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Gait control after stroke

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Chapter 1

General introduction and
outline

1. The neuromuscular control of hemiparetic gait

One of the most common symptoms of a supratentorial cerebrovascular accident (or stroke) is hemiparesis, i.e. weakness of muscles on the body side contralateral to the lesion. One of the consequences of hemiparesis is that functional motor tasks like walking may become difficult to perform or even impossible. Although the majority of stroke patients that initially suffers from gait impairments eventually relearns to walk, for a substantial number of patients these problems persist throughout the subacute and chronic phases of stroke (Jorgensen et al. 1995; Baer and Smith, 2001). Because the ability to walk represents an important ingredient of independent functioning, much clinical effort is aimed at the restoration of gait ability in persons with post-stroke hemiparesis. In order for treatment to be efficient (i.e. to attain optimal effects within restricted time frames), it is important that (1) concepts and ideas that guide selection of rehabilitation strategies are based on general principles of motor control and learning, and that (2) the effectiveness of treatments is empirically established.

Common to all clinical interventions that are used for gait re-education, is the intention to modify the control processes that underlie the production of gait. From exercise therapy to the use of gait orthoses (e.g. to facilitate foot clearance during the swing phase), all interventions attempt to alter the way in which gait is controlled, although in different ways. If we want to understand the effects of these treatment strategies or design new ones, we need to have a clear idea of how healthy gait is controlled, how these control processes are altered in hemiparesis, and which aspects of gait control are sensitive to treatment and recovery. Obviously, clinical measures of overall mobility, ambulatory independence, or participation in ADL are of very limited value in addressing control aspects of gait. Information on the gait control should be obtained primarily by studying the movements, forces, and moments involved in gait, as well as their neural and neurophysiological origins.

A crucial element in the chain of processes that leads to the realization of gait is the activity of muscles, because this represents the sole mechanism that allows the walker to exercise active control over the movement of body segments. The contractile force that is exerted by a muscle needs to be scaled appropriately for each instant of the movement sequence, resulting in continuous changes in the magnitude of force output as a function of gait cycle time. Consequently, electromyographic recordings of muscle activity during gait are often described in terms of their 'timing' and their 'amplitude' as separate elements of muscular

control. The focus of this thesis will be primarily on the way hemiparetic walkers control the timing of muscle activity in the lower extremities. Although the defining characteristic of hemiparesis relates to weakness of the muscles contralateral to the lesion (which may be expressed in a reduction in the amplitude of muscle activity), aspects of neuromuscular coordination can be captured better by looking at the temporal features of muscle activity. This is particularly relevant because the re-coordination of gait represents an important goal in gait re-education programs for patients with post stroke hemiparesis.

What information on the control of gait is contained in the temporal layout of muscle activity? The goal of walking is to move the body from location A to location B. In order to achieve this goal, four major subtasks have to be accomplished. First, propulsion has to be supplied to the body to cause it to move. Second, for the larger part of the gait cycle, $2/3^{\text{rd}}$ of total body mass has to be carried by only one leg so that continuous postural control has to be exerted over the body to ensure (mediolateral and antero-posterior) balance during walking. A third and related coordinative challenge is to supply adequate support against gravity for the head-arm-trunk segment, particularly during the single support phase. Finally, during the forward progression of the leg in the swing phase, sufficient vertical clearance of the foot has to be realized in order to prevent premature contact with the support surface which may result in stumbling. For healthy walkers and patients alike, these four subtasks have to be accomplished adequately, because failure on either one of these subtasks implies either falling or standing still. Because these subtasks are temporally coupled to particular phases of the gait cycle, safe and effective realization of gait requires accurately timed muscle activity. In healthy walkers, this generally results in rather stereotyped patterns that have been documented extensively (Inman et al., 1981; Winter, 1991; Perry, 1992).

