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Communication

Indices to describe different muscle activation patterns, identified during treadmill walking, in people with spastic drop-foot☆

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Abstract

This study was concerned with individuals who were unable to effectively dorsiflex their ankle when walking, as a result of a lesion of the central nervous system (CNS). Indices that categorise and quantify different patterns of calf and anterior tibial muscle activation patterns during treadmill walking have been derived from a sample of fifteen individuals with established hemiplegia following stroke and twelve age-matched individuals without impairment. As subjects walked on a treadmill, force sensitive foot-switches under the heel and first metatarsal head allowed EMG signals from the calf and anterior tibial muscles to be related to phases of the gait cycle. Normal activation periods for each muscle group were identified as percentiles of the gait cycle and indices for muscle activation periods were derived using ratios of integrated EMG during selected periods.

Indices were derived that identified statistically significant differences, between normal and hemiplegic subjects, in calf activation during both push-off phase ($P<0.001$) and early stance phase ($P<001$), but not activation of tibialis anterior during swing ($P=0.325$) Observation suggested that integrated tibialis anterior activity during swing phase in hemiplegic subjects was not dissimilar to normal subjects, but the profile in hemiplegic subjects tended to lack the normal second peak of activity at initial foot contact.

The reasons for drop-foot were shown to be varied and complex. The indices defined may be useful for directing therapy and measuring outcome. © 2001 IPEM. Published by Elsevier Science Ltd. All rights reserved.

Keywords: Hemiplegia; Spasticity; Gait; EMG; Muscle activation patterns

1. Introduction

Lesions within the central nervous system (CNS), such as stroke, often result in abnormal gait patterns, particularly ineffective ankle dorsiflexion during swing phase and failure to achieve heel strike at initial floor contact; dropped foot. These abnormalities are associated with a complex pattern of dysfunction including spasticity, muscle weakness, impaired sensory-motor control and long-term mechanical changes in muscles and joints [1–5]. Treatment of these conditions may employ a variety of interventions; including physical therapy, orthotic, systemic and local medication and functional electrical stimulation (FES) [6], but the choice of treatment is often subjective. It is proposed that a better understanding of muscle activation patterns during walking may assist in the selection of treatment and allow objective evaluation of outcome.

In normal walking, the primary roles of muscle activity at the ankle are for foot clearance during swing phase, weight acceptance during stance, and propulsion at push-off. From typical sample EMG recordings in normal human gait, the interplay between, tibialis anterior and triceps surae, can be shown [7]. In tibialis anterior there is a strong burst of activity immediately after heel strike controlling plantarflexion. Muscle activity then remains low until toe-off. At toe-off, this muscle becomes active again to lift the foot clear of the ground; peaking during early swing. In triceps surae, there is a gradual increase in activity as the muscle eccentrically controls passive dorsiflexion as the body's centre of gravity moves forward over the foot during

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stance. This activity acts to maintain stability during stance. There is then a strong period of concentric activity from just before heel-off to just before toe-off, termed the push-off phase [8]. Following a lesion within the CNS, the interplay between these two muscles may be disturbed by, increased sensitivity to passive stretch, inappropriate co-activation, loss of voluntary activation and weakness of muscles [1,2,4]. Although the cause and consequences of a dropped foot cannot be considered solely at the ankle, describing ankle muscle activity may provide useful information for the development of a clinical tool to direct and evaluate treatment.

Earlier researchers have studied the timing and amplitude of muscle activity during walking in normal and hemiplegic patients [9,10], but little work has been done to categorise different types of abnormality in hemiplegia.

Dietz showed that, whereas in normal subjects increased ankle torque during stance phase was concurrent with activity in triceps surae, in some cases of equinus abnormality, no relation was observed [5]. Dietz deduces that in these cases, increased ankle torque is due to mechanical tension in triceps surae; a phenomenon that he terms the pseudo stretch reflex. This suggests that hypertonicity may be due to a combination of dynamic factors — increased tonic stretch reflex and co-activation and mechanical changes within the muscle and structures around the joints.

The multi-dimensional nature of spasticity has also been investigated by Knutsson, who identified different types of abnormal muscle activation patterns during walking [1]. Knutsson observed that hemiplegic patients tended to fall into one of three types.

