Chapter 3

Abnormalities in the temporal patterning of lower extremity muscle activity in hemiparetic gait

accepted pending revisions
Abstract

Following hemiparetic stroke, the timing of lower extremity muscle activity during gait often undergoes radical changes. In the present study, we compared the duration of activity in Biceps femoris (BF), Rectus femoris (RF), Tibialis anterior (TA) and Gastrocnemius medialis (GM) for 4 subphases of the gait cycle (first double support phase (DS1), the single support phase (SS), the second double support phase (DS2) and the swing phase (SW)) and compared these between 24 hemiparetic stroke patients and 14 healthy controls. In the upper leg, durations of BF and RF activity during SS were significantly longer on the paretic side (70% for BF, and 78% for RF) as well as on the nonparetic side (71% for BF, and 81% for RF), when compared to controls (45% and 53% for BF and RF, respectively). As a result, the duration of BF-RF coactivity during SS was longer in both legs of patients with stroke (61% in the paretic and 62% in the nonparetic leg) relative to control values (25%). In addition, during DS1 of the paretic leg, the total amount of BF-RF coactivity was abnormally long (82% vs 57% in controls). In the lower leg, longer total durations of GM activity were found during DS1 on the paretic side in people with stroke (51%) than in controls (38%). In the paretic TA, longer durations of activity were observed during SW (73% vs 60% in controls), whereas smaller total durations of activity were found during SS (28% vs 48% in controls). No statistically significant differences were found between the paretic and nonparetic leg within patients, except for the mean total duration of TA activity during DS1 (50% and 69% for the paretic and nonparetic leg, respectively). Overall, these results suggest that, despite large interindividual differences, some common disturbances can be observed in the temporal layout of muscle activity and coactivity associated with hemiparetic gait. Although these disturbances are more pronounced in the paretic leg, muscle activation patterns of the nonparetic leg also display some clear abnormalities.
Introduction
In healthy as well as in pathological human gait, the following 5 major locomotor subtasks need to be accomplished (Winter, 1987): absorption of mechanical energy during the early stance phase, the provision of body support, the maintenance of postural balance, the realization of foot clearance during the swing phase, and the generation of propulsion (either by the stance limb or by other mechanisms) to effect forward progression. For each instant in the gait cycle, the muscle activity involved in the production of gait needs to reconcile these time dependent task demands with the relevant (bio-)mechanical constraints (resulting from e.g. joint configurations and external moments). As a consequence, in healthy gait, the temporal characteristics of gait related muscle activity follows rather stereotyped patterns. Following cerebral stroke, the temporal ordering of muscle activity during walking is often disrupted, either through impairments in the central control of the timing of muscle activity, or through the development of compensatory neuromuscular strategies.

Over the past five decades, a number of studies have been conducted to assess the patterning of lower extremity muscle activity during hemiparetic gait. A common conclusion has been that there is considerable intersubject variability, and that abnormalities in muscle patterning only apply to subgroups of patients, and not to the hemiparetic population as a whole (cf. Knutsson and Richards, 1979; Shiavi et. al, 1987). Among the more common timing abnormalities found in hemiparetic gait are the absence or reduced amplitude of specific components of the activation pattern (e.g. the burst around the transition from swing to stance in Tibialis anterior; (Perry, 1993; Burridge et. al, 2001), the prolongation of existing bursts of muscle activity during the stance phase (most notably in the muscles of the upper leg Hirschberg and Nathanson, 1952; Shiavi et. al, 1987), and the premature activity of the calf muscles during the early stance phase (Perry et. al, 1978; Knutsson and Richards, 1979). A common problem regarding the interpretations of the results from these studies, is that it is often difficult to assess the exact criteria that have been used to assign such predicates as 'premature' or 'prolonged' to electromyographic patterns. The reason for this is that the detection of abnormalities in EMG patterns has usually been based on visual inspection of ensemble averaged EMG profiles, using undefined criteria (but see Peat et. al, 1976; Lamontagne et. al, 2000). However, the growing interest in the use of clinical gait analysis for diagnostic and evaluative purposes requires the availability of objective and quantitative data on abnormalities in the muscle activation patterns of patients.
with hemiparesis. Furthermore, because of the large intersubject variability with regard to these abnormalities, it is necessary to have information on the relative frequencies of these abnormalities in the hemiparetic population.

