Gait control after stroke

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Summary

Supratentorial stroke may cause weakness or paralysis on the side contralateral to the lesion. As a consequence, functional motor tasks like walking may become difficult or impossible. Because mobility represents a key aspect of independent functioning in daily life, much time and clinical effort is aimed at the restoration of gait following stroke. To obtain optimal treatment results, it is important that the effectiveness of interventions is empirically established and that new treatment strategies are firmly grounded in generally accepted (neuro-) physiological and biomechanical principles of walking. Because the defining feature of hemiparesis relates to the impaired functioning of muscles, dynamic electromyography may provide important information on the control of hemiparetic gait. An important aspect of electromyographic profiles obtained during walking is the timing of muscle activity. At each moment of the gait cycle, the amplitude of muscle activity has to be adapted to momentary task demands (e.g. the supply of body support during stance, foot clearance during swing, etc.). As a consequence, each muscle involved in the production of gait shows its own stereotyped temporal pattern. We know from earlier studies that the patterns of gait related muscle activity in persons with hemiparesis is often characterized by an abnormal temporal structure. This thesis is primarily concerned with these particular abnormalities: what are their origins, do they affect gait function, and do they change over the course of gait recovery? The studies that are described in this thesis attempt to contribute to a better understanding of the causes of gait problems in hemiparesis and how patients to manage to optimize gait performance when muscle function is impaired. Furthermore, these studies may help to decide whether the treatment of gait impairments should be concerned with altering the temporal patterning of muscle activity.

Because a large portion of the hemiparetic population walks at abnormally slow speeds, an adequate interpretation of EMG patterns in this group requires insight into the mechanisms of speed regulation on the lower end of the gait speed continuum. Therefore, **chapter 2** reports on the effects of very slow gait speeds on the neuromuscular patterning in a group healthy young adults. Extreme reductions in walking speed may 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 when gait speed was varied systematically from normal speeds to almost standing still, and to investigate the neuromuscular gain functions that reflect the phase dependent effects of walking speed on EMG amplitude. Nine

healthy young adults walked at seven different walking speeds (1.39, 0.83, 0.28, 0.22, 0.17, 0.11, and 0.06 m s^{-1}) while EMG was recorded from eight 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 m s^{-1}, epochs of Rectus femoris, Biceps femoris and Tibialis anterior activity were found that were typical for defined speed ranges. When walking speed was decreased further (form 0.28 m s^{-1} to 0.06 m s^{-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 during stance, and the altered dynamics of the swinging limb at very slow speeds.

Following hemiparetic stroke, the temporal ordering of lower extremity muscle activity during gait often undergoes radical changes. Although several studies have dealt with the identification of abnormalities in neuromuscular patterning, the majority of these studies used either visual inspection or unreliable numerical methods (e.g. threshold detection) to evaluate the temporal characteristics of EMG patterns. **Chapter 3** describes a study in which the duration of activity in Biceps femoris (BF), Rectus femoris (RF), Tibialis anterior (TA) and Gastrocnemius medialis (GM) was quantified 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 between 24 hemiparetic stroke patients and 14 healthy controls. Periods of muscle activity and inactivity were identified by means of cluster analysis of the filtered and rectified EMG signal. 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 abnormalities 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.

The objective of the study described in **chapter 4** was to establish whether functional recovery of gait in patients with post-stroke hemiparesis coincides with changes in the temporal patterning of lower extremity muscle activity and coactivity. To this end, electromyographic (EMG) data from both legs, maximum walking speed, the amount of swing phase asymmetry and clinical measures were obtained from a group of hemiparetic stroke patients during their postacute rehabilitation, as early as possible after admission in a rehabilitation centre (mean time post stroke 35 days) and 1, 3, 6, and 10 weeks later. EMG data from the first assessment were compared to those obtained from a group of healthy controls to identify abnormalities in the temporal patterning of muscle activity. Within subject comparisons of patient data were made over time to investigate whether functional gait recovery was accompanied by changes in the temporal patterns muscle (co-)activity. EMG patterns during the first assessment showed abnormally long durations of activity in the paretic biceps femoris (BF), in the rectus femoris (RF) of both legs during the single support (SS) phase, and for the paretic gastrocnemius medialis (GM) during the first double support phase (DS1). In addition, the duration of BF-RF coactivation was longer on the paretic side than it was in controls. Over time, the level of ambulatory independence, body mobility, and maximum walking speed increased significantly, indicating that substantial improvements in gait ability occurred. Despite these improvements, durations of muscle (co-) activity and the level of swing phase asymmetry did not change during rehabilitation. More specifically, timing abnormalities in muscle (co-)activity that were found during the first assessment did not change significantly, indicating that these aberrations were not an impediment for functional gait improvements.

From chapter 4, it can be concluded that modification or normalization of the temporal patterning of gait related muscle activity in the lower extremities is not a prerequisite for functional recovery of gait in patients with post stroke hemiparesis. As a consequence, modification of the temporal coordination of

muscle activity during gait should not be regarded an important clinical goal during post-acute rehabilitation. Physiological processes other than improved temporal muscular coordination, such as the increase in muscle force output, must be important determinants of the restoration of ambulatory capacity after stroke.