Type I — those whose predominant abnormality was premature calf activation.
Type II — those who demonstrated co-activation between tibialis anterior and triceps surae.
Type III — those whose predominant abnormality was lowered EMG activity in various muscles.

This study will develop the work of Knutsson and follows on from previous work by the authors [11–13] who used ratios of integrated EMG during defined phases of movement to describe patterns of modulation between tibialis anterior and triceps surae during passive and active ankle dorsiflexion in an ankle rig.

2. Objective

To define indices to describe patterns of calf and anterior tibial muscle activation in normal and hemiplegic subjects during treadmill walking.

2.1. Subject selection

Fifteen subjects with residual hemiplegia after stroke (more than six months after onset) who had been referred for treatment with a common peroneal nerve stimulator were invited to take part along with twelve unimpaired subjects of similar age. Each hemiplegic subject was unable to achieve normal heel strike when walking unaided. Some subjects made initial floor contact with the toe, others with the heel and toe together. All subjects were able to walk 10 m; walking aids permitted, and had no other serious medical conditions likely to influence walking or response to treatment.

3. Method

Local Research Ethics Committee approval was obtained for the study. Tests were conducted on subjects as they walked on a treadmill. The speed of the treadmill was slowly increased, until a comfortable walking speed of between 1 and 2 km/h was reached. Each subject was reassured that the treadmill could be stopped at any time and they were encouraged to use the hand support for stability. Using a treadmill enforced a more rhythmic gait on subjects than they may have achieved during ‘free’ walking.

Force sensitive resistor (FSR) footswitches were placed in the shoe to identify specific points in the gait cycle. A switch under the heel identified heel strike/rise and, because of the unreliability of measuring the real time event of toe strike and rise, the ‘toe’ switch was positioned under the first metatarsal head. Thus this switch represented toe rise and strike. Potential inaccuracies that might have occurred using this method, or by switches not being triggered until sufficient force was applied, were avoided by using the same footswitch positions on all occasions and in all subjects — both normal and impaired.

Surface EMG signals were recorded from tibialis anterior and triceps surae muscles using *Medelec* EMG amplifiers. Sample rate was 500 Hz. The skin was prepared by gentle abrasion and wiped with surgical spirit. Activity from tibialis anterior was recorded 1.5 cm from the anterior border of the tibia at the junction between the upper and middle third of a line between the tibial tubercle and the inter-malleolar line. Activity from triceps surae was recorded 1.5 cm, lateral to a line between the tendo-achilles and the popliteal crease at the junction between the upper and middle thirds. In this position signals from the lateral head of gastrocnemius and from soleus would have been detected. Signals were pre-amplified at source (gain × 15, bandwidth 10–1000 Hz, common-mode rejection 100 dB at 50 Hz). Further amplification was provided, before each signal was rectified, then integrated using a low pass filter, using a time

constant 13.8 s. Before the subject began walking the EMG signal was viewed on an oscilloscope to ensure that there were no artefacts. Appropriate adjustment was made to the electrode positions.

When the subject was walking at a comfortable speed, approximately 3 min of data were recorded. Raw and integrated EMG signals and FSR signals were digitised at a rate of 500 samples/second and recorded on the text lines of a video-tape using the *Softel* system. A five-second sample was selected from the earliest stable part of the recording, as representative of the subject's walking. This reduced the risk of contamination of the sample due to either fatigue or habituation. Three strides could be identified from each five-second sample in most subjects, two where walking was very slow. Two or three samples of data therefore allowed six strides to be averaged using the software.

Software, written in *Microsoft Excel Visual Basic*, was used to analyse the data from the walking test. Smoothing of foot switch and EMG data was applied in software to remove erroneous spikes. Transition times of heel and 'toe' switches were identified from these complete gait cycles. Using the RMS EMG envelope, activity in the triceps surae and anterior tibial muscles could be related to phases in the gait cycle. Precise activation times were defined using percentiles of the complete gait cycle. EMG signals were integrated between the defined sub periods of the gait cycle and activity expressed as a ratio of the activity over the complete gait cycle. Ratios, rather than absolute measurements were used, to eliminate variations due to electrode contact, skin condition, depth of subcutaneous fat and proximity of electrodes to motor points encountered when comparing different subjects, and the same subject on different occasions.

