# CHAPTER 7

## GENERAL DISCUSSION AND CONCLUSIONS

## GENERAL DISCUSSION

In this thesis it was attempted to answer several questions regarding the gait of stroke patients:

- 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?

In chapter three it was shown that knee, hip and pelvis kinematics do not need to recover to a normal pattern to regain functional levels of walking ability. Certainly, patients whose gait patterns had recovered to normal all showed functional recovery, however, some patients had developed gait patterns that deviated from normal patterns and still showed functional recovery. Neither is symmetry of gait a requirement for functional recovery of walking ability, as was shown in chapter four. Again, patients whose step length symmetry and single support duration symmetry had recovered to values within the normal range, all showed functional recovery, but several patients whose gait symmetry had not recovered to normal values showed functional recovery of walking ability as well.

These findings have implications for rehabilitation programs. Commonly used rehabilitation programs such as Neurodevelopmental Treatment (NDT, Bobath) are specifically focused on restoring normal gait with a symmetrical pattern.[1] One of the rationales upon which this goal is based, is that walking according to normal gait patterns is energy efficient and less effortful. This is certainly true for healthy subjects and normal gait patterns can be considered optimal for a normal system.[2,3] However, this does not necessarily mean that normal gait patterns are

optimal also for stroke patients. Indeed, in stroke patients structural changes have taken place in the central nervous system, which have changed the state of the system and the rules that generally apply to a normal system do not necessarily apply to a control system hit by a stroke. To quote Olney et al.: "… one would not expect a bilateral machine with motors of unequal power on each of its sides to produce an optimal solution by using equal outputs from those motors.".[4] The changed motor patterns, which may be far from perfect for a normal system, may reflect adaptations that fit with the altered state of the system. These adaptive changes are the result of the plasticity of the brain and therapies should not always and by definition be aimed at "correcting" these changes. Or as Latash et al. concluded: "If you see, in a clinical setting or in an experiment, a motor pattern that is very different from what is observed in unimpaired, control subjects, do not jump to the conclusion that it is a sign of inability of the CNS to behave correctly, which, as such, should be corrected.".[5]

The role of compensatory mechanisms after brain damage was already described by Luria[6] in the forties of the last century. He stressed the importance of processes such as functional reorganisation and functional adaptation. In his view a damaged brain is capable of restoring functions by reorganising intact parts of the brain, which will then take over the impaired functions. He argued that reorganisation of surviving structures is the main mechanism of recovery of function because neuronal structures in the cortex, once destroyed, do not regenerate. More recent imaging studies, investigating the plasticity of the brain[e.g. 7,8] have shown that some cortical areas are indeed capable of functional reorganisation and that training influences the speed of the reorganisation.

The self-reorganising capacity of the brain does, however, not necessarily need to result in compensatory motor behaviour and more specific, compensatory gait patterns. Reorganisation within the brain may very well lead to fully normal gait patterns; an assumption, which is supported by the fact that several stroke patients, who are initially impaired in walking ability, eventually recover to more or less normal gait patterns. Apparently, in these patients the brain showed sufficient remaining capacity to restore their gait to normal. However, when the remaining capacity of the brain is insufficient, functional reorganisation may

result in compensatory gait patterns. One could argue that these patterns reflect the most optimal solution the system is able to find given the fact that it is damaged. Therapists should, rather than moulding the movement patterns of these patients towards a normal pattern, attempt to teach and to assist these patients in using their adapted movement patterns to its fullest extent.

Obviously it will be very important to identify which patients would benefit from a more compensation-oriented therapy and which have the capacity to follow the therapeutic route toward the reacquisition of a normal gait pattern. Therefore, it is necessary to identify what compensatory gait patterns exist in chronic stroke patients and how they can be detected or predicted already in early post-stroke stages. In chapter three two compensatory gait patterns were identified: a stiff-knee gait and a pendulum movement of the pelvis that compensates for a reduced hip swing amplitude. In the study we showed that it was possible to identify the patients who would eventually develop a stiff-knee gait on the basis of the data of the first post-stroke gait analysis. Future research should determine whether this prognostic criterion is valid also for larger groups of patients. If so, a study could be performed on the efficacy of compensatory stiff-knee gait training.

In chapter six it was shown that neglect patients showed a larger lateral deviation in their walking trajectory when they walked towards a target compared to stroke patients without neglect or healthy control subjects. Based on Karnath's model on neglect,[9] in which neglect is explained by a systematic shift of the subjective body midline to the ipsilesional side, we expected neglect patients to show a deviation in their walking trajectory to the contralesional side. In order to align their subjective body midline, which is shifted to the ipsilesional side, with the target, patients need to rotate their body to the contralesional side, which will introduce a heading error. In accordance with the findings in chapter five, this would result in a curvilinear path deviating to the contralesional side. This expectation appeared true for half of the patients. It appeared that the direction of the deviation was strongly related to the walking ability of the neglect patients. Neglect patients with good walking ability showed a deviation to the contralesional side; whereas neglect patients with poor walking ability showed a deviation to the ipsilesional side. We argued that when a neglect patient's

walking ability is impaired, walking towards a target may actually be seen as a dual task: heading control *and* walking. An impaired walking ability will cause a higher task priority of walking compared to heading control. Consequently, patients will, instead of actively maintaining the correct heading, apply a strategy of walking straight ahead. The displacement of the subjective body midline to the ipsilesional side causes the feeling of "straight ahead" to be shifted to the ipsilesional side. Therefore, walking straight ahead will cause the patient to diverge to the ipsilesional side, only occasionally adjusting their heading since heading control has become a secondary task.

