Gait control after stroke

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Chapter 2

Speed related changes in muscle activity from normal to very slow walking speeds

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Abstract

The study of neuromuscular activity at very slow walking speeds may lead to a better understanding of the mechanisms underlying speed regulation during walking, and may aid the interpretation of gait data in slowly walking patient groups. Extreme reductions in walking speed will cause changes in locomotor task demands that may eventually result in modifications of the patterning of muscle activity that underlies walking. The aim of the present study was to investigate patterns of lower limb muscle activity during very slow walking (< 0.28 ms^{-1}), and study the neuromuscular gain functions that reflect the phase dependent effects of walking speed on EMG amplitude. Nine healthy young adults walked at 7 different walking speeds (1.39, 0.83, 0.28, 0.22, 0.17**,** 0.11, and 0.06 ms^{-1}) while EMG was recorded from 8 lower extremity muscles. Results showed that the phasing of muscle activity remained relatively stable over walking speeds despite substantial changes in its amplitude. However, between 1.39 and 0.28 ms^{-1}, epochs of Rectus femoris, Biceps femoris and Tibialis anterior activity were found that were typical for specific speed ranges. When walking speed was further decreased to almost standing still (0.06 ms^{-1}), negative gain values were found in Peroneus longus during midstance and Rectus femoris in late swing, indicating the emergence of new bursts of activity with decreasing walking speed. It is proposed that these extra activities may be attributed to increased demands on postural stability, and the altered dynamics of the swinging limb at very slow speeds.

Introduction

During walking, the ability to adjust the speed of progression is an important mechanism that adapts locomotor activity to changes in environmental demands (e.g. to accommodate time pressure or to enhance safety). In general, changes in walking speed require the adjustment of a neuromuscular gain factor, whereas the global timing characteristics of the muscle activity patterns are essentially preserved (Milner et. al, 1971; Murray et. Al, 1984; Nilsson et. al, 1985; Shiavi et. al 1987 ; Yang and Winter,1985; Hof et. Al, 2002). This strategy for speed regulation, through which a phase specific gain factor modulates relatively invariant basis patterns, simplifies control, and enhances computational efficiency.

The main role of the muscles in the regulation of walking speed is to control the accelerating and decelerating forces of individual body segments in order to establish safe forward progression (Yang and Winter,1985). As a result, the amplitude of muscle activity increases with walking speed because of the need for larger muscular force output. This normal positive relationship may be challenged if walking speed is strongly reduced, due to changes in the underlying locomotor task demands. Extreme reductions in walking speed will substantially prolong the time spent in double support, and one may expect a switch from locomotor to merely postural muscular synergies. Also, the larger horizontal excursions of the centre of mass associated with slow walking may necessitate more explicit muscular efforts to maintain frontal plane balance during walking (cf. Bauby and Kuo, 2000). This presumed relationship between speed reduction and dynamic instability is further substantiated by recent evidence showing that a decrease in walking speed may cause an increase in potentially destabilising vestibulospinal drive (Brandt et. al, 1999), probably due to diminished proprioceptive input (Dietz et. al, 2001; Horak and Hlavacka, 2001). Strong reductions in walking speed may also affect the neuromuscular control of the swinging limb. While at comfortable and fast speeds, the swinging leg describes a largely ballistic trajectory and moves under passive, gravitational control (Mac Mahon, 1984), strongly reduced walking speeds may necessitate a more active mode of control to counteract gravity and guarantee sufficient 'air time' for the swinging leg. .

Alterations in swing phase control, and the increased emphasis on dynamic balance, may induce modifications in the patterning of lower limb muscle activity that are uniquely related to these very low speed ranges. Within normal ranges of walking speed (0.75 – 1.75 ms^{-1}), speed effects on electromyographic patterns can be described quite accurately by superimposing a few speed related gain functions

on a small number of speed independent basis patterns (Hof et. al, 2002). It follows that speed induced changes in the timing of muscle activity are established primarily through modulation by muscle specific gain functions of a relatively invariant locomotor rhythm (cf. Duysens and van de Crommert, 1998). Given the associated changes in locomotor task demands described above, one may wonder how these same principles are extrapolated to the lowest speed ranges.

The primary aim of this study was to investigate changes in speed related neuromuscular gain, when walking speed was systematically varied from normal speeds to almost standing still. While the lower boundaries of the walking speed continuum are not usually encountered during normal human walking, they still may provide information on the strategies used by the neuromuscular system to induce speed modifications. The present study does not provide a database for systematic comparison of patient data with standardised values, yet the results from the present study may still be of clinical interest. Since reduced walking speed is a key feature of pathological gait (Van Emmerik and Wagenaar, 1996), clinical gait analysis as well as gait research on pathological populations may profit from the assessment of neuromuscular patterns associated with very slow walking. More specifically, studies on very slow walking may help to dissociate between those aberrations that are directly related to pathological changes in neuromuscular coordination, and those that reflect adaptations to speed related changes in locomotor task demands.

