THE RELATIONSHIP BETWEEN ABNORMAL PATTERNS OF MUSCLE ACTIVATION AND RESPONSE TO COMMON PERONEAL NERVE STIMULATION

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Abstract
This study investigated the relationship between response to common peroneal nerve stimulation and abnormal ankle movement and muscle activation patterns. Response to stimulation was measured by changes in the speed and effort, (Physiological Cost Index) of walking. Abnormal ankle movement and muscle activation were measured in an ankle rig by: ability to follow a tracking signal moving sinusoidally at either 1 or 2Hz, resistance to passive movement and EMG activity during both passive and active movements. Indices were derived to define EMG response to passive stretch, co-activation and ability to activate muscles appropriately during active movement. Different abnormal patterns of muscle activation and statistically significant differences between normal and hemiplegic subjects were identified in calf muscle (p<0.002), but not in anterior tibial muscle activation patterns (p=0.574). Poor voluntary control, abnormal antagonist co-activation and an increased response to passive stretch of the calf muscles was related to good response to stimulation, (p<0.05). Neither mechanical resistance to passive movement nor inability to activate the anterior tibial muscles predicted response. The benefit of common peroneal stimulation may therefore be due to inhibition of spastic calf rather than activation of the anterior tibial muscles.

Introduction
In 1993 a prospective study was conducted in this department1 with a small sample of patients with established hemiplegia and dropped foot. Significant improvement in walking speed and effort was measured when a single channel common peroneal stimulator was used to improve ankle dorsiflexion during walking. Closer examination of the results identified that, although 11 patients experienced more than 10% improvement in walking parameters two had minimal benefit and three none at all. These results posed the question - could patients who were likely to benefit from FES be identified by their pattern of abnormal muscle activation?

Patients with hemiplegia as a result of an UMN lesion are usually assumed to have spasticity characterised by brisk reflexes, clonus and an increased stretch reflex. Other components of the UMN syndrome, such as muscle stiffness, inappropriate activation or poor voluntary control may also be important factors influencing movement patterns2,3 and response to FES.

Method
Patients with hemiplegia (post six months), who had a dropped foot, and twelve age matched normal subjects were recruited. Hemiplegic subjects were monitored for four weeks before using FES. Stimulation was used in the same way as in the previous prospective study using the ODFS II. An adjustable rate of stimulation rise and fall of between 0 and 4s accommodated calf spasticity at the start and end of stimulation.

Measurement of muscle activation patterns
Ankle angle, resistance to passive movement and muscle activation patterns (EMG) were monitored in an ankle rig that supported the lower leg and foot, and was hinged to allow movement in the sagittal plane4. The ankle was moved, either by the subject themselves.
(active test) or by the researcher (passive test). The task was to keep the ankle, represented by a cross on a computer screen; inside a computer generated elliptical target that oscillated in Simple Harmonic Motion across the screen. Frequency of oscillation was either 1 or 2Hz. A strain gauge, close to the fulcrum, on the lever that moved the ankle, measured the torque (Nm) exerted when the ankle was moved passively. Surface EMG signals from the calf and anterior tibial muscles were recorded using Medelec contact electrodes. The signals were rectified, amplified and filtered. Voltages from each of these sensors were recorded digitally on video tape through a ‘Softel’ datalog recorder. Sample rate was 500 / s. Tests were performed through an arc of 30 degrees.

**Analysis of the ankle rig data and calculation of the indices**

Samples of data, retrieved from the video recordings, were analysed in Microsoft Excel

*Derivation of the indices using the EMG signals*

To overcome the problem of variations in EMG amplitudes associated with repeated surface recordings, muscle activity was quantified using ratios of EMG activity during two defined periods of movement. In the passive task peak torque was measured when the ankle was moved into dorsiflexion (TQ). The stretch index was derived to quantify the reflex muscle activity in the calf muscles, during passive stretch (SI).

\[
\text{Stretch Index (SI)} = \frac{\text{Sum of the EMG activity in the stretch phase}}{\text{Sum of the EMG activity in the shortening phase}}
\]

*Voluntary Tracking (VT)*

Accuracy in following the tracking signal (VT) was defined as: the ratio between the tracking signal peak and goniometer peak

*Calf Modulation index (CMI)*

When a normal subject follows the tracking signal reciprocal inhibition is seen between antagonist muscles. In subjects where co-activation was identified (calf activity during active dorsiflexion), it was quantified by the following ratio. In normal reciprocal inhibition therefore the CMI would be small

\[
\text{CMI} = \frac{\text{Sum of calf EMG activity from mid position to max dorsiflexion}}{\text{Sum of calf EMG activity from mid position to max plantarflexion}}
\]

*Tibialis anterior modulation index (TAMI)*

\[
\text{TAMI} = \frac{\text{Sum of EMG activity from peak dorsiflexion to peak plantarflexion}}{\text{Sum of EMG activity from peak plantarflexion to peak dorsiflexion}}
\]

identified pattern of activity in the anterior tibial muscles during active ankle movement. No subjects, either normal or hemiplegic, had more than minimal co-activation between the anterior tibial and calf muscles during plantarflexion. A high TAMI was therefore considered to represent poor activation of the anterior tibial muscles during dorsiflexion.