Despite these findings, it cannot be excluded that recovery related changes in neuromuscular patterning may be evident in other measures than the mean duration of muscle activity within defined gait phases. More specifically, it was hypothesized that gait recovery is associated with the automatization of newly formed patterns rather than with the formation of new temporal patterns during gait, and that recovery results in a more consistent production of neuromuscular patterns. A study to test this hypothesis is described in **chapter 5**. In this study, principal component analysis was used as a data filtering method to investigate the stride to stride variation in the gain (i.e. scaling of the amplitude) of muscle activity patterns, and the stride to stride residual variability (i.e. random fluctuations in amplitude and phase shifts), of Biceps femoris (BF), Rectus femoris (RF), Tibialis anterior (TA) and Gastrocnemius medialis (GM), of both legs in 14 ambulant hemiparetic stroke patients and 15 controls. Patients were assessed early after admission to a rehabilitation center (on average 35 days post stroke) and 1, 3, and 6 weeks later. The results showed that, at the time of the first gait assessment, the amount of stride to stride gain variability as well as the residual variability of BF was abnormally high compared in both legs, compared to the control group. Over time, clear improvements were made in general body mobility, ambulatory independence and maximum walking speed, indicating that recovery of gait had occurred. Over the course of recovery, a significant decrease in global pattern variability was found in the paretic BF, indicating smaller variations in the gain of activity patterns for this muscle. For the other muscles that were studied, no recovery related decreases were observed in either the gain variability or the residual variability of muscle activity patterns. These findings provide support for the idea that temporal patterns of gait related muscle activity are established early after stroke and that recovery of gait is associated with the automatization of these patterns resulting in a decrease in the stride to stride variability of neuromuscular pattern production during gait.

The majority of gait studies on hemiplegic gait have dealt with simple gait tasks in which patients are required to walk 'from a to b' on a straight walkway, or to walk a defined time or distance on the treadmill. Obviously, walking in the real world entails more than walking straight and unperturbed trajectories, and future

research should address the adaptability of hemiparetic walking under more complex conditions. **Chapter 6** of this thesis describes a study on adaptability of spatiotemporal stride parameters in a group of stroke patients. Whereas several animal studies have indicated the important role of the motor cortex in the control of voluntary gait modifications, little is known about the effects of cortical lesions on gait adaptability in humans. Obstacle avoidance tasks provide an adequate paradigm to study the adaptability of the stepping pattern under controlled, experimental conditions. In the present study, an exploratory assessment was made of the failure rate, the preferred stepping strategies (step lengthening vs step shortening), and the spatiotemporal stride characteristics (percentage increases in stride length, duration, and velocity of the crossing and postcrossing strides) during obstacle avoidance in 11 hemiplegic stroke patients and seven healthy controls. Patients were less successful in avoiding obstacles than controls (14% failure rate vs 0.5% in controls), independent of whether the affected or the unaffected leg led the obstacle avoidance. The number of failed trials increased systematically when the available response time became shorter. During successful trials, lengthening of the step was generally preferred over shortening. This bias towards step lengthening was more pronounced in stroke patients (step lengthening in 91% of the trials vs 75% in controls), irrespective of the side of obstacle presentation. For both groups, overall strategy preference did not adhere to a principle of minimal foot displacement, since step lengthening was used even if it would be more spatially efficient to shorten the step. No statistically significant group differences were found for the increases in length, duration, and velocity of the crossing and postcrossing strides. However, for a subgroup of more slowly walking patients, large percentage increases were found in crossing stride length, duration, and velocity. Similar results were obtained for the postcrossing stride, indicating that, for this subgroup of patients, restoration of the normal walking cadence was more difficult. Overall, no systematic differences were found between the affected and the unaffected leg in stroke patients with respect to failure rates, stepping strategies, or spatiotemporal measures of obstacle avoidance. The present findings suggest that the ability to adequately modify the stepping pattern in response to imposed spatiotemporal constraints is impaired in persons with stroke, especially when modifications have to be performed under time pressure. In addition, the stepping strategies employed by subjects with stroke are different from those found in controls, possibly to reduce the complexity of the avoidance maneuver and to enhance safety. Finally, unilateral cortical damage results in an impaired ability to

avoid obstacles on both sides of the body, suggesting that the reduced ability of stroke patients to negotiate obstacles may be related to problems of a more general coordinative nature.

The main conclusion that can be drawn from this thesis is that the patterns of gait related muscle activity in hemiparetic stroke patients are different from those of healthy walkers, but that these abnormalities do not form an impediment for functional gait recovery. The majority of the identified abnormalities possibly reflect compensatory neuromuscular strategies that are developed early after stroke, and that may facilitate, rather than impede, gait performance. This thesis further showed that gait recovery may be associated with a more consistent production of muscle activation patterns during gait. This may indicate that the newly developed activation patterns are automatized over the course of gait recovery. Because automatization of these patterns can only develop when task specific afferent information is available, these findings provide indirect support for the idea that gait ability should be trained in a task specific manner, and with high intensities. Nevertheless, a number of important questions are still unanswered. Future research should reveal which aspects of the muscle activation patterns of hemiparetic walkers reflect primary impairments in neuromuscular control, and which aspects represent compensatory strategies. Further, studies on timing abnormalities in stroke should make use of more sophisticated analytical tools that take into account the continuous nature of EMG patterns. The customary reduction of EMG signals to step functions (on/off detection) may result in a loss of valuable information on the muscular control of gait.