3.1. Derivation of indices

Indices to describe EMG activity during treadmill walking were based primarily on visual inspection of repeated EMG–FSR graph data from the 12 normal subjects, using time-correlated muscle activity and foot-switch conditions. From visual observations of EMG and foot-switch data from hemiplegic subjects walking, three areas of abnormality were identified:

- activity in tibialis anterior during the swing phase;
- premature calf activity in early stance phase;
- calf activity during push-off.

Indices were then derived to identify and quantify these. Data collected from the normal subjects was used to define the normal range for each index.

3.2. Activity of tibialis anterior during swing phase

From normal EMG recordings tibialis anterior was seen to be active during the swing phase, with two bursts of activity. The first at the toe rise to lift the foot clear of the ground, the second from just before heel strike to toe strike to control ankle plantarflexion. Tibialis anterior activation index (TAAI) was used to represent this, and was defined as:

$$\frac{\text{Sum of TA activity between toe off and toe strike}}{\text{Sum of TA activity during one complete gait cycle}}$$

In normal subjects, it would be expected that the index would be close to unity, whereas in subjects who are unable to effectively activate this muscle at the appropriate time, the TAAI would be smaller.

3.3. Premature calf activity in early stance

In normal subjects, the calf muscles are, except for some co-activation with tibialis anterior to stabilise the ankle during initial floor contact, almost silent during the first 20% of the gait cycle, as shown in Fig. 1. Abnormal premature calf activity may occur in early stance when the ankle is passively dorsiflexed by the forward movement of the body over the foot. Activity may be caused by an increased reflex response to passive stretch, typical of spasticity. The premature calf activation index (PCAI) was therefore defined as:

$$\frac{\text{Sum of calf EMG toe strike +20\% of the gait cycle}}{\text{Sum of calf EMG during one complete gait cycle}}$$

In normal subjects we would anticipate that the PCAI would be small, whereas in subjects who present with premature calf activity it would be increased towards unity.

3.4. Calf activity at push-off

In normal subjects, the calf becomes active during terminal stance phase, raising the subject's centre of gravity. Without this activity forward propulsion relies on the pseudo-stretch reflex observed by Dietz [5] and torque about the ankle is generated by mechanical constraint in the calf muscle. From the tests with normal subjects, the average period of calf activity was defined as 11% of the gait cycle before heel rise to 9% of the gait cycle after heel rise. The push-off index (POI) was therefore defined as:

$$\frac{\text{Sum of call EMG from 11\% ofgait cycle before heel rise to 9\% of gait cycle after heel rise}}{\text{Sum of calf EMG during one complete gait cycle}}$$

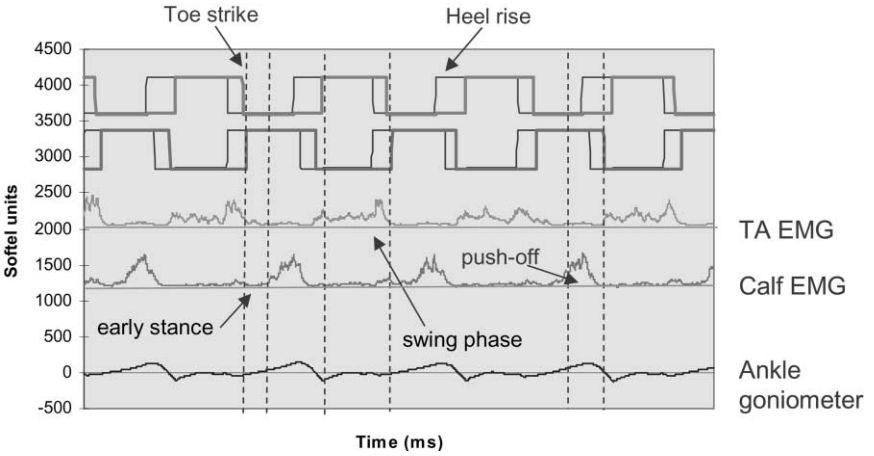

Fig. 1. Illustrates activity in the gastrocnemius and tibialis anterior muscles and their relationship to foot-switch conditions in a normal subject (subject 12) during treadmill walking. Minimal co-activation is observed between the two muscle groups and minimal calf activity during early stance phase. In this example POI=0.85, PCAI=0.06. (Mean normal POI=0.67; PCAI=0.12).