Methods

Subjects

Nine healthy subjects volunteered in this study (5 females, 4 males; mean (SD) age 22.4 (2.35) years; height 1.81 (6.7) m; body mass 66.6 (7.84) kg). None of the subjects suffered from any neurological or orthopaedic disorder that may interfere with the goals of the study.

Procedure

Subjects walked on a motor driven treadmill (2.0 * 0.7 m) at seven different walking speeds (1.39, 0.83, 0.28, 0.22, 0.17, 0.11, 0.06 ms^{-1}). Two 40 second registrations were completed at each walking speed in two quasi-randomised series of 7 trials. The order of each series was the same for all subjects. In order to get accustomed to walking on the treadmill, subjects were allowed to walk on the treadmill at a self-

selected speed. During the experiment, no particular instructions were given with respect to stride length or cadence.

Data recording

Electromyographic (EMG) recordings were made using 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). The electrodes were placed on the skin to measure activity from 8 lower extremity muscles: M. Biceps Femoris (BF), M. Semitendinosus (ST), M. Rectus Femoris (RF), M. Vastus Medialis (VM), M. Gastrocnemius Medialis (MG), M. Soleus (SO), M. Peroneus Longus (PL) and M. Tibialis Anterior (TA). Electrodes were placed according to SENIAM conventions (Freriks et. al, 1999). Simultaneously, footswitch data of the heel and toe were collected using custom made footswitches. Signals were fed into a K-lab SPA 20/8 pre-amplifier with a common mode rejection ratio > 95 db and a noise level of < 1 μV rms. Incoming signals were 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). The incoming electromyographic signals were monitored on-line, to ensure sufficient quality of the data. In case of obviously distorted signals, e.g. due to movement artefacts or poor skin electrode contact, the necessary measures were taken (renewal of electrodes; skin abrasion etc.).The EMG signals as well as the footswitch data were digitised at 2400 Hz and stored on computer hard disk for offline processing.

Data processing

Footswitch data were used to determine initial contact (IC) and toe off (TO) of each recorded step. EMG signals were full-wave rectified and low-pass filtered at 25 Hz using custom Matlab® software. After exclusion of steps that contained clearly distorted signals, the electromyographic data were time normalised with the gait cycle taken as 100%. Earlier studies have shown that walking speed not only affects step length and step duration, but also the relative durations of the stance and swing phase (Nilsson et . al, 1985; Shiavi et. al, 1987). Therefore, in order to justify a point to point comparison between EMG profiles found at different walking speeds, a second normalisation procedure was applied in which the stance and swing phases were normalised separately, to 150 and 100 data points respectively.

For both types of time normalised data, the individual strides were averaged for each subject to obtain an ensemble average for every subject at each walking speed. In order to obtain an accurate estimate of the individual ensemble averages, as much strides as were available for each condition were used to calculate the average profiles (mean number of strides ranged from 15.5 (sd = 8.7) at 0.06 ms^{-1}, to 73.5 (sd =4.0) at 1.39 ms^{-1}). For each muscle, these individual averages were then amplitude normalised with the peak amplitude at the fastest walking condition (1.39 ms^{-1}) set to 100%. Finally, these data were averaged over all 9 subjects, to obtain a group mean envelope for each of the 8 muscles for each of the 7 walking speeds.

Data analysis

To estimate the gain functions that describe the speed related changes in EMG amplitude over the time normalised gait cycle, the walking speeds of the individual subjects were first normalised to body height (Hof et. al, 2002) as follows:

$$\hat{v}_{ni} = \frac{v_n}{\sqrt{g l_{0i}}} \qquad [1]$$

where

$\hat{v}_{ni}$ = normalized speed of subject i for walking speed condition n

v_n = treadmill speed for walking speed condition n

g = acceleration of gravity (= 9.81 m/s^2 on earth)

l_{0i} = body height of subject i

Next, the slope of the presumed linear relationship between walking speed and EMG values was calculated, as follows:

$$b_{b,a} = \frac{\left(\sum_{i=1}^{9} \frac{x_{ijb} - x_{ija}}{9} \right)}{v_b - v_a} \quad \text{for j =1…250} \qquad [2]$$

where

$b_{b,a}$ = rate of change between walking speed conditions a and b in normalised EMG amplitude per unit change in walking speed (= ms^{-1})

x_{ijb} , x_{ija} = mean EMG value of subject i (1..9) at instant j (1..250) at speed b/a

$v_b - v_a$ = difference between walking speeds v_b and v_a (ms^{-1})

This is identical to the slope value found by regressing the amplitude normalized EMG-values on walking speed using a simple least squares criterion (see figure 1). This type of analysis was used to compare differences in EMG amplitude between the following pairs of walking speed conditions: (a) 1.39 ms^{-1} vs. 0.83 ms^{-1} , (b) 0.83 ms^{-1} vs 0.28 ms^{-1}, and (c) 0.28 ms^{-1} vs. 0.06 ms^{-1}.