Although the assessment of hemiparetic gait has usually been restricted to the activity of individual muscles, a more elaborate picture of neuromuscular coordination may be obtained if the relative temporal coordination of agonist-antagonist muscle pairs would also be addressed. With regard to hemiparetic stroke, coactivation is of particular interest, since it has been suggested that the synergistic, mass activity of flexors (during the swing phase) and extensors (during the stance phase) represents a key characteristic of muscle control in this population (Brunnstrom, 1966; Perry, 1993). Furthermore, increased durations of coactivity between agonist/antagonist pairs may represent an important coordinative strategy to adapt to primary impairments in muscle force output (Lamontagne, 2000).

Based on objective statistical criteria, the present study tries to identify common abnormalities in the temporal layout of lower extremity muscle activity and coactivity in hemiparetic gait. The analytical approach used involves averaging the duration of activity over individual steps, for 4 distinct phases in the gait cycle (first and second double support phases, single support phase, and swing phase). This way, we assessed the patterns of activity and coactivity of both the paretic and the nonparetic lower extremity in patients with hemiparetic stroke. This strategy was chosen because hemiplegic gait is often associated with abnormalities in the temporal layout of the gait cycle (von Schroeder et al., 1995; Roth et al., 1997). As a consequence, (group) comparisons of EMG timing characteristics in terms of percentage gait cycle time are difficult to make (e.g. muscle activity at 70 % of gait cycle time may correspond to swing activity in healthy subjects or to stance activity in the non paretic leg of a hemiplegic walker). In this study, we tried to avoid this potential problem by calculating the mean duration of activity for four subphases of the gait cycle. In case temporal abnormalities were identified on the group level, an additional analysis of individual patient data was performed to obtain the relative frequency of this abnormality. In order to quantify the amount of time that a muscle was active during different phases of the gait cycle, EMG signals were dichotomised into periods of muscle activity and muscle inactivity. Quite commonly, detection of muscle activity is achieved by setting an a priori threshold, either in terms of the absolute amplitude of the signal or in terms of the variance of the signal (see Hodges, 1996 for a review). However, the choice of these threshold levels is rather arbitrary, and should be tuned according to the properties of the
analysed signal (e.g. to the signal to noise ratio) for optimal results. Therefore, in the present study, we employed an analytical strategy that does not depend on a priori threshold settings, using an application of k-means clustering of the rectified and low pass filtered signal.

Table 1: Patient characteristics

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Methods

Subjects

Twenty-four patients with stroke (14 female) and 14 healthy control subjects (8 female) participated in this study. The mean age in the patient group was 58.58 years (sd = 13.17). The mean time post stroke was 8.75 months (sd=6.16), although this varied considerably among patient (range 3 – 21 months). Fourteen patients suffered from right sided hemiparesis and 10 patients from left sided hemiparesis. The mean age of the control group was 42.85 years (sd=12.3).

The inclusion criteria for patients were: (1)- a first-ever supratentorial stroke (due to either hemorrhage or infarction), (2)- admission to the rehabilitation center for restoring independency of gait, and (3)- have a Functional Ambulation Categories score of at least 2 (‘Patient needs continuous or intermittent support of one person to help with balance or coordination’) and at the most 4 (‘Patient can walk independently on level ground, but requires help on stairs, slopes or uneven surfaces’). Exclusion criteria were: medical conditions that are known to affect walking performance, other than stroke; severe forms of aphasia or cognitive problems that would hinder comprehension or cooperation; severe emotional or behavioral problems, and severe visuospatial neglect, as indicated, by abnormal scores on two or more of the following tests: the line bisection test (Schenkenberg et. al, 1980), the letter cancellation task (Diller et. al, 1974), the Bells test (Gauthier et. al, 1989), and the clock drawing test (Wilson et. al, 1987). None of the control subject reported to have a history of neurological or orthopaedic problems that could have affected the ability to walk.

