CHAPTER 1

INTRODUCTION

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Each year approximately 30.000 new stroke patients are reported in the Netherlands.[1] Due to the ageing of the population it is expected that this number will increase rapidly the next few decades, since stroke incidence increases exponentially with age.[2,3] An estimated 70% of the patients who survive a stroke are unable to walk independently during the first three to four weeks post-stroke.[4] Clearly, regaining independent walking ability forms a major goal of all rehabilitation programs and is, indeed, of great significance to patients who have suffered a stroke.[5,6] Although the reported figures vary, approximately 50-85% of the patients who survived a stroke will eventually regain some degree of walking ability.[4,7] Several studies show that most of the motor recovery following stroke occurs within the first 3 months post-stroke and that the initially steep recovery curve levels at about 6 months to a year post-stroke.[4]

Characteristics of hemiplegic gait

The population of stroke patients is a heterogeneous group. Severity but also location and type of stroke determine to a large extent the symptoms and outcome. Hence, patients who eventually regain some form of walking ability may vary greatly in walking speed, spatio-temporal characteristics and kinematic gait patterns. Nevertheless, in a number of studies it was attempted to classify hemiplegic gait patterns[8-12] and it appears that some specific movement patterns can be observed in sub-groups of patients.

The average walking speed of stroke patients is lower than that of healthy controls but the reported values vary depending on the severity of the stroke, the time post-stroke and the age of the subjects.[13] Compared to healthy controls, patient's stride lengths are smaller and the duration of gait cycles is longer.[14] Hemiplegic stroke patients show prolonged double support phases in their gait cycle, especially the double support phase that precedes the swing phase of the hemiplegic side. It is assumed that this is caused by a prolonged duration of the pre-swing phase on the hemiplegic side, as a result of insufficient power and inappropriate initiation of hip flexor muscles.[10] Furthermore, the single support

phase on the hemiplegic side is relatively short in relation to the duration of a complete cycle,[10,13] a finding which is related to a decreased ability to bear weight on the hemiplegic side.[15,16]

Concerning patterns in joint kinematics Olney and Richards[13] concluded in their review that hemiplegic gait can be classified by a combination of (1) a reduced hip joint angle amplitude in the sagittal plane, caused by a decreased hip flexion at heel-strike and a decreased hip extension at toe-off, (2) a reduced knee joint angle amplitude caused by increased knee flexion at heel-strike and decreased knee flexion at toe-off and during swing and (3) increased plantar flexion of the ankle at heel-strike and during swing and decreased plantar flexion at toe-off. Abnormalities in these joint kinematics often lead to secondary compensations in other body segments. For example, a reduced knee flexion during swing can be accompanied by circumduction or upward pelvic tilt.[17,18]

The relation between gait characteristics and functional recovery

Functional recovery of walking ability is often quantified by employing clinical measures, such as the Bartel Index,[19,20] the Rivermead Mobility Index[21] or the Functional Ambulation Categories[22] but also gait velocity[23] or walking distance[24] are frequently used as measures for recovery. Recovery of body functions, such as muscle strength, can be assessed by measures such as the Fugl Meyer[25] or the Motricity Index.[26] It is clear that these measures correlate highly: when body functions such as muscle strength recover to normal, chances are high that functional recovery of walking ability will be present as well. Another means of recording recovery of gait after stroke is gait analysis in which the specific characteristics of hemiplegic gait patterns can be analysed. Again, patients whose gait patterns recover towards a normal gait pattern will most likely show functional recovery. For example, patients who show a higher degree of symmetry in their gait, generally walk faster than those who show an asymmetrical gait pattern.[27,28]

These high correlations between functional recovery and recovery of body functions and gait patterns, however, do not necessarily mean that the recovery of body functions or gait patterns towards a normal value are always a

requirement for functional recovery. In some patients the impaired body functions may lead to gait patterns that reflect compensations, which may be energy inefficient in a normal gait pattern and are therefore considered pathological. In a stroke patient, however, structural changes have taken place in the central nervous system and the changed motor patterns may reflect adaptations that are optimal for the altered state of the system. The relation between the recovery of gait patterns and functional recovery of walking ability might therefore be less straightforward as one may initially think. The observed spatio-temporal and kinematic changes, described above, may in fact be compensatory patterns that facilitate functional recovery of walking ability.

Unilateral spatial neglect and walking

In many studies it is shown that the presence of unilateral spatial neglect is associated with poor outcome after stroke and impedes functional recovery.[29-33] Patients with neglect perform at a lower level than patients without neglect on both cognitive and sensory-motor measures, show poorer recovery of motor function and are more impaired in activities of daily living.[34,35] Although extensive research is available on the relation between neglect and motor performance concerning the upper extremities,[36-38] little is known on how neglect influences motor performance with regards to the lower extremities and in particular walking. There is of course a strong indirect relation between the presence of neglect and impaired motor ability, caused by the severity of the stroke: the more severe and extensive the lesion, the more impaired motor ability and the more persistent and severe neglect will be. However, there *are* stroke patients who are able to walk and suffer from neglect and clinicians report specific movement patterns in these patients during walking. They tend to drift towards the not neglected side and bump into objects, like doorposts, on the neglected side. This seems to be contradictory, because in order to bump into an object on the neglected side, a patient would need to drift towards the neglected side.