Regression of the amplitude normalized EMG values (1 .. 250) on walking speed (1.39, 0.83, 0.28, 0.06 ms^{-1}) yields slope coefficients for each point in the normalized gait cycle. These coefficients quantify the linear dependency of EMG amplitude on walking speed, and thus reflect the gain applied by the neuromuscular system to establish changes in walking speed. This approach is conceptually similar to

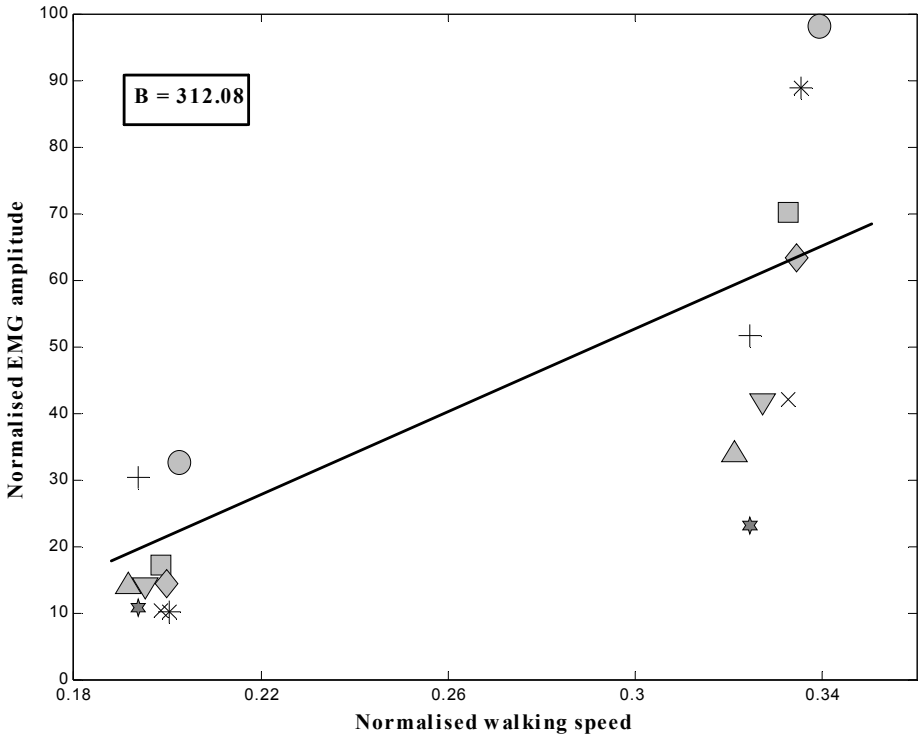

Figure 1. Example of the calculation of the gain factor 'B' for one point on the normalised gait cycle of the RF (j =150) between 2 walking speed conditions (treadmill speeds: 0.83 ms^{-1} vs. 1.39 ms^{-1}). Depicted are the amplitude normalised EMG values for all 9 subjects at both speeds, and the regression line with slope B = 312.08.

that used by Hof et al. (2002) for the modeling of speed effects on EMG (see also Ramsay and Silverman, 1997). In the present study, this approach is chosen to compare speed effects between different pairs of walking speed conditions. It is important to note that this type of analysis reflects both the magnitude of the speed effect as well as its direction: positive slope values indicate an increase in EMG

amplitude with increasing walking speed, whereas a negative value indicates an increase in EMG amplitude with decreasing walking speed (i.e. a negative linear relationship).

An important assumption underlying this type of analysis is that all points of the gait cycle at different walking speeds are appropriately aligned and share a similar time base. Because the relative durations of swing and stance phases are known to vary with walking speed (Nilsson et. al, 1985), a simple point-to-point comparison between walking speeds based on the normalized step cycle would be inappropriate. For instance, activity found in the late stance phase at lower speeds, would be compared to early swing activity at higher speeds if conventional step cycle normalization would be applied. Therefore, differences in stance – swing distribution were corrected by normalizing stance and swing phase separately to 150 and 100 point respectively.

Results

Effects of walking speed on the group ensemble average

After normalizing the step cycle to 100%, the group average profiles were calculated for every muscle at each of the 7 walking speeds. In general, the amplitude of muscle activity decreased with decreasing walking speed. When the peak amplitude of the group ensemble average was taken as 100%, peak activity at 0.06 ms^{-1} was found to be decreased to the following percentages: BF = 16.7%, ST = 17.6%, RF = 38.2%, VM = 18.0%, MG = 39.9%, SO = 24.4%, PL = 44.0%, and TA = 31.8%.

