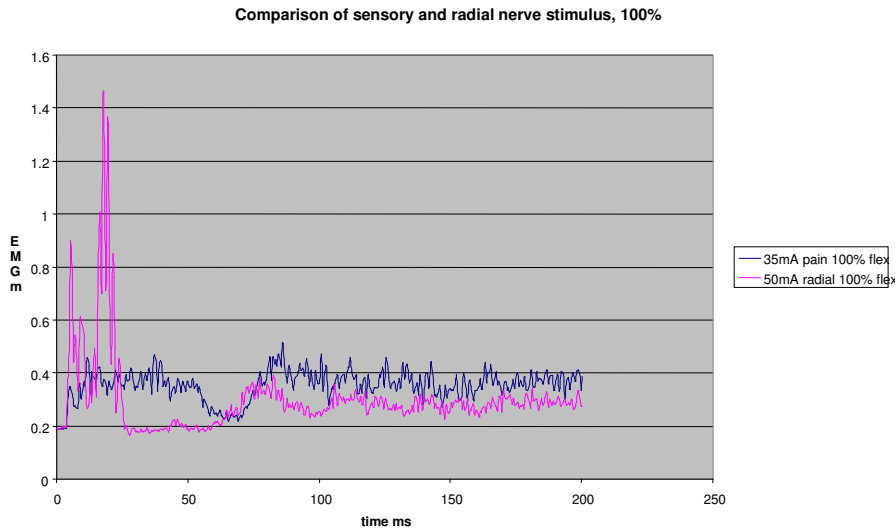Comparison of sensory and radial nerve stimulus, 100%

Figure 9 shows EMG from the flexion muscles obtained under radial nerve stimulation and digital nerve stimulation. As with the extension EMG, there is a period of inhibition after about 50 ms when stimulating the digital nerve (35mA). Again this is slightly longer and later than the inhibition seen when stimulating the radial nerve (50mA).

Discussion

When the forearm extensors are stimulated an antidromic impulse is sent back up the activated alpha motor nerve axons. This collides with and annihilates any motor impulse coming down from the cord, which would have gone towards maintaining the ongoing EMG. Some antidromic impulse may not collide with descending impulses and so may activate the recurrent axon collaterals in the cord to the Renshaw cell, which inhibit the motor neurone. The sudden slight shortening of the muscle unloads the muscle spindles (which are in parallel with the muscle fibres). This results in a brief cessation of activity in the Ia afferents from the spindles. This withdraws a source of activation of the motor neurone. The sudden rise in muscle tension during the electrically induced twitch may activate the Ib afferents in the Golgi tendon organs (which are in series with the muscle fibres). These are inhibitory to the motor neurones. Ib afferents may also be directly stimulated, resulting in a similar effect[4].

The above effects all involve fast conducting type I afferent and efferent fibres and are therefore likely candidates for the explanation of the first period of inhibition. The separation of the second periods seen in figure 4 is consistent with a conduction velocity of about 60ms, suggesting that it too involves fast conducting fibres but may also be due to a longer pathway involving the cortex. It is therefore suggested that the second period may be due to sensory effects.

The effect of inhibition of voluntary EMG activity has obvious implications for using EMG from the same muscle that is stimulated to control a FES device. The effect is to reduce the

gain of the system as the input command increases. Moreover the effect is not linear, changing with voluntary effort and stimulation intensity. The effect of reflexes must also be taken into account, as a positive feedback effect may be possible. On several occasions when testing the system it was found to be difficult to stop the stimulation by removal of the voluntary effort, suggesting that a reflex was being detected as voluntary EMG and therefore providing a continuing input signal. Sometimes it was possible to end stimulation by actively flexing the wrist, suggesting that the reflex may be attenuated by the mechanism of reciprocal inhibition.

Conclusions

A system has been designed for assisted hand opening following stroke controlled by voluntary EMG from the same muscle group that is stimulated. Initial experience has highlighted some areas of difficulty with its use and the effect of EMG inhibition due to electrical stimulation described. While the effect of inhibition has implications for the design of EMG controlled FES devices, studies with people who have spastic hemiplegia are required to illustrate likely differences in reflex and inhibitory effect.

Are there any implications for understanding the effects of electrical stimulation in spasticity from this work? It is likely that the effects would be different with someone who has hemiplegia. It is likely that the observed effects would be subject to different excitatory and inhibitory effects from higher centres. However, the measurements demonstrate that electrical stimulation causes competing inhibitory and excitatory effects in the reflex pathways and by repetition may lead to strengthening of the synaptic connections involved.

References

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