Some of the walking behaviour in neglect patients may be explained by a model proposed by Karnath and by recent work on prism adaptation treatment of neglect. Karnath et al.[39] argued that the process of converting the sensory input

coordinates from the periphery into an egocentric, body-centred coordinate system is disturbed in neglect patients. The disturbed coordinate conversion introduces a systematic error, which results in a deviation of the spatial reference frame towards the ipsilesional side. They argue that neglect patients will systematically displace their subjective orientation of the sagittal midplane, i.e. their 'subjective straight ahead', to the ipsilesional side. Karnath et al.[40] confirmed this hypothesis in a laser-pointing task performed by neglect patients. Patients were asked to point a laser to the position, which they felt to lie exactly 'straight ahead'. Patients positioned the laser approximately 15° to the ipsilesional side of their objective body orientation. Karnath's model is further supported by the results of prism adaptation studies.[41,42] These studies show that neglect patients may benefit from prism adaptation treatment; a treatment which is specifically aimed at shifting the subjective body midline (back) to the contralesional side.

How may this theory refer to walking? When walking straight ahead, but not specifically towards a target, healthy subjects will walk toward what they feel as 'straight ahead'. This would cause neglect patients to drift towards their ipsilesional side, since their subjective 'straight ahead' is displaced to this side. However, when explicitly walking towards a target, such as a doorway, patients will need to maintain their heading by aligning their subjective body midline with the target. This means that neglect patients need to rotate their objective body position to the contralesional side, to adjust for the ipsilesional displacement of their subjective body midline, which will cause a heading error to the contralesional side. Similar heading errors can be induces by means of prism glasses. It was shown that when subjects walk towards a target wearing prism glasses, their walking trajectory will describe a curvilinear path deviating to the side of the heading error. In other words, the heading error to the contralesional side in neglect patients will cause them to walk towards the target describing a walking trajectory, which is curved towards the contralesional side. This may explain the reports of clinicians that when neglect patients walk through a doorway they frequently bump into the door post on their neglected side.

AIM AND OUTLINE OF THE THESIS

The studies presented in this thesis were performed as part of a national program, which main goal was to gain more insight into the mechanisms that are involved in the recovery of gait after stroke and to what extent these mechanisms may be influenced. These insights are a necessary prerequisite for developing rehabilitation programs based on firm scientific principles, a basis lacking in most existing programs. This thesis will focus on the recovery of kinematic and spatio-temporal gait characteristics and their relation with functional recovery of walking ability after stroke. Furthermore, the specific effect of neglect on the walking trajectory of stroke patients will be investigated. In the thesis it will be attempted to answer the following questions regarding the gait of stroke patient.

- Is recovery of joint kinematics towards a normal pattern a requirement for functional recovery of walking ability?
- Is symmetry of gait a requirement for functional recovery of walking ability?
- Can compensatory gait patterns be detected, and if so, do they facilitate functional recovery of walking?
- If compensatory gait patterns develop in time, can it be predicted in an early stage *which* patients develop these patterns?
- What is the effect of neglect on the walking trajectory of stroke patients?

To be able to quantify asymmetries in gait while subjects walk on the floor, instead of on a treadmill, it was necessary to develop a device that records the positions of the feet. From these positions spatial parameters such as step- and stride length and temporal parameters such as toe-off and heel-strike can be calculated. The design and validation of this device is presented in chapter 2 of this thesis. Chapter 3 and 4 show the results of a cohort study in which the recovery of joint kinematics and spatio-temporal characteristics in stroke patients is investigated during a period of 48 weeks post-stroke and related to the functional recovery of walking ability.

It is known that primary sensory systems such as proprioception and the vestibular system deteriorate with age. Since stroke patients are generally older people it is important to investigate to what extent the deteriorated proprioceptive and vestibular systems may contribute to errors in heading control, before the effect of neglect on heading is evaluated. Therefore, in chapter 5 the effect of ageing on the ability to maintain heading while prism glasses induce a heading error during walking is investigated and data are presented on how well older people are able to adapt to this visual distortion in comparison to younger subjects. Chapter 6 addresses the effect neglect has on the walking trajectory in stroke patients and why heading control may differ between different neglect patients.

A concluding discussion is presented in chapter 7. An attempt is made to answer the questions raised above. Clinical implications and possible implementations are discussed, and suggestions are made for future research.

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