For a few instances, EMG bursts were found to be very velocity-specific. In 5 out of 9 subjects, an extra burst of muscle activity was found in the BF at speeds of ≤ 0.28 ms^{-1}. This burst typically occurred around the transition between stance and swing phase, and varied considerably in amplitude between the 5 subjects. However, in all 5 cases, this burst occurred selectively at speeds ≤ 0.28 ms^{-1}, was absent at the two fastest speeds, and tended to decrease in amplitude when speed became slower than 0.28 ms^{-1}. Although similar episodes of activity were apparent in ST as well, these bursts were considerably smaller in amplitude than those found in BF. A typical example of this extra burst in the BF, taken from a single subject can be seen in figure 3. Note that for this particular subject the amplitude of this extra burst in the BF at 0.28 ms^{-1} exceeds the peak amplitude of this muscle at 1.39 ms^{-1} by 22%. Although a similar phenomenon can be seen in ST, the amplitude of this burst is much smaller than in the BF and reached only 28 % of peak ST activity

at 1.39 ms^{-1}, for this subject. Inspection of figure 2 further suggests that a large burst emerges in RF during late stance / early swing phase at 1.39 ms^{-1}. Although this burst was never absent, it was present only in a strongly reduced form at speeds ≤ 0.83 ms^{-1}. This speed dependent burst was found in all of our 9 subjects. Note that a similar epoch of speed dependent activity is not apparent in VM.

During normal human walking, TA shows two distinct bursts of activity during the course of the gait cycle. The group ensemble averages for this muscle show that its second burst (late swing – early stance phase) is more strongly affected by walking speed than is its first burst (early swing). At very slow walking speeds (≤ 0.28 ms^{-1}), the second burst is reduced strongly in amplitude so that the overall profile at these speeds has arather uniphasic appearance.

Although the amplitude of SO and MG activity is strongly reduced at slow speeds, the ensemble average profiles at these speeds still shows a clear resemblance with the typical profiles found at normal speeds. In PL, a local change in the shape of its burst becomes apparent at very slow speeds, due to a shift in the peak of activity from late stance to midstance.

Changes in the neuromuscular gain functions

Figures 4 and 5 depict the estimated gain functions for all eight muscles, as they were found for three specific comparisons between walking speeds (1.39 ms^{-1} vs. 0.83 ms^{-1}; 0.83 ms^{-1} vs. 0.28 ms^{-1}; 0.28 ms^{-1} vs. 0.06 ms^{-1}). Between 1.39 and 0.28 ms^{-1}, gain values are generally positive, indicating an decrease in EMG amplitude with decreasing walking speed.

In BF, a short phase with negative slope values was found at late stance / early swing between 0.83 ms^{-1} and 0.28 ms^{-1}, whereas between 0.28 and 0.06 ms^{-1}, large positive gain values were found for this phase of the gait cycle. This suggests that, during this particular phase, BF activity increases when walking speed approaches 0.28 ms^{-1} and then again decreases when walking is further slowed down to speeds slower than 0.28 ms^{-1}. As becomes apparent from figure 4, a similar relationship between walking speed and EMG amplitude was not found for ST. As could be expected, in RF an episode of large positive gain values can be found in late stance / early swing, indicating that , for speeds between 0.83 and 1.39 ms^{-1}, EMG amplitude is strongly dependent on walking speed for this phase of the gait cycle (see figure 4). Note that this epoch of positive slope values is absent for comparisons between the other speeds. Another salient feature of the RF gain functions is the episode of negative values that is found during late swing between