A therapy that is specifically aimed at restoring the shifted subjective body midline in neglect patients is the prism adaptation treatment (PA-treatment).[10] During a PA-treatment patients perform a pointing task with their ipsilesional hand for several minutes while prism glasses induce a visual shift to the ipsilesional side. The prismatic shift initially causes a pointing error toward the ipsilesional side. However, within a few pointing movements patients correct this pointing error by shifting their pointing movement to the contralesional side. This causes a misalignment between the visual and proprioceptive representation of space; that is, patients see the target right in front of them but they feel that they are pointing towards the contralesional side. After several minutes the misalignment extinguishes, a process known as realignment of the proprioceptive and visual representation of space.[11] Patients now experience the pointing movement towards the contralesional side as pointing straight ahead. After prism glasses are removed the realignment causes a pointing error to the contralesional side, an effect know as the prism adaptation after-effect. In neglect patients this after-effect can be accompanied by amelioration of their neglect and while the after-effect diminishes within several minutes the amelioration of neglect may last several days to months.[12]

If the lateral deviation of the walking trajectory in neglect patients is indeed caused by the shift of the subjective body midline then improvement of neglect by means of PA-treatment should decrease the lateral deviation. An experiment in which the effect of PA-treatment on neglect and walking trajectories is investigated should learn us more about the value of the proposed model. A

prospective study in which the walking trajectory of stroke patients is recorded at several times could further investigate the theory regarding the effect walking ability has on the walking trajectory in neglect patients. If a neglect patient, whose walking ability initially is impaired, shows recovery of walking ability then, according to the theory, the deviation in the walking trajectory should shift from the ipsilesional side to the contralesional side. A third interesting experiment would be to introduce an attention-demanding task in neglect patients with recovered walking ability during walking. It is expected that when task priority of heading control is lowered by means of introducing a second, attention-demanding task, a more "walking straight ahead" strategy will be applied as it is the case in neglect patients with impaired walking ability. This would cause the walking trajectory to shift from the contralesional side to the ipsilesional side.

Optic flow theories have long been the major explaining theories in heading control. Optic flow refers to the radial patterns of light, generated by self-motion, which can be used to deduce heading. The concept of optic flow was introduced by Gibson[13,14] who argued that the region of the optic flow field that is not moving, the focus of expansion, is used to maintain the correct heading. However, in recent studies[15,16] the need for optic flow in heading control is challenged and it is argued that maintaining the correct heading during walking is based upon the perception of target location in relation to body orientation rather than upon the use of optic flow. Analogue to Rushton's findings,[15] the findings in chapter five and six support the idea of egocentric direction control; optic flow theories are unable to explain the curvilinear walking trajectory in subjects wearing prism glasses and neglect patients who misperceive their subjective body midline.

It was shown in several brain imaging studies[17,18] that MT/V5/V5a in the human brain is the area involved in the extraction of optic flow and, therefore, believed to play a major role in heading control. The idea of egocentric direction control indicates, however, that other brain areas are involved in heading control. In a recent study[19] Karnath challenged the general assumption that cortical lesions in the inferior parietal lobule (IPL) and the temporo-parieto-occipital (TPO) junction

were associated with unilateral neglect.[eg 20,21] According to the study, lesions in a region along the right mid superior temporal gyrus (STG) were critically associated with neglect. Karnath argued that, because the STG is located between the dorsal and ventral visual processing pathways and receives input from both routes, it may serve an important role in spatial exploration and be involved in the transformation process that converts sensory input coordinates from the periphery into an egocentric, body centred coordinate system. However, Mort et al.[22] showed that in a group of 14 middle cerebral artery neglect patients only 7 had lesions in the STG. The critical area in this patient group involved the angular gyrus and the IPL. Apparently, the STG is not a critical area for the presence of neglect. But if the STG *is* involved in the construction of an egocentric, body centred coordinate system, which is a crucial aspect in egocentric heading control, then the presence or absence of lesions in the STG may be an alternative explanation for the differences we found in heading control in neglect patients. It may also be the case that when lesions are present in the STG, neglect patients will show an impaired heading control, while neglect patients in which the STG area is spared, are able to maintain a proper heading. A study in which walking trajectories of neglect patients are recorded combined with detailed information regarding the lesion locations of these patients should answer this question. Furthermore, a brain imaging study in which a task is employed that requires activation of the mechanisms involved in egocentric direction control, may show if the STG is indeed involved in this process in healthy subjects. Crucial in such a task would be that subjects somehow receive feedback regarding (parts of) their body orientation in relation to perceived target location.

## CONCLUSIONS

To return to the questions that were raised in the beginning of this thesis we may conclude the following. Neither the recovery of joint kinematics towards a normal pattern nor symmetry of gait is a requirement for functional recovery of walking ability after stroke. Compensatory gait patterns can indeed be detected

and it appears that they are not by definition harmful but may facilitate functional recovery, although this needs to be investigated further. It also is possible to detect in an early stage which patients will develop compensatory gait patterns; at least for a stiff-knee gait pattern. Regarding the effect neglect has on the walking trajectory of stroke patients, we may conclude that when neglect patients actively maintain their heading, while walking towards a target, they will deviate to the contralesional side. However, when heading control changes to a "walking straight ahead" strategy this deviation shifts to the ipsilesional side.

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