Following stroke, the function and tissue properties of muscles, and their timing characteristics during walking often change dramatically. Damage to cortical or subcortical motor areas may lead to paresis in muscle groups contralateral to the lesion, due to the reduced number of active motor units, their reduced firing rates, and their less synchronized firing patterns (Young and Mayer, 1982; Gemperline et al., 1995). A few weeks after stroke, spasticity may evolve as a result of central disinhibition of reflexes which may eventually result in a structural shortening of the involved muscles (i.e. contractures). These impairments may all affect the temporal layout of muscle activity over the gait cycle. The temporal abnormalities that characterize the neuromuscular control of hemiparetic gait have been a

frequently adopted subject for research, and in the following section a brief overview is given of these studies.

2. The temporal patterning of muscle activity in hemiparetic gait: a brief overview

A common conclusion from studies on the temporal patterning of muscle activity in stroke patients, is that there is a large variation between patients. This variation is due to the individual differences in the size, the type, and location of the lesion, as well as to the different compensatory strategies that are used by patients to optimize locomotor output in response to the altered neuromuscular constraints. As a consequence, gait related muscle activity in hemiparesis cannot be described by a single stereotyped set of muscular activation patterns, and attempts that have been undertaken to categorize patients according to the abnormalities in their muscle activity patterns have not been very convincing (Knutson & Richards, 1979; Shiavi et al., 1987). However, on the level of individual muscles a few common characteristics can be identified. Perhaps the most common temporal abnormality in the electromyographic patterns in post stroke hemiparesis is the prolonged activity of hamstring and quadriceps muscles during the stance phase. Whereas in healthy subjects these muscles are active only during the late swing and early stance phase, several studies in hemiparetic patients have shown that the activity of these muscles extends to midstance and may even cover the entire stance phase (e.g. Hirschberg and Nathanson, 1952; Peat et al., 1976; Knutson and Richards, 1979; Shiavi et al., 1987). Although the prolonged activity of upper leg muscles may be interpreted as part of a involuntary extensor synergy (cf. Perry, 1993), it can alternatively be explained as a voluntary compensatory reaction to provide an additional support moment during the stance phase through increased muscular stiffness at the hip and the knee joints.

Spasticity may be another source of temporal abnormalities in the patterning of muscle activity in patients with upper motor neuron lesions. In particular, spasticity of the calf muscles has been reported on several occasions and is often associated with premature activity of these muscles during the initial stance phase (Hirschberg & Nathanson, 1952; Perry, 1978, 1993; Knutson & Richards, 1979; Lamontagne et al. 2001 etc.). In addition, calf muscle spasticity has also been associated with increased coactivation levels between calf muscles and the tibialis anterior during the late swing and early stance phase (BurrIDGE et al., 2001; Hesse et al., 1996) or with the absence of the stance-swing burst of tibialis anterior (BurrIDGE

et al., 2001; Perry, 1978, 1993). The presence of calf muscle spasticity has often been attributed to disinhibited stretch reflexes. However, it is difficult to state unequivocally whether the reported timing abnormalities are a direct result of spasticity or whether these features reflect an adaptive strategy to prevent foot collapse when the foot lands in a plantarflexed position as a result of spasticity of the calf muscles, contractures, or ankle dorsiflexor weakness. In this context, it is interesting to note that similar patterns of premature calf muscle activity have also been reported for habitual toe walkers (Papariello et al., 1985; Policy et al., 2001; Griffin et al., 1977; Davids et al., 1999). Similarly, the reduced amplitude of Tibialis Anterior activity during end swing/ early stance that has been reported (BurrIDGE et al., 2001; Perry, 1978, 1993) may reflect the reduced need for dorsiflexor activity in case one lands on a plantarflexed foot.