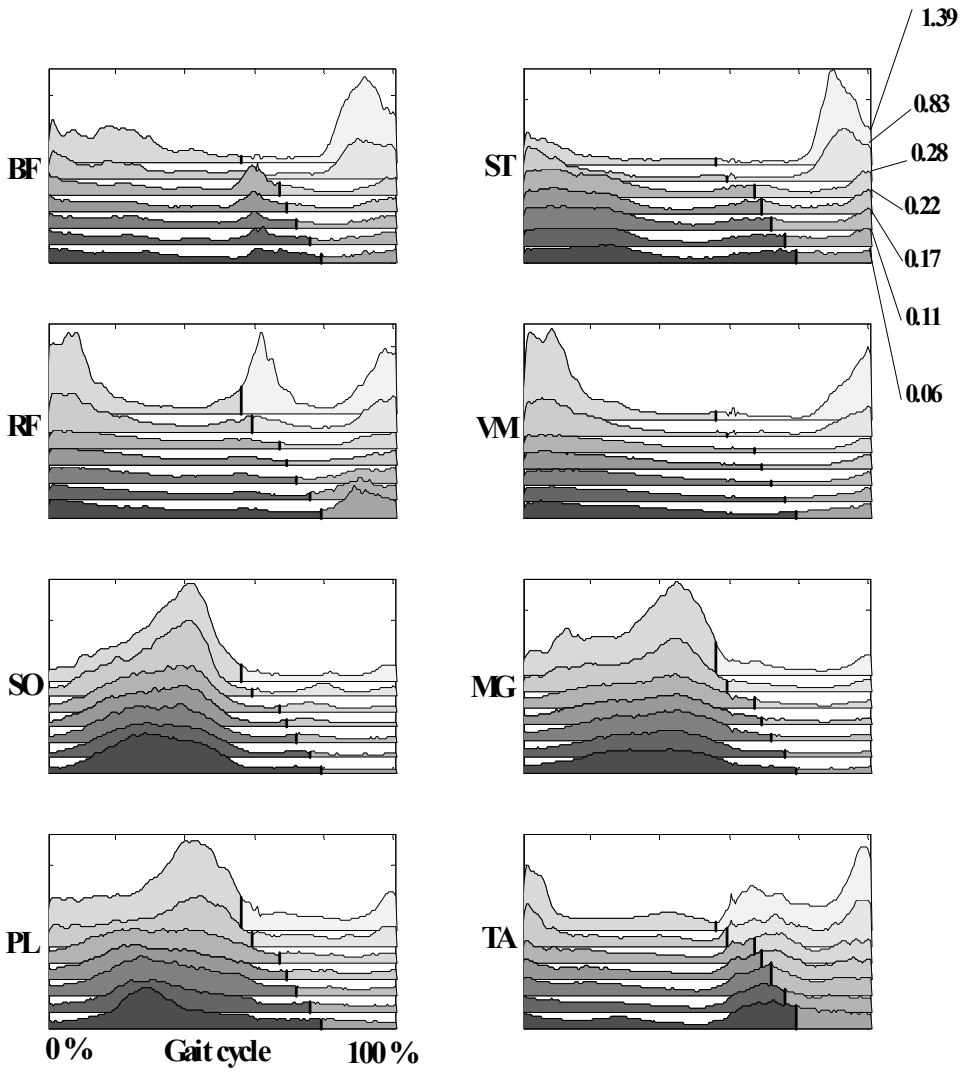

Figure 2 Group ensemble average profiles for all 8 muscles at all 7 speeds employed in the experiment. Profiles are shown in order of speed, with the treadmill speeds given on the right (ms^{-1}). (Abbreviations: BF = Biceps Femoris , ST = Semitendinosus, RF = Rectus Femoris, VM = Vastus Medialis, MG = Medial Gastrocnemius, SO = Soleus, PL = Peroneus Longus and TA = Tibialis Anterior)

0.06 and 0.28 ms^{-1}. Apparently, the amplitude of muscle activity increased for this period of the gait cycle when walking speed became increasingly slow.

In PL, a short period with negative gain values can be seen during midstance, reflecting increased activity for this phase at very slow speeds (see figure 5). For SO and MG, the gain functions remain relatively constant over all 3 speed comparisons, indicating that the activity of these muscles continues to be sensitive to speed changes, even at the slowest walking speeds. Note that, within the lowest speed ranges, the PL slope function deviates rather clearly from those found for

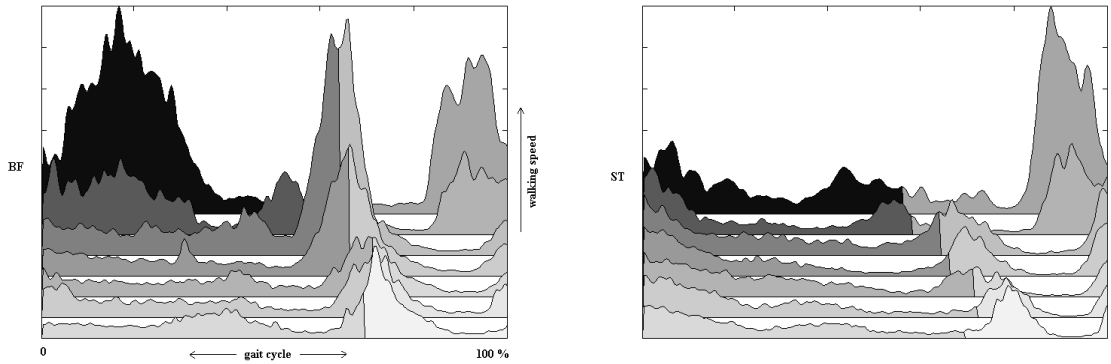

Figure 3 Left panel: Average EMG profiles of BF for one subject. Right panel: Average EMG profiles of ST for the same subject. Note the extra burst in BF that occurs in late stance/ early swing phase at slower walking speeds ($\leq$ 0.28 ms^{-1}). Also note the much smaller amplitude of a similar burst in ST.

MG and SO, despite the relative similarity of the slope functions of MG, SO and PL between 0.28 ms^{-1} and 1.39 ms^{-1}.

Discussion

The results of the present study show that, in general, the amplitude of lower extremity muscle activity tends to increase with the speed of progression. In RF, TA, PL and BF, changes in the pattern of activity were found that could be uniquely related to specific speed ranges. For most muscles, the gain functions that regulate the speed related adaptations in muscle activity appeared to be relatively stable down to speeds as slow as 0.28 ms^{-1}. When speed was decreased to below 0.28 ms^{-1}, negative gain values could be detected in some muscles (PL and RF) that are indicative of an increase in activity with decreasing speed.