In normal subjects who have an effective push-off it was anticipated that this index would be close to unity, whereas in subjects with a less effective push-off and calf activity at terminal stance it would be smaller.

3.5. Statistical analysis

Data was not normally distributed. Non-parametric statistical tests to measure differences between normal and impaired subjects were therefore applied. Each hemiplegic subject was classified for each type of muscle activation pattern (index) into three categories: normal, mild and severe abnormality. For each index, values on the abnormal side of the median identified a mild abnormality, on the normal side no abnormality. Where the index fell within the first quartile range the abnormality was classed as 'severe'. For some indices higher values represented greater abnormality in others abnormality was identified by a low index value. More specific classification was thought to be innappropriate bearing in mind the small size of the sample (15 impaired and 12 normal) and lack of previous experimental evidence.

4. Results

Table 1

4.1. Normal muscle activation patterns

Fig. 1

4.2. Measured indices of muscle activity

Table 2

4.3. Premature calf activation

Although some unimpaired subjects demonstrated some co-activation between the calf and anterior tibial muscles, the PCAI measured in the sample of hemiplegic subjects was statistically significantly higher than in the sample of unimpaired subjects as shown in Table 2.

All but one of the subjects who had a PCAI in the last upper quartile made initial foot contact with the toe. It should however be noted that premature calf activation was not the only cause of this. Mechanical restraint should also be considered, and, in two subjects who had low TAAI, it was more likely to be due to an inability to maintain activation of the anterior tibial muscles.

Where premature calf activity was seen in hemiplegic subjects it was sometimes prior to, or at the same time as, toe strike, when it often appeared as a spike of short duration, lasting until the heel struck the ground as shown in Fig. 2. In Fig. 3, however, premature calf activity during the first 20% of stance phase was more likely due to a stretch response during passive ankle dorsiflexion when both heel and toe were on the ground.

4.4. Tibialis anterior activation index

In seven subjects the TAAI indicated that the activity in tibialis anterior during the swing phase of walking was poor (<1SD below the normal mean). In the example shown in Fig. 4, the amplitude of tibialis anterior activity was reduced, less modulation was seen, and activity began and ended prematurely. In this sample TAAI was 0.26. However, the TAAI did not distinguish normal from hemiplegic subjects. Although the TAAI was useful to identify subjects who were unable to activate their anterior tibial muscles during swing, it cannot be assumed that a normal TAAI was indicative of a normal profile of activity. For example, in some subjects who had normal, and sometimes at the upper range of normal TAAI, the profile of activity differed from that of normal subjects. In the case of one subject who had a TAAI of 0.87 (normal mean, 0.64), although tibialis

Table 1
Demographic data of the normal and hemiplegic subjects who took part in the study

	Mean age at start of trial	Sex	Cause of stroke	Side of hemiplegia	Mean time from stroke
Subjects (n=15)	60 years 10 months	11 male, 4 female	8 infarct, 3 haemorrhage, 3 unknown, 1 emboli	10 right, 8 left	2 years 11 months
SD	11 years 6 months				3 years 2 months
Normals (n=12)	55 years 6 months	5 male, 7 female			
SD	16 years 2 months				

Table 2
Showing the median, IQR (inter-quartile range) max and min values of each index in normal and hemiplegic subjects, the results of the statistical tests and the classification of abnormality into moderate and severe