Prior to the study, all patients underwent a clinical examination by an experienced physician, in which problems in trunk control, the degree of lower extremity motor selectivity (i.e. the Brunnstrom motor stage), and impairments in sensibility were scored (cf. de Haart et. al, 2004). Table 1 provides a summary of the patient characteristics. All participants gave their written informed consent. The study was approved by the regional medical-ethical committee Arnhem-Nijmegen.

Setup and protocol

All subjects walked on a motor driven treadmill (walking surface 200 by 70 cm) for 40 seconds. The minimum number of strides that had to be collected during this period was set to 10. Patients walked at a self-selected speed. The mean walking speed in the patient group was 0.35 ms\(^{-1}\) (sd=0.21), although this varied considerably among patients (range 0.11 to 1.06 ms\(^{-1}\)). Patients were allowed to hold
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on to a rail in front of the treadmill to maintain their balance. The height of the rail was adjusted for each patient, so that it could be used for subtle balance corrections only, and not for body unloading. Although all patients wore a harness for safety, no body weight support was provided. Patients were not allowed to wear an orthosis. Neither were they allowed to receive manual assistance from a therapist. For half of the control subjects, the treadmill speed was set to 0.56 ms\(^{-1}\), whereas for the other half treadmill speed was set to 0.28 ms\(^{-1}\). In the analysis, these 14 control subjects were considered as one group with a mean gait speed of 0.42 ms\(^{-1}\).

Data recording
Disposable surface electrodes (MediTrace ECG 1801 Pellet, (Ag/AgCl)) with a diameter of 10-12 mm and a minimum inter-electrode distance of 24 mm (Graphics Controls, Buffalo NY, USA) were used to record activity from Biceps femoris, Rectus femoris, Tibialis anterior, and Gastrocnemius medialis, from both legs. Electrodes were placed according to SENIAM conventions (Freriks et. al, 1999). Incoming signals were fed to a K-lab SPA 20/8 pre-amplifier (common mode rejection ratio > 95 db; noise level of < 1 µV rms), and subsequently high pass filtered using a third order Butterworth filter (-3db point at 20 Hz), and low pass filtered using a second order Butterworth filter (-3db point at 500 Hz). Next, the EMG data were digitised at 2400 Hz., and stored on computer hard disk. A light reflective marker was attached to the heel of both feet, and recorded by 5 infrared cameras (PRIMAS \textsuperscript{TM}). These kinematic data were sampled at 100 Hz, stored on computer hard disk, and used for detection of the stance and swing phase of the gait cycle (see below).

Detection of muscle activity
This detection process involves several steps, and is illustrated in figure 1. First, the electromyographic data were high pass filtered at 10 Hz to attenuate the effects of movement artefacts. Next, the signals were full wave rectified and low pass filtered at 25 Hz (figures 1a and 1b). Subsequently, the linear envelope of the EMG signal was transformed into a set of discrete values using a k-means clustering algorithm (figure 1c). The goal of k-means clustering is to find similarities between data points and to group these data points according to their similarities. This is accomplished by assigning all individual data points to one of ‘k’ clusters, such that some metric (e.g. the squared Euclidean distance) relative to the centroids of the clusters is minimized. Eventually, all data points are grouped into one of ‘k’ disjoint clusters.
Figure 1. Example of the detection of muscle activity in EMG signals. The top left panel shows a raw EMG signal. The top right panel shows the same signal after rectification and low pass filtering at 25Hz. The bottom left panel shows the same signal after transformation into discrete values using k-means clustering (number of clusters = 5) of the rectified and filtered data. Muscle activity and muscle inactivity were separated by assuming that data points in the cluster with the lowest mean value corresponded with episodes of muscle inactivity and data points belonging to other clusters to muscle activity. The bottom right panel shows the original raw EMG signal with the detected periods of activity indicated in grey shaded areas.

(Mac Queen, 1967). Note that the number of clusters is set a priori, but that the rest of the grouping process is totally unsupervised.