In order to exert maximum experimental control over the extremely low walking speeds employed in this study, it was decided to conduct the experiment on a treadmill rather than on a walkway. When walking on a solid surface, extremely slow speeds may be realised by sequencing of more or less 'static' postures. In contrast, on the treadmill,both cadence and step length are imposed on the subject by the moving walking surface. In principle this may have affected our results to some extent but the differences are likely to be very small. Indeed, the limited number of EMG studies that have compared treadmill walking and floor walking, failed to show clear differences in the underlying patterns of muscle activity, other than minor changes in the amplitude of muscle activity **(**Murray et. al, 1985; Arsenault et. al, 1986). While these studies did not extend to the very low speed range it is unlikely that major differences should appear.

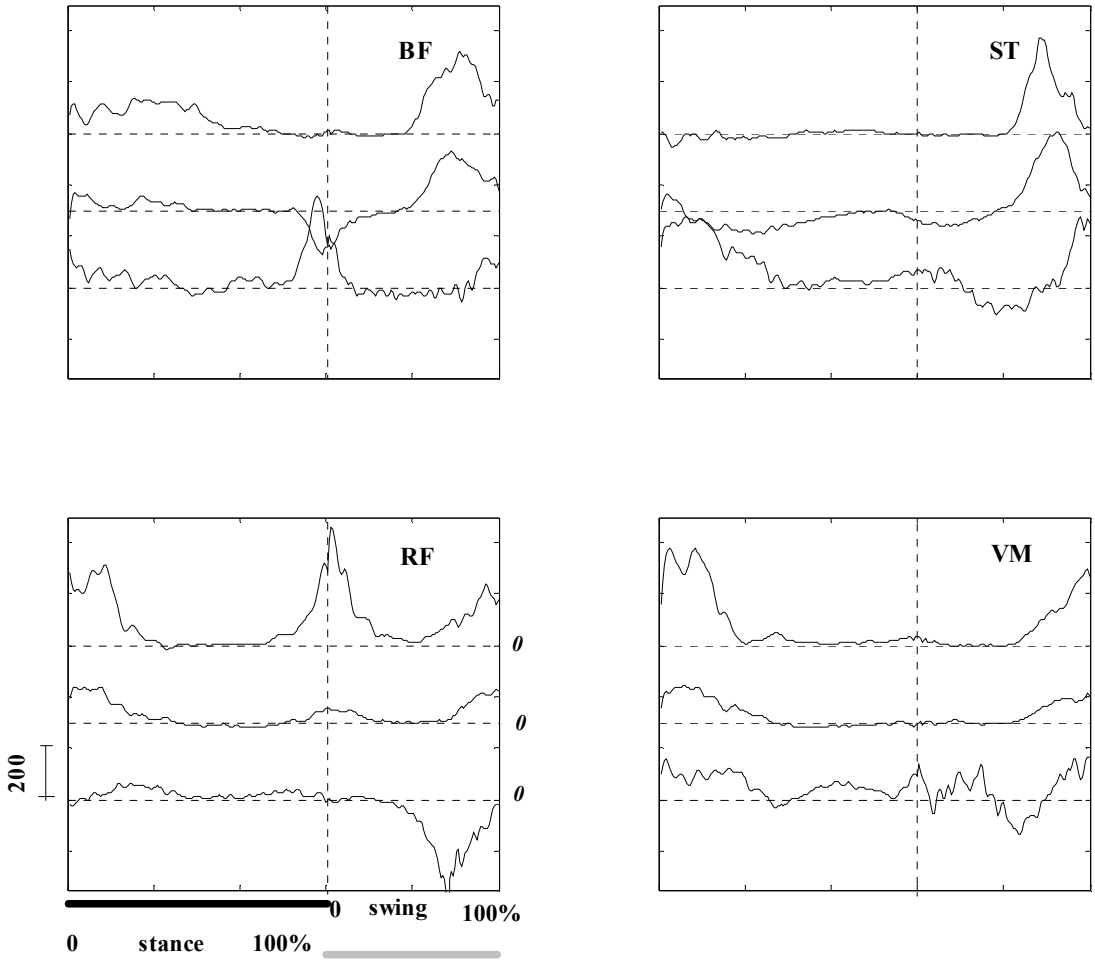

Figure 4 Estimated gain functions for BF (Biceps Femoris), ST (Semitendinosus), RF (Rectus Femoris) and VM (Vastus Medialis) for 3 separate comparisons of walking speeds: Top = 1.39 ms^{-1} vs. 0.83 ms^{-1}; Middle = 0.83 ms^{-1} vs 0.28 ms^{-1}; Bottom = 0.28 ms^{-1} vs 0.06 ms^{-1}; Positive slope values indicate a postive linear relationship between walking speed and EMG amplitude (i.e. a decrease in EMG amplitude with decreasing walking speed), whereas negative slope values indicate a negative linear relationship between walking speed and EMG amplitude (i.e. an increase in EMG amplitude with decreasing walking speed). Vertical lines indicate the onset of the swing phase. Note that the gain functions are offset to enhance clarity.

