Variation in system gain when using voluntary EMG to control electrical stimulation of the same muscle

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Abstract

While many people who have compromised hand function following stroke are able to produce some voluntary grip, they often have problems overcoming spasticity in the flexor muscles to open the hand. Our aim is to use residual EMG from the extensor muscles to control an EMG device to stimulate the same muscles. However, the voluntary EMG is significantly modulated by stimulation, an effect that is predominantly inhibitory. When voluntary EMG is used as a control input, the gain of the system changes dependant on the intensity of the stimulation and the time since the stimulation pulse. This paper describes these changes in a subject with normal neurology and demonstrates, in this case, that EMG sampled between 40 and 60ms post stimulus will give reliable results up to a stimulation intensity of 30mA. If higher currents were used, EMG sampled between 70 and 80 ms would be required.

1 Introduction

In Britain each year there are approximately 120,000 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 of the posterior interosseous nerve will open the hand by causing extension of the wrist, fingers and thumb. Our aim is to use the residual EMG from the extensor muscle group to control the stimulation, enabling the hand to be opened at will.

Recording EMG from a stimulated muscle presents some difficulty. The first is the presence of the stimulation artefact and M wave, large signals compared to the voluntary EMG signal. Following the stimulation pulse, there is a reverse flow of current and this causes an exponential shift in EMG base line voltage. These effects can be removed by blanking of the amplifier and the use of a high pass filter. A Butterworth filter of 200Hz was used [1]. The second problem is that the voluntary EMG signal is significantly affected by the stimulation. Figure 1 shows a recording of averaged rectified EMG while performing a maximum voluntary isometric contraction of wrist and finger extensors and stimulating with an intensity of 40mA, a pulse width of 300µs, a frequency of 4.8Hz and a blanking period of 4ms. The subject is a 44 year old female with normal neurology.

Figure 1

The first 10ms of the trace is dominated by the stimulation artefact and M wave. At low stimulation intensities the first effect seen is the appearance of the H reflex between 18 and 28 ms after the stimulation. This reflex is modulated by voluntary effort increasing with %MVC but reducing at higher stimulation intensities. The H reflex is followed by a period of inhibition. It is likely the dominant effect in this period is due to Renshaw Cell inhibition, particularly at higher levels of stimulation. Renshaw Cell activation is due to antidromic impulses in the α motor neurones. At higher levels of stimulation a second wave
form is seen at around 50ms post stimulation and is probably due to activation of the type II afferents from the muscle spindles and / or cutaneous afferents. Following this reflex there is a period of inhibition which is related to type III afferents. Finally, there is an increase in activity, which may be a EMG rebound activity [2].

If EMG is used to proportionally control the amplitude of the stimulation, the inhibitory and excitatory effects can be considered as changes in the gain of the system where gain is defined as the measured EMG signal divided by the intended voluntary effort (mV / %MVC). In the absence of stimulation there is a near linear relationship between voluntary EMG and measured force. Therefore the gain of the system is near constant for any given sample of EMG or any degree of voluntary effort. If stimulation causes a reflex to occur, superimposed on the voluntary EMG signal, this can be considered as an increase in gain. However, this increase is transitory, only persisting as long as the reflex lasts. Similarly, inhibition of the EMG signal due to stimulation reduces the measured EMG and so can be considered as a reduction in gain.

2 Method

To examine the change in gain following the stimulation period, the EMG was sampled and averaged in 10 ms windows as illustrated in figure 1. Additionally, the EMG was averaged in a 10 to 80ms window and also a 40 to 70 ms window. Longer periods were not considered as these would require slower stimulation frequencies (<12.5Hz), too slow to produce a fused tetanic contraction.

![Figure 2](image_url)

**Figure 2**

Measurements were performed isometrically using the experimental set up shown in figure 2. 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 feedback of the wrist torque, allowing the subject to maintain the required level of %MVC. Following opto-isolation, filtered and rectified EMG, wrist torque, and the stimulation synchronisation signal were sampled at 2381Hz 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 volunteer, a 44 year old female with normal neurology, was asked to perform isometric contractions of the wrist and finger extensors at 0%, 25%, 50%, 75% and 100% MVC while the extensor group were stimulated. The procedure was repeated for stimulation intensities of 0, 10, 20, 30, 40 and 50 mA.

2.1 Analysis

The averaged rectified EMG traces were split into sample windows shown in figure 1. In each window there were 24 samples. The data samples were then plotted in groups of like stimulation current against %MVC. Figure 3 shows Rectified EMG plotted against %MVC for the 30ms to 40ms window for data recorded at 0mA, 10mA, 20mA, 30mA, 40mA and 50mA. Using linear regression, the best fit line for each group was derived and the gradient, intercept and R² value calculated.

![Figure 3](image_url)

**Figure 3**

3 Results

Figure 4 shows the calculated gain plotted against stimulation current. Figure 5 shows the difference between the estimated Y axis intercept when there is no stimulation compared to stimulating at 10mA, 20mA, 30mA, 40mA and 50mA. Figure 6 shows the calculated R² value plotted against stimulation current.
The predominant effect of electrical stimulation on voluntary muscle activity is inhibition in the period in the period 10 to 80 ms following the stimulation pulse. In the first 10ms following stimulation the recorded EMG signal is completely dominated by the M wave that follows stimulation. Consequently, measured EMG activity is largely related to the intensity of stimulation, illustrated by the shift in y-axis intercepts and there is very little correlation with %MVC. This is also seen to a lesser extent in the 10 to 20 ms window although overall gain reduces with increasing current. At higher stimulation levels this window sees encroachment of the M wave / artefact which lengthens with increased current. This may be due to recruitment of smaller nerve fibres at higher stimulation intensities. These fibres innervate slower muscle, which have a more prolonged M wave. It may be this effect which is dominant at 50mA. The dominant effect seen in the 20 to 30 ms period is the H reflex due to stimulation of the Ia afferents. This reflex is greater at mid to low levels of stimulation. This is because it is subject to antidromic annihilation in the motor neurone nerve. Consequently the gain falls at 40 and 50 mA.

Significant inhibition is seen in the 30 to 40ms period with less inhibition between 40 and 60ms. A second period of inhibition occurs between 60 and 70 ms at the higher stimulation currents only.

### 4 Discussion and Conclusions

At stimulation intensities of 30mA and less, consistent results are seen from 40ms onwards. Above 30 mA best results are seen between 70 and 80ms. This would limit the stimulation frequency to 12.5Hz. Clinical experience from use of the Freehand system shows that it is possible to achieve a fused tetanic contraction at 12Hz once the muscle has been trained [3]. However, many Freehand users experience some degree of vibration at this frequency and prefer the Freehand's alternative frequency of 16Hz. This gives noticeably less tremor than 12Hz as well as a slightly stronger contraction. 16Hz would be feasible, allowing a sample window between 40 and 60ms post stimulation but would limit the system to lower stimulation intensities.

### References


### Acknowledgements

This work was funded by the Wessex Rehabilitation Association and by the Department of Medical Physics and Biomedical Engineering, Salisbury District Hospital.