In the rectified and filtered EMG signal, periods of muscle inactivity are represented by data with a relatively stationary (i.e. not time varying) mean. For dichotomisation of the signal into periods of muscle activity and inactivity, we can capitalise on this property by using the k-means clustering algorithm. Because data points that correspond to muscle inactivity form a relatively homogeneous dataset, these data will be classified as belonging to one separate cluster by the k-means clustering algorithm. By definition, this will be the cluster with the lowest mean value. Data that belong to this cluster are assumed to correspond with periods of muscle activity and, consequently, data that are assigned to other clusters (i.e.
clusters with higher mean values) are classified as representing periods of muscle inactivity. Figure 1d shows an example of the eventual classification result superimposed on the original EMG signal. Note that even short and transient epochs of muscle activity are detected in the signal, and that the presently used method does not make any a priori assumptions about the number of bursts that should be present in the gait cycle for a particular muscle. This property may be particularly convenient for the study of timing characteristics in abnormal populations when the number of bursts to be detected are difficult to specify in advance (e.g. when specific bursts are missing or when multiple bursts are present within one gait cycle). Because of this property, and the detection process is completely unsupervised, it may be particularly useful for applications in clinical gait analysis.' The adequateness of the present procedure depends to some extent on the cut-off frequency of the low pass filter used (10 Hz in this case), and on the number of clusters ‘k’ that is set a priori. Further note that in case of a homogeneously distributed data set (i.e. a muscle displays no activity or continuous activity), this algorithm renders meaningless results. Visual inspection of the raw data showed that no such signals were present in the data set used for this study. For all data analysed in this study, the number of clusters was set to 5.

**Data analysis**

Heel strike and toe off for each leg were determined using speed distribution analysis of the heel marker data (Peham et. al, 1999). This information was used to determine the swing phase (SW), the first double support phase (DS1), the second double support phase (DS2), and the single support phase (SS), for each leg (for an explanation of these gait phases, see figure 2a). For each of these subphases of the gait cycle, the duration was determined as a percentage of the total gait cycle time. For all of the four subphases, the percentage of time for which a muscle was active was determined for each step, and subsequently averaged over all steps, for each subject. Similarly, the average percentage of time that a muscle pair was active simultaneously was calculated within each leg, for BF and RF, and for GM and TA, to obtain an estimate of the duration of coactivity of these muscle pairs. All EMG signals and marker data were analysed using custom Matlab™ software.

**Statistical analysis**

Differences between patients and controls (the between factor GROUP) in the relative duration of the DS1 and the SS phase (% of gait cycle), and in the mean
total durations of muscle (co-) activity for each of the 4 gait phases, were tested by means of a Mann-Whitney U test. Within the patient group, differences between the paretic and nonparetic leg (within factor ‘SIDE’) with regard to these variables were assessed using a Wilcoxon matched pairs signed ranks test. For each of these tests, Bonferroni corrections were made to maintain the familywise alpha level at .05.

In case of a statistically significant difference in EMG values for the patient and the control group, two additional analyses were performed. First, an analysis was performed to compare two subgroup of patients that were classified according to the severity of their paresis. Patients with lower extremity Brunnstrom scores \( \leq 4 \) (i.e. at most ‘increased muscle tone with alternating gross movements in extension and flexion synergies’) were considered ‘more severely affected’, whereas patients with Brunnstrom scores >4 (i.e. at least ‘muscle tone normalization with some degree of selective muscle control’) were considered ‘less severely affected’.

Secondly, the number of patients were counted that had mean individual values above or below the mean of the control group +/- 2 sd’s. For these selected patients, the ensemble averaged EMG patterns were calculated. For this purpose, the EMG signals were time normalized with respect to the stance and swing phase separately, to ensure adequate temporal alignment between individual time series. Next, the averaged patterns of each subject were amplitude normalized with respect to the peak value of each pattern to allow comparisons between EMG patterns despite interindividual differences in the amplitude of muscle activity.

Results

The mean number of strides that could be obtained from patients was 19.54, but varied considerably among subjects (sd=6.64; range 10 to 33 strides). Within the control group, the mean number of strides recorded was 25.71 (sd=10; range 10 to 48 strides).