Lower leg muscles

In the calf muscles, it was found that the activity during late stance (50 – 80% of the stance phase) is strongly affected by walking speed. The large positive gain factor values indicate that the amplitude of MG and SO activity during this phase of the gait cycle is strongly dependent on walking speed, a result that is in agreement with earlier work (Duysens et. al, 1991). Quite in contrast, MG and SO activity during 30 – 50% of the stance phase appears to be relatively stable over walking speeds. The latter finding is in accordance with an earlier study by Winter

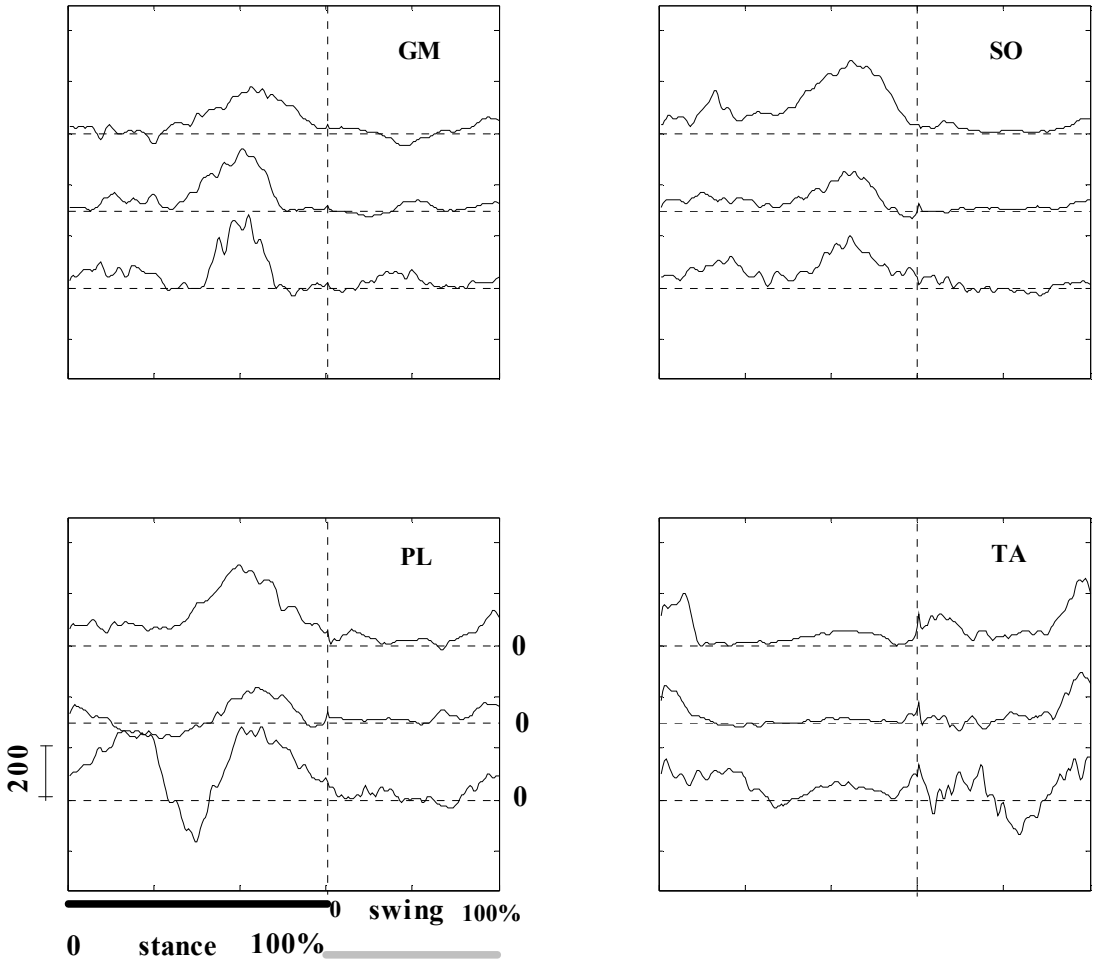

Figure 5 Estimated gain functions for MG (Medial Gastrocnemius), SO (Soleus), PL (Peroneus Longus) and TA (Tibialis Anterior) for 3 separate comparisons of walking speeds: Top = 1.39 ms^{-1} vs. 0.83 ms^{-1}; Middle = 0.83 ms^{-1} vs 0.28 ms^{-1}; Bottom = 0.28 ms^{-1} vs 0.06 ms^{-1}; For further explanation, see figure 4

(1983) who found that the amount of energy absorption by the ankle plantarflexors during weight acceptance was relatively insensitive to differences in walking speed. Furthermore, a recent simulation study showed that MG and SO deliver nearly all of the positive work during late to terminal stance (40-60% of the gait cycle) and that the contribution of these two muscles to forward progression is larger than that of all other muscles taken together (Neptune et. al, 2001). The importance of this mechanism is underscored by the finding that late stance MG and SO activity continues to be affected by walking speed, even within the lowest speed range (0.28 – 0.06 ms^{-1}). Apparently, even when walking speed is decreased to almost standing still, the plantar flexors still contribute to the production of forward motion. The present result of a continuous scaling of EMG during late stance also demonstrates that detection of leg loading in this phase through calf muscle tendon organs could

be a useful mechanism to control swing onset in humans, in analogy with the cat, for the full speed scale (Duysens et. al, 2000).