	Tibialis anterior activation index (TAAI)		Calf push-off index (POI)		Premature calf activation index (PCAI)	
	Normal	Hemiplegic	Normal	Hemiplegic	Normal	Hemiplegic
Median	0.70	0.64	0.68	0.26	0.12	0.34
IQR	0.12	0.17	0.12	0.05	0.06	0.07
Max	0.87	0.88	0.83	0.40	0.20	0.45
Min	0.53	0.26	0.41	0.19	0.04	0.22
Median+IQR	0.82	0.81	0.80	0.31	0.18	0.41
Median−IQR	0.58	0.47	0.56	0.21	0.06	0.27
Mann–Witney U-test hemiplegic vs. normal impairment score		P=0.325		P<0.001		P<0.001
Range for moderate abnormality		0.64–0.47		0.26–0.21		0.34–0.41
Range for severe abnormality		<0.47		<0.21		>0.41

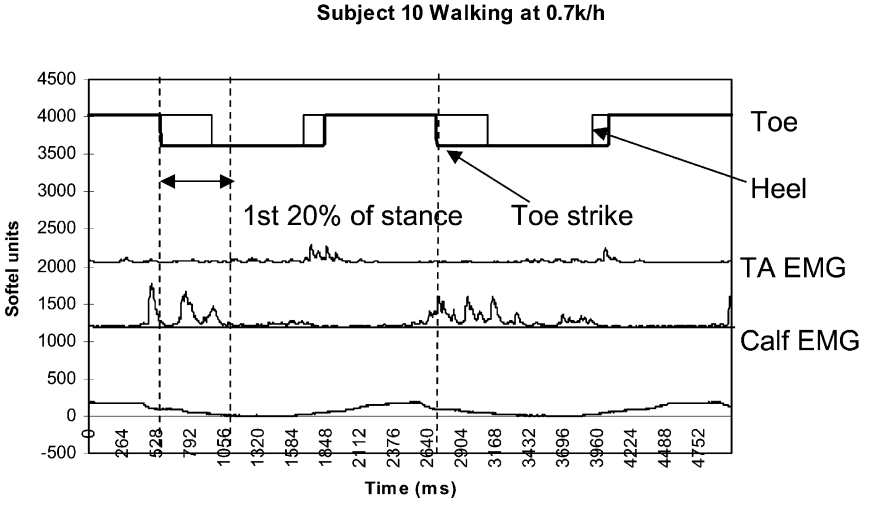

Fig. 2. Illustrates a subject (subject 10) with a high premature calf activation index (PCAI) 0.62. In this example the toe struck the ground before the heel and was preceded by ankle plantarflexion and calf activity. The calf continued to be active during stance, but there was minimal activity at push-off (slightly more on the second step than the first). Push-off index (POI) in this example was 0.18.

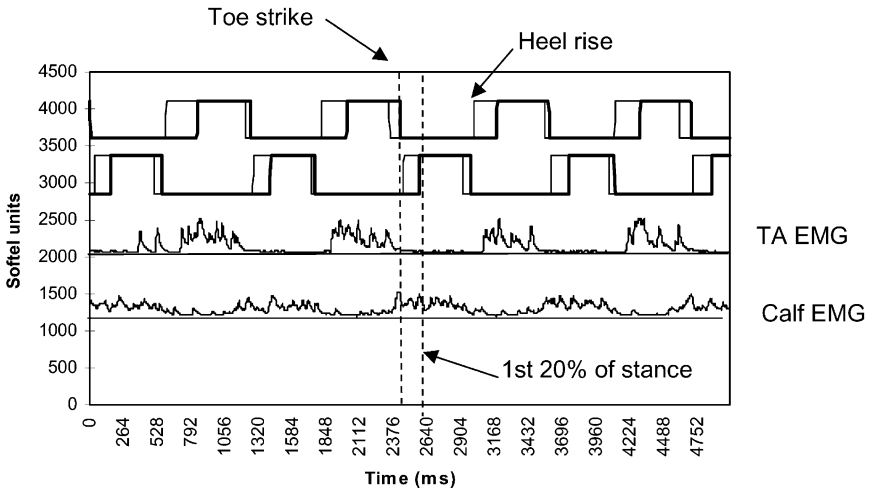

Fig. 3. Illustrates a subject (subject 16) with a premature calf activation index (PCAI) of 0.34. Heel struck the ground just before toe. The calf was active during the first 20% of stance phase, but not before initial ground contact. Tibialis anterior was active during swing, but activity ended before heel strike.