A. Duration of subphases of the gait cycle

Comparisons of the duration of the DS1 (= contralateral DS2 phase) and the SS (= contralateral SW phase) subphases of the stride cycle were made between patients with stroke and control subjects. The mean duration of DS1 in the control group (14% of gait phase duration, sd=5; p<.05) was significantly shorter than in the paretic leg (19% of stance phase duration, sd=7; p<.05) but not for the nonparetic leg of patients (18%, sd=5; p=.083). With regard to the SS phase, the mean
Figure 2. A. Explanation of the gait phase classification used in this study. B. Mean (+ sd) relative duration of the DS1 and SS phases of the gait cycle for the paretic leg (black), the nonparetic leg (grey) and for controls (white). Note that the DS2 and SW phases are not shown because they are equivalent to the contralateral DS1 and SS phase respectively.

duration for the paretic leg (31%; sd=10) as well as for the nonparetic leg (32%; sd=9) was significantly shorter than the duration found in the control group (37%; sd=7; p<.05). Also, the relatively small difference between SS duration of the paretic and nonparetic leg (mean difference nonparetic leg-paretic leg = 1.27%; sd=3.82), corresponded with a statistically significant result for the factor SIDE (p<.05) for this phase of the gait cycle.

B. Temporal patterning of muscle activity over the four subphases of the gait cycle.
The average durations of muscle activity for all 4 gait phases and the four muscles of both legs are depicted in Figure 3.
Gait control after stroke

**Biceps Femoris (BF).**

Increased durations of BF activity during the SS phase were found, in the paretic (70%, sd=20; p<.05) as well as in the nonparetic leg (71%, sd=20; p<.05) when compared to controls (45%, sd=20). On the paretic side, 15 patients showed increased durations of BF activity during the SS phase (see panel A of figure 4), whereas an equal number of patients increased durations on the nonparetic side (see panel B of figure 4).

**Rectus Femoris (RF).**

Increased durations of BF activity during the SS phase were found, on both the paretic side (78%, sd=29; p<.05) and the nonparetic side (81%, sd=24; p<.05) compared to the control group (53%, sd=35). On the paretic side, 14 patients showed increased durations of BF activity during the SS phase (see panel C of figure 4), whereas 16 patients increased durations on the nonparetic side (see panel D of figure 4).

![Figure 3](image)

*Figure 3.* Mean (+ sd) duration of muscle activity for Biceps femoris (top left panel), Rectus femoris (top right panel), Tibialis anterior (bottom left panel), and Gastrocnemius medialis (bottom right panel), for each of the four subphases of the gait cycle (ds1= first double support phase; ss= single support phase; ds2= second double support phase; sw= swing phase).
During the SS phase, the mean relative duration of TA activity was shorter in the paretic leg of patients (28, sd=28) than it was in controls (48, sd=29; p<.05). The ensemble averaged pattern of activity of the 12 patients that scored below the mean -2sd’s value of the control group are depicted in panel E of figure 4. In contrast, the mean relative duration of TA activity on the paretic side during the SW phase was longer for patients (73, sd=17) than it was in the control group (60, sd=31; p<.05). This abnormality was apparent in 13 patients (see panel F of figure 4). Also, within the stroke group, the mean percentage of TA activity during DS1 was significantly

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**Figure 4**: Ensemble averaged EMG patterns for subgroup of patients with specific abnormalities. Panel A: longer mean total duration of SS activity in the paretic BF (n=15); Panel B: longer mean total duration of SS activity in the nonparetic BF (n=15); Panel C: longer mean total duration of SS activity in the paretic RF (n=14); Panel D: longer mean total duration of SS activity in the nonparetic RF (n=16); Panel E: shorter mean total duration of SS activity in the paretic TA (n=12); Panel F: longer mean total duration of SW activity in the paretic TA (n=13); Panel G: longer mean total duration of DS1 activity in the paretic GM (n=10); Ensemble average EMG patterns for the control group are shown in the left column.
higher on the nonparetic (mean 69%, sd=23) than on the paretic side (mean 50%, sd=31). Further analysis showed that the smaller percentages of TA activity during the DS1 phase of the paretic leg were largely due to decreased durations of activity in the more severely affected subgroup (28% (sd=22) vs. 63% (sd=29) in the less severely impaired subgroup (p<.05)).

**Gastrocnemius Medialis (GM).**
The mean relative duration of GM activity during DS1 for the patients’ paretic leg (51, sd=26) was significantly longer than in the control group (36, sd=26; p<.05). This abnormality was apparent in 10 of the 24 patients (panel G of figure 4) Further analysis showed that the percentage DS1 activity in the paretic GM was significantly larger in more severely impaired patients (66%, sd=15) when compared to the rest of the patient group (42%, sd=27; p<.05).