At speeds ≥ 0.28 ms^{-1}, both the global phasing characteristics of PL, SO and MG, as well as their associated gain functions show strong similarities. However, at speeds < 0.28 ms^{-1}, the PL gain function shows a short episode with negative values, indicating an increase in activity with a decrease in walking speed. It is believed that PL plays a role in the maintenance of frontal plane balance during walking, and especially its activity during the single support phase appears to be important in this respect. An earlier study on the function of PL during walking showed that PL activity during foot flat is sensitive to speed changes, and that its activity in this phase increases when walking speed is decreased (Louwerens et. al, 1995). It is known that walking speed affects the mediolateral distribution of foot loading, and that the pattern of foot loading shifts to the lateral side of the foot when walking speed is decreased (Perttunen and Komi, 2001). Because the lateral component of the ground reaction force as well as the amount of foot eversion are correlated to the amplitude of PL activity during midstance (Matsusaka, 1986), it can be argued that the negative relationship that was found between walking speed and PL activity for speed < 0.28 ms^{-1} serves to generate additional foot everting forces in order to prevent excessive foot inversion during the single support phase.

During normal human walking at regular speeds, TA activity is represented by a biphasic pattern that peaks in early swing (first burst) and in late swing-early stance (second burst). In the present study, it was found that the second burst of TA activity is more sensitive to changes in walking speed than the first burst, eventually resulting in a near absence of the second burst at the slowest speeds. While the first burst of TA activity serves to dorsiflex the foot and achieve foot clearance during early swing, the second burst represents an anticipatory response to the upcoming leg loading during early stance. Because the amount of leg loading increases with walking speed, the amplitude of the second TA burst that anticipates this loading can be expected to be velocity-dependent. In contrast, foot clearance must be achieved regardless of walking speed, which explains why TA activity during early swing appears to be relatively insensitive to speed changes. The strong speed sensitivity of the second TA burst (end swing-early stance) may be of clinical interest since the absence or reduced amplitude of this burst has been reported in Parkinsonian and hemiparetic persons (Cioni et. al, 1997; Burridge et. al, 2001). Because these pathologies are usually associated with strongly reduced gait speeds,

the results from the present study suggest that such phenomena could at least partly be explained by a speed effect.

Upper leg muscles

Between 0.83 ms^{-1} and 1.39 ms^{-1}, a substantial increase in RF activity was found at terminal stance - early swing. Although this burst was never absent, not even at the slowest speeds, peak activity in this phase at 0.83 ms^{-1} was only 20.8% of peak RF activity at 1.39 ms^{-1} in the same phase. This phenomenon has been described on numerous occasions in the literature (Shiavi et. al, 1987; Perry J, 1992; Scott et. al, 1994; Annaswamy et. al, 1999; Nene et. al, 1999). This particular epoch of RF activity is assumed to generate an additional hip moment to accelerate the leg forward at higher walking speeds, and is probably related to the deceleration of the shank during initial swing. At walking speeds ≤ 0.28 ms^{-1}, additional RF activity was found during late swing while similar speed dependent effects were absent in VM. The independence of the activities in these two muscles is important to demonstrate that in this case RF activity did not result from vastus cross talk. Recent evidence has indeed pointed out that end swing RF activity is sometimes due to cross talk from the vasti (Nene et. al, 2002).

The origin of the negative speed-amplitude relationship that was found for RF at the end of the swing phase at very slow speeds is likely to be found in its action on the hip. It can be hypothesized that, at very slow walking speeds, the pendulum like properties of the swinging leg can no longer be utilized for forward progression throughout the full swing phase. Instead, the passive dynamics may force the leg to swing backwards and to prematurely end the swing phase. Additional RF activity may be used to establish extra hip flexion and counteract this problem.

In BF, an extra epoch of activity was found during late stance/ early swing when walking speed was decreased to 0.28 ms^{-1}. When speed was lowered to < 0.28 ms^{-1}, this burst gradually decreased in amplitude. Although this speed dependent component of BF activity has been described on previous occasions in the literature (Shiavi et. al, 1987; Perry, 1992), it did not yet receive much attention. The observation that this phenomenon did not occur in all of our subjects suggests that it is possibly related to a neuromuscular strategy to adapt to exceptionally low walking speeds. Since similar bursts of activity were either absent or of considerably smaller amplitude in ST, it can be assumed that this speed dependent phase of BF activity is related to the production of a force aimed at an late stance exorotation of

the lower leg (both muscles are synergists for hip extension and knee flexion but they differ with respect to exorotation).

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