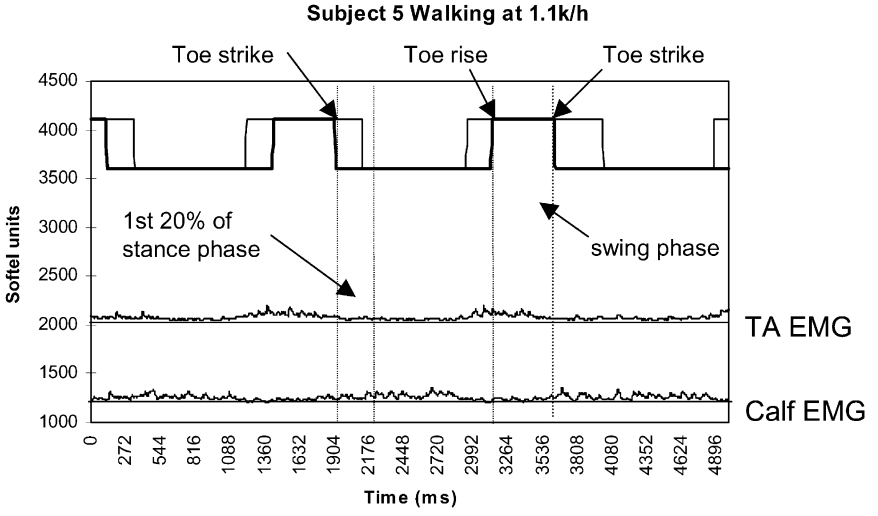

Fig. 4. Illustrates a subject with a low TAAI (0.26). Amplitude was reduced, there was no second peak of activity at initial floor contact and the toe struck the ground before the heel. Little calf modulation was observed, but sufficient to give a push-off (POI=0.25).

anterior activity began appropriately it declined before heel strike, as shown in Fig. 5.

4.5. Push-off index

The POI was always greater in normal than in hemiplegic subjects as shown in Table 2. Among the hemiplegic subjects, some demonstrated little calf activity during any period of the gait cycle, as shown in Fig. 4. whereas in others modulation was seen but activity began earlier than normal as shown in Fig. 5, and diminished before heel rise. The POI did not distinguish between these two abnormalities; the calculated indices for these two cases being 0.25 and 0.24, respectively.

5. Discussion

Analysis of the data from all hemiplegic subjects indicated that the cause of drop-foot was not necessarily an inability to activate the ankle dorsiflexors during walking. Different types of abnormal muscle activation were identified in different individuals, but individuals were not observed to change from one type to another. In some individuals it was not possible to identify a single mechanism for their foot drop; they did not fall clearly into one of Knutsson's three categories. From further examination of the results, it was apparent that the indices alone did not present the complete picture of muscle activation. Nevertheless the indices identified significant

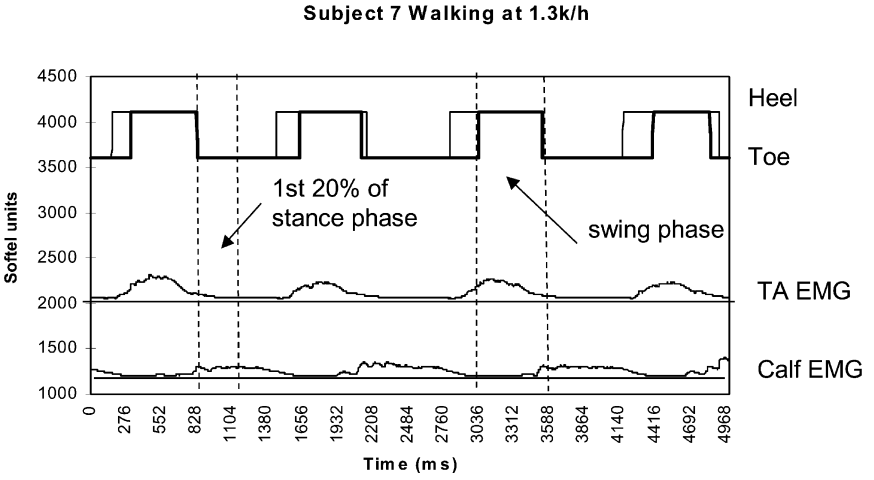

Fig. 5. Illustrates the subject with the highest tibialis anterior activation index (TAAI) and EMG amplitude near to normal. The second peak, seen in most normal subjects, was absent and although activity began appropriately it declined before toe strike. In this example, therefore, a normal TAAI did not indicate normal activity. The effect of the early decline in tibialis anterior activity may have accounted for the almost synchronous heel and toe strike. Calf modulation was observed but activity began earlier than normal (POI=0.24).