**C. Coactivation between muscle pairs over the four subphases of the gait cycle**
Figure 5 depicts the mean coactivation level for BF-RF and TA-GM.

**BF-RF coactivation.**
Because the mean total duration of BF and RF activity during the SS phase were abnormally long, the amount of BF-RF coactivity exceeded control levels (25, sd=21) for both the paretic (61, sd=31; p<.05) and the nonparetic leg (62, sd=31; p<.05). In the paretic leg, abnormally long durations of BF-RF coactivity was found for 18 patients, whereas abnormally long coactivity durations in the nonparetic leg were observed in 15 patients. In addition, during DS1, the relative duration of BF-RF coactivity in the paretic leg (82, sd=17) was longer than in controls (57, sd=33; p<.05), with 13 patients showing above normal durations of coactivity for this muscle pair. Although the same tendency was observed for the nonparetic leg during DS1, this effect did not reach statistical significance (p=.068).

**TA-GM coactivation.**
No statistically significant differences in TA-GM coactivation were found for any of the gait phases, either between patients and controls, or between the paretic and the nonparetic leg.
Figure 5. Mean (+ sd) relative time two muscles in a muscle pair are simultaneously active for each of the four subphases of the gait cycle (ds1 = first double support phase; ss = single support phase; ds2 = second double support phase; sw = swing phase). Shown are the duration of coactivation between Biceps femoris and Rectus femoris (top left panel), Biceps femoris and Tibialis anterior (top right panel), Biceps femoris and Gastrocnemius medialis (left middle panel), Rectus femoris and Tibialis anterior (right middle panel), Rectus femoris and Gastrocnemius medialis (bottom left panel) and Tibialis anterior and Gastrocnemius medialis (bottom right panel).

Discussion
Based on statistical criteria, we identified abnormalities in the temporal patterning of lower extremity muscle activity and coactivity in hemiparetic gait. The results suggest that the typical temporal lay-out of gait related muscle activity that is observed in healthy persons is disturbed in the majority of hemiparetic stroke patients. Despite large interindividual differences, we were able to find some systematic differences in the temporal patterns of muscle activity and coactivity.
between hemiparetics and controls. Although disturbances are more pronounced in the paretic leg, muscle activation patterns of the nonparetic leg also display some clear abnormalities.

**Duration of subphases of the gait cycle**
The temporal lay-out of the step cycle was significantly altered in stroke patients when compared to controls. In particular, stroke patients spent significantly less time in single support, and more time in the double support phases. Although the difference in DS1 duration of the nonparetic leg form control values did not reach statistical significance, the same difference in SS duration did. These results confirm findings by others showing that the time spent in phases when full body weight has to be supported by only one leg is reduced in hemiparetic gait (Shiavi et al, 1987; Lehman et al. 1987; Olney et. al, 1991). These temporal abnormalities might be related to the diminished propulsive power in the paretic leg, leading to reductions in swing phase duration on the nonparetic side, or to an adaptive mechanism that is used to secure dynamic balance and body support by prolonging the time spent with both legs on the ground (Olney and Richards, 1996). The employment of such a strategy may also be influenced by psychological factors, e.g. self confidence. Although statistically significant differences were found for the duration of the SS phase between the paretic and nonparetic leg, the magnitude of these differences was small: The single support phase was only 1.3% of stance phase duration longer on the nonparetic side. This relatively small difference may be partly related to the use of a treadmill in the present study. As was shown by Harris-Love and co-workers (2001), the durations of gait cycle subphases (e.g. single support duration, stance-swing ratios) can become more symmetric in stroke patients when they walk on a treadmill compared to overground walking.