Post stroke hemiparesis may also be characterized by spasticity of the quadriceps (Faist et al., 1999; Yelnik et al., 1999) resulting in overactivity of the rectus femoris during the swing phase of gait (cf. Perry, 1987; Sutherland et al., 1990). Although this particular timing abnormality has often been associated with spastic stiff legged gait, the production of excessive knee extension moments may not be the only factor that contributes to insufficient knee flexion during swing. Other factors, such as abnormal kinematic conditions at toe-off and the initial conditions of the swing phase, e.g. through weakness of hip flexors or ankle plantarflexors, may also contribute to the diminished knee flexion seen in stiff-knee gait (Kerrigan et al., 1991; Kerrigan and Glenn, 1994; Piazza and Delp, 1996; Goldberg et al., 2003).

3. Outline of this thesis

The overview given above is by no means complete, and the body of literature on timing abnormalities in hemiparetic gait is still growing. Nevertheless, there are still a number of intriguing questions that need to be answered. Perhaps the most important question is whether the aberrant temporal control of muscle activity in hemiparesis does, or does not, impair gait function. Although the literature cited above makes clear that hemiparetic gait is often characterized by different temporal patterns of muscle activity, there is still no convincing evidence to show that these abnormalities are related to impaired gait functioning. This question cannot be answered by simply correlating ambulatory dysfunctions with temporal abnormalities, since gait inabilities and temporal aberrations may both stem from a single source without being directly related (e.g. reductions in muscle force output

may impair gait function and may also induce different neuromuscular control strategies). One way to study the relationship between walking ability and neuromuscular abnormalities is to use a longitudinal design and assess whether recovery related developments in gait function correspond to changes in the temporal structure of muscle activity during gait. Such a study will be described in *chapters 4 and 5* and forms the core of this thesis. Information on the relationship between functional recovery and neuromuscular control is important because it may shine light on the question whether modification or normalization of neuromuscular patterns should be a target for gait rehabilitation. Furthermore, the growing popularity of electromyographic analysis in the evaluation of treatment requires a good understanding of neuromuscular patterns in hemiparesis and how they are related to gait function.

Following stroke, gait speed is often dramatically reduced (von Schroeder et al., 1995), and studies on the temporal patterning of muscle activity in stroke patients need to account for the possible effects of gait speed. Earlier studies have shown that walking speed strongly affects the amplitude of muscle activity but that the temporal characteristics remain largely unchanged (Hof et al., 2002; Ivanenko et al., 2005). However, the speeds that were studied have been confined to speeds faster than 0.56 ms^{-1} , and it is not known how the timing of muscle activity is affected at slower gait speeds. At the very low end of the walking speed continuum, task demands may become significantly different from those experienced at comfortable speeds. In particular, demands in terms of postural control may be increased, and altered swing phase dynamics may lead to a different, possibly more active, control of the swinging limb. In *chapter 2*, the activity patterns of 8 lower extremity muscles will be studied while walking speed was varied systematically from normal speeds (1.38 ms^{-1}) to almost standing still (0.06 ms^{-1}). Patterns of muscle activity obtained at different speeds were used to calculate the gain functions that describe the local modifications in the amplitude of muscle activity that are used to implement speed changes.

In *chapter 3*, abnormalities in the temporal patterning of muscle activity will be described in a group of 24 hemiparetic stroke patients. As early as 1951, Wortis and colleagues recorded muscle activity from relevant muscle groups in patients with post stroke hemiparesis, an endeavour that has been repeated by others on several occasions since (Hirschberg and Nathanson, 1952; Pinzur et al., 1987; Peat et al., 1976; Knutson & Richards, 1976; Shiavi et al., 1987). A common problem with regard to these studies is that abnormalities were identified on the basis of

visual inspection or unsound statistical criteria (e.g. threshold detection), and that it is often difficult to interpret such predicates as ‘premature’ or ‘prolonged’ in the description of muscle activation patterns. Furthermore, little is known about the frequency of particular timing abnormalities and their relationship with the severity of the paresis. This information is important, especially since the increased popularity of clinical gait analysis requires quantitative and statistically tested databases. In chapter 3, cluster analysis of the rectified and filtered EMG signal will be employed to describe the duration of (co-)activity during 4 distinct phases of the gait cycle for 4 important muscles in both legs. In addition, the relative frequency of particular abnormalities was noted, as was their relationship with a clinical measure of recovery (i.e. the Brunnstrom motor stage).