differences in calf activation between hemiplegic and normal subjects and whether this was due to lack of activity at push-off, inappropriate activity in early stance or both.

Muscle activation periods were defined by foot-switch conditions. It has been acknowledged that footswitch conditions did not correspond with actual toe and heel contact times and that position of the switches influenced related muscle activation periods and thus the index value. Inaccuracies may have occurred particularly if initial floor contact was with the lateral border of the foot (due to inversion at the ankle) followed by contact with either the 1st metatarsal head or heel. Because the 'toe' switch was positioned under the first metatarsal head the recorded initial contact time would in these cases have been later than the real contact time. Using a third switch or an array of switches might provide more accurate information and should be considered in future applications.

Indices related to the amount of muscle activity, thus PCAI is small for normal subjects — as activity during this period of the gait cycle in abnormal. Conversely, the TAAI and POI are large in normal subjects as activity is expected during these periods. If the indices were to be used clinically this method might be confusing and values would be better described so that either 0 or 1 always defined 'normal'.

Premature calf activity was observed in some cases prior to initial foot contact. This observation can also be seen in normal subjects walking on their toes and could therefore be considered a normal response to an abnormal gait pattern (Dietz — personal communication, July 1998). It is therefore possible that this phenomenon was distinct from premature calf activity observed earlier than normal but after toe strike. The latter is likely to be associated with the passive stretch of the calf as the body moved forward over the fixed foot and may explain the correlation between the premature calf activation and an increased stretch reflex [11].

TAAI profile was in some cases abnormal, lacking the double peak seen in normal subjects, and yet these subjects had a normal TAAI. The absence of the burst of activity normally occurring at heel strike may account for their resultant flat foot walking. Furthermore, it is important to acknowledge that a low TAAI does not necessarily indicate a weak tibialis anterior, rather an inability to activate the muscle appropriately during walking.

The study was conducted using a treadmill. This allowed walking speed to be monitored and controlled, but the patterns of muscle activation observed may have been different from those observed during floor walking. A review of the literature comparing treadmill with floor walking [14,15] did not allow clear conclusions to be made but obvious differences such as the conflict between visual information and sensation of movement and the speed being enforced rather than selected cannot be ignored. It was considered important to analyse samples taken early in the walking test to avoid habituation [15].

A further weakness of the study that should be acknowledged is the variation in walking speed. A compromise was made between asking all subjects, both normal and hemiplegic, to walk at the same speed and allowing free choice of walking speed. To accommodate the slowest hemiplegic walking speed would have enforced an unnaturally slow speed on most other subjects, therefore all subjects walked at between 1 and 2 km/h.

The indices derived in this study have been applied in a further research project investigating the use of Botulinum Toxin A (BTXA) in the treatment of calf spas-

ticity. In this study it will allow the response to BTXA to be related to the pre-treatment calf activity during walking and to detect changes in pattern of activity as a result of treatment. For the indices to be more widely used they will need to be validated with a large sample of normal and spastic subjects and against other conventional measures of spasticity.

6. Conclusions

Indices to describe calf and anterior tibial muscle activity during walking have been derived and tested with a small sample of normal and hemiplegic subjects. Statistically significant differences in the pattern of calf activity emerged between the normal and hemiplegic subjects but the indices did not detect a significant difference in tibialis anterior activity, despite clearly different profiles.

The study has highlighted variation between subjects, all of whom had a dropped-foot associated with hemiplegia following stroke. An interesting observation has been that in many cases innappropriate calf activity may contribute to the problem as much as, if not more than, an inability to activate the tibialis anterior muscle.

Acknowledgements

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