**Upper leg muscles**
A prominent feature of the temporal patterning of leg muscle activity in patients with hemiparesis was the prolonged activity of BF and RF during the SS phase of the paretic as well as the nonparetic leg. As a result, the duration of BF-RF coactivity during the single support phase of both legs was also significantly prolonged. In addition, longer durations of BF-RF coactivity were found during the DS1 phase of the paretic leg in more than half of the patients. The present results confirm those from earlier studies (Hirschberg and Nathanson, 1952; Peat et. al, 1976; Knutsson and Richards, 1979; Shiavi et. al, 1987) suggesting that the
prolonged activity of quadriceps and hamstring muscles during the stance phase represents a common feature of neuromuscular coordination in hemiparetic gait. Analysis of the individual patient data further showed that this abnormality was present in the majority of patients, in the paretic as well as in the nonparetic leg. The latter finding confirms observations made by others indicating that aberrant muscular control in the upper leg of patients with hemiparesis is a bilateral phenomenon (Shiavi et. al, 1987; Wortis et. al, 1951).

Because the periods of prolonged SS activity of the upper leg muscles coincided with the normal activity periods of ankle plantar flexors, one may consider the abnormal upper leg activity during the SS phase as part of a more general extensor synergy during this phase of the gait cycle. Mass extensor activity in post stroke hemiparesis has been attributed to impairments in the central control of walking resulting in the development of primitive synergistic extensor activity of hip, knee and ankle muscles during the stance phase (Brunnstrom, 1966; Perry, 1993). Although the present results do not justify definite statements on the origins of this phenomenon, there are some indications that the mass extensor activity that was found in this group of patients is not necessarily related to impairments in the pattern generating mechanisms (resulting in a less selective recruitment of leg muscles), but rather reflects an adaptive locomotor strategy. First, the amount of BF-RF coactivity during stance that we found was not related to a clinical measure of the level of muscle selectivity (i.e. the Brunnstrom stage of recovery), suggesting that impairments in the ability to selectively recruit muscles during isolated movements cannot explain the patterns of mass extensor activity found during gait. Furthermore, the patterns of increased durations of quadriceps-hamstrings coactivation during stance is not unique for stroke and has been found e.g. in spinal cord injury (Leroux et. al, 1999), and diabetic neuropathy (Kwon et. al, 2003), as well as in infant stepping (Okamoto et. al, 2003). These findings suggest that the prolonged stance activity of upper leg muscles could be related to a more general neuromuscular strategy that may serve to supply additional support, e.g. to the knee. This may be related to insufficient calf muscle strength since these muscles provide the largest contribution to body support during the SS phase (Kepple et. al, 1997) Possibly, impaired muscle strength on the nonparetic side may explain why similar patterns of adaptive coactivity of extensor muscles were present in the nonparetic leg as well. As was shown by Adams and coworkers (Adams et. al, 1990) the reduction in strength in the nonparetic leg is most notable in the distal muscles, e.g. the ankle plantarflexors. The increased duration of BF- RF coactivity may
alternatively be explained as a strategy to hold the head-arm-trunk segment against gravitational forces in case of forward postural lean (Olney and Richards, 1996), or to provide knee stability in order to prevent knee hyperextension or knee collapse.

**Lower leg muscles**

In GM, significantly longer durations of GM activity were found during the DS1 phase, suggesting that the onset of GM activity occurred earlier in the gait cycle. Overactivity of the calf muscles during the early stance phase appears to be a fairly common abnormality following stroke (e.g. Knutsson and Richards, 1979; Perry, 1978), a phenomenon that has been ascribed to the lack of cortically mediated inhibition of stretch reflexes and the presence of a spastic dropfoot during swing (Yelnik et. al, 1999; Burridge et. al, 2001). However, similar patterns of premature calf muscle activity can be observed during toe walking in healthy subjects (Perry et. al, 2003) which raises the question whether early activation of the calf muscles during stance is a direct sign of spasticity in these muscles, or whether it is indirectly related to spasticity and represents an adaptive strategy to control the ankle when the foot makes initial contact in a plantarflexed position.

In the paretic leg of patients, the mean total duration of TA activity during the SS phase was shorter than it was in controls. Since TA activity during the SS phase may be involved in the control of postural balance during walking (Louwerens et. al, 1995), this specific finding may be reflective of impaired balance control in hemiparetic walkers. A second abnormality in the paretic TA activity of patients was the longer total duration of activity that was found during the SW phase. This may be due to the inability to rapidly activate and deactivate this muscle during this phase of the gait cycle. In order to compensate for decreased force output of this muscle, patients may prefer a pattern of continuous activation in order to overcome problems in the selective recruitment and de-recruitment of this muscle. As an alternative, the larger durations of paretic TA activity during swing could be interpreted as an effort to overcome mechanically related resistance of the ankle plantarflexors during this phase of the gait cycle (Dietz et. al, 1981).