Chapter 4 addresses the question whether gait recovery in the subacute phase of stroke coincides with changes in the gross temporal patterning of muscle activity. The majority of electromyographic studies on hemiparetic gait were designed to identify abnormalities in muscle activity patterns, or addressed whether particular interventions aimed at the promotion of gait ability (e.g. the use of orthoses, neurolytic drugs, treadmill training, etc.) resulted in an altered temporal control of muscle activity. However, it is still unclear whether recovery of gait is associated with changes in the patterning of muscle activity. Although a number of studies have shown that clinically successful interventions may coincide with changes in muscle activity patterns (e.g. Hesse et al, 1996, 1999c; Geboers et al., 2002; Buurke et al., 2004) it remains unclear whether these changes play any causal role in establishing gait improvements, or if they represent an epiphenomenon of the altered neurophysiological or biomechanical constraints induced by the intervention. Until recently, only one study addressed the changes that occur over the course of gait recovery. Shiavi and co-workers (1987) performed a study in which electromyographic profiles from the lower extremity of 12 patients were compared between the early (1-10 weeks) and the late (6-24 months) phases post stroke, using a modification of the classification proposed by Knutson and Richards (1979). Although the authors noticed changes in the type of muscle synergies displayed by patients, these changes were identified on the basis of visual inspection and it remained unclear whether changes in gait speed could have affected these patterns. In the study described in chapter 4, the duration of (co-) activity from four important muscles in both legs was assessed, during the subacute phase of stroke (early after admission, 1, 3, 6, and 10 weeks later) using objective, statistical criteria. Furthermore, gait speed was kept constant throughout the

assessments. In addition, the general body mobility (Rivermead Mobility Index), ambulatory independence (the Functional Ambulation Categories), swing phase asymmetry, and maximum gait speed on the treadmill were scored to assess whether gait recovery did occur.

Chapter 5 describes a study in which the recovery related changes in the stride to stride variability of muscle activity are addressed. It can be argued that the early phases of gait recovery after stroke involve the formation of new neuromuscular patterns, but that recovery during the subacute phase of stroke may be concerned primarily with improved stability of these newly acquired patterns. Studies on the learning of bimanual coordination patterns have shown that the global coordinative patterns are established already early during training, and that the larger part of the learning phase is concerned with improvements in the cycle to cycle consistency of the pattern (Wenderoth et al., 2001). Apparently, the formation and stabilisation of coordinative patterns may follow different time courses. Similar principles may hold for gait relearning after stroke. Stride to stride variability of neuromuscular patterns can arise from (i) Variations in amplitude within one common pattern of activity (gain variability), and (ii) Random (i.e. not belonging to a common pattern) variations in amplitude or random phase shifts. These two sources of variability should be clearly distinguished. Recently, the use of principal component analysis has been proposed as a data filtering method (Daffertshofer and Lamoth, 2004; Lamoth et al., 2004). In chapter 5, this method will be utilized to assess recovery related changes in the gain variability and the residual variability of muscle activity patterns.

The majority of studies on gait control in hemiparesis deal with unperturbed gait in which subjects are instructed to walk 'from A to B' at a more or less fixed speed (but see Lamontagne et al., 2002, 2005). As a consequence, the spatiotemporal step parameters used by patients will be relatively fixed from stride to stride. Nevertheless, the ability to make on-line modifications in step length represents an important mechanism that can be used e.g. to accommodate different walking surfaces or to enhance ambulatory safety. In the study that is described in *chapter 6*, an obstacle avoidance paradigm is used to investigate the ability of hemiparetic subjects to alter their step length during ongoing gait. Obstacles were presented to the paretic and the non-paretic leg in patients, under varying degrees of time pressure. Assessments were made of the failure rates, the stepping strategies used (stride shortening or stride lengthening), and the changes in stride length, duration, and velocity of the crossing stride and the post crossing stride.

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