**Design of the study**

An AB, case study design was used, subjects were assessed three times over a period of between four weeks to determine baseline values (period A). The ODFS was then set-up and adjusted and subjects reassessed after one and three months use of the stimulator (period B).
Outcome measures
Response to stimulation was measured by changes in walking speed and Physiological Cost Index (PCI), measured both with and without stimulation over a 10 metre walk.

Statistical analysis
Data were not normally distributed, therefore non-parametric tests were used. Median and mean values have been quoted. Relationships between normal and hemiplegic subjects and between variables within groups were measured using Spearman’s rank correlation coefficient.

Results
Analysis of the data indicated that the cause of drop-foot was not necessarily an inability to activate the ankle dorsiflexors during walking\(^2,3\). Different types of abnormal muscle activation were identified and a correlation between type and response to stimulation was identified. Normal data was used to define the normal range for each of the indices. Values and indices derived identified significant differences between hemiplegic and normal subjects in all baseline parameters except the tibialis anterior modulation index (TAMI).

Identification of abnormal patterns of muscle activity
Each subject was classified for each variable. An abnormal index or value was defined as one that was on the abnormal side of the median. The following categories were used:
- Those unable to follow the tracking signal (VT)
- Those with a stretch response (measured by stretch index during passive movement) (SI)
- Those who were unable to activate the anterior tibial muscles appropriately (TAMI)
- Those with co-activation of the calf during active dorsiflexion (CMI)
- Those with a high resistance to passive movement (TQ)

All subjects who were able to follow the tracking signal were also able to activate their anterior tibial muscles appropriately, the converse was not true suggesting that other factors such as co-activation and mechanical resistance to movement may influence the subjects’ ability to follow the tracking signal.

A significant correlation (at the 0.05 level) was identified between the following variables:
- TAMI and VT (0.67) suggested that the ability to activate the anterior tibial muscles appropriately was an important factor in control of voluntary movement independent of other abnormalities.
- TQ and VT (0.56) suggested that ankle stiffness hindered control of voluntary movement.
- Baseline PCI and the following variables - VT 1Hz (0.69) co-activation (0.59), TAMI (0.54) suggesting that inability to control voluntary movement and inappropriate calf activity during movement increases the effort of walking.

Changes in walking speed and PCI
A statistically significant increase in walking speed and reduction in PCI was measured when the stimulator had been used for three months. An orthotic effect, measured by improvement in these parameters when the stimulator was used and a therapeutic effect, measured as improvement after three months, when the stimulator was not switched on.

Relationship between baseline measurements and response to stimulation
Statistical tests suggested that the following baseline parameters would predict a good orthotic effect, measured by increase in walking speed with stimulation at the three month assessment:
• An inability to actively dorsiflex the ankle (0.721 p<0.05)
• Calf co-activation at 1Hz (0.658 p<0.05)
• A high stretch index. (0.459 p<0.05)
• A low baseline walking speed (0.488 p<0.05) and a high baseline PCI (0.760 p<0.05)

Subjects were therefore classified into subgroups to identify whether response to stimulation was indeed affected by type of abnormality.

Table 1. Numbers of subjects in subgroups who had above average (median) improvement in walking speed when the stimulator was worn measured at the fifth assessment (S5-NS5)

<table>
<thead>
<tr>
<th>Sub group</th>
<th>No. of subjects in sub group</th>
<th>No. of subjects with above average improvement</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal stretch index</td>
<td>12</td>
<td>3</td>
</tr>
<tr>
<td>High torque and normal stretch index</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Able to follow the tracking signal *</td>
<td>5</td>
<td>0</td>
</tr>
<tr>
<td>Unable to follow the tracking signal and co-activated</td>
<td>6</td>
<td>4</td>
</tr>
<tr>
<td>Unable to follow the tracking signal and had a high stretch index</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>Unable to follow the tracking signal, co-activated and had a high stretch index</td>
<td>3</td>
<td>3</td>
</tr>
</tbody>
</table>

* Only one subject who was able to follow the tracking signal also had high torque.

Discussion

Different types of abnormal patterns of muscle activation were identified. - increased stretch reflex, co-activation and weakness. Mechanical changes in the muscle sometimes caused an increase in torque, unrelated to muscle contraction.

The results of this study suggest that, although inability to actively dorsiflex the ankle is a predictor of positive response to stimulation, people who have inappropriate calf activity, either an increased response to stretch or co-activation, also respond well. In these cases Stimulation may be effective through inhibition of the calf⁶ as well as excitation of the anterior tibial muscles. In cases where rapid ankle dorsiflexion causes a stretch reflex in the calf, stimulation may be ineffective unless a slow rising ramp is used to produce a slower ankle dorsiflexion.

References