The group differences that were found with regard to the temporal structure of TA and GM activity did not result in statistically significant differences in the duration of TA-GM coactivity between controls and patients or between the paretic and the nonparetic leg. Previously, Lamontagne and coworkers (Lamontagne et. al, 2000) found abnormally high levels of TA-GM coactivity during the DS1 and DS2 phase of the nonparetic leg, whereas decreased levels of coactivity for this muscle
pair were found during the SS phase of the paretic leg. A potential explanation for these seemingly conflicting findings may be that the present study was performed on a treadmill, whereas the Lamontagne et al study (2000) was conducted on a walkway. In a study comparing treadmill walking and overground walking in hemiparetic walkers, Hesse et. al (1999) found reductions in the levels of TA-GM coactivation, which may partly explain the absence in the present study of any significant group differences with respect to this measure.

Limitations
In this paper, we described the temporal structure of gait related muscle activity in hemiparetic stroke patients and healthy controls. However, due to the detection method employed and the description in terms of subphases of the gait cycle, a comparison with previous studies on temporal patterning in healthy and hemiparetic gait is not straightforward. Whereas the majority of detection strategies that have been described in the literature involve detection of discrete bursts of activity by means of threshold methods, the presently employed clustering method is based on identification of periods of muscle in-activity, allowing the detection of episodes of muscle activation with a very low amplitude. As a result, this method will likely lead to a more liberal detection of activity which may explain why the detected patterns in healthy controls are somewhat different from what has been reported in the literature. In particular, the relatively long total durations of BF (45% of total SS phase duration), RF (53%), and TA (48%) activity during the SS phase do not appear to be in agreement with what has been reported by e.g., Perry (1992) who found cessation of activity at 5 %, 15%, and 10% of gait cycle time for BF, RF, and TA, respectively. The relatively longer duration of SS activity for BF, RF and TA that were found in the present study for healthy subjects may represent subtle neuromuscular adaptations to the very low gait speeds employed that, due to their very low amplitude, may not have been captured by threshold based detection methods. Note that the presently adopted detection method makes no a priori assumptions about the level of muscle activity that should be considered as 'meaningful', which may result in a more liberal detection of activity compared to threshold based detection methods. Depending on its application, this may or may not be a desirable feature.

Another point that needs to be mentioned is that the gait speeds employed by patients (mean=0.35 ms\(^{-1}\)) and controls (mean=0.42 ms\(^{-1}\)) were not identical. However, it is not very likely that this relatively small difference in gait speed
between the two groups has confounded our results. Other studies have shown that substantial differences (> 0.56 ms\(^{-1}\)) in gait speed are required to induce differences in the timing of muscle activity (e.g. Hof et. al, 2002; den Otter et al., 2004).

A final point that needs to be mentioned is that the present study addressed the temporal structure of muscle activity during treadmill walking. This is especially true since the temporal layout of gait subphases may be different between overground walking and treadmill walking (Hesse et. al, 1999). Although this difference should be taken into account, electromyographic studies comparing treadmill and overground walking have failed to show clear differences in terms of muscular timing (Murray et. al, 1985; Arsenault et. al, 1986).

**Conclusion**

To summarize, the present data show that it is possible to detect some common timing abnormalities in the lower extremity muscle activity of post stroke hemiparetic walkers, despite large variations between individual patients. Although some of these abnormalities may be related to primary impairments in the temporal control of muscle activity (e.g. the premature activity of GM) others are likely to reflect compensatory activity that is used to optimize the gait pattern in the presence of muscular weakness and reduced coordinative control (e.g. the increased durations of BF-RF coactivity during the SS phase). Within patient comparisons between the paretic and the nonparetic leg did not show systematic differences, with the exception of the shorter mean duration of paretic TA activity and the tendency towards a longer paretic GM activity during the DS1 phase of the paretic leg. Future research will need to study the patterning of muscle activity in more complex gait tasks to reveal the functional implications of aberrant muscular timing patterns in hemiparetic gait.

**References**

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