

# **THE ASSESSMENT OF TRUNK MOTOR CONTROL IN HEALTH AND AFTER STROKE**

By

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## **DECLARATION**

I, Martina Mockova confirm that the work presented in this thesis is my own. Where information has been derived from other sources, I confirm that this has been indicated in the thesis.

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## **ABSTRACT**

This study investigated the static and dynamic function of trunk muscles in sitting after stroke. A new apparatus using a novel force-transducing system was developed that enables to study the time-course of development of trunk force magnitude and direction in sitting with or without the pelvis stabilised. In addition, reaction forces between the buttocks and the seat and between each foot and the ground are measured with three independent force plates.

24 healthy subjects were required to exert maximal forces in eight directions, with and without pelvis fixation. Accuracy of the trunk force measurement system was assessed without pelvis fixation by comparing trunk forces with reaction forces from the force plates. The agreement analysis showed a mean bias of the new system of only  $-3.0$  N for force magnitude and  $0.72$  degree for force direction. The trunk force measurement system was sensitive enough to detect effects of movement direction and pelvis fixation on maximum force magnitude. When subjects repeated the test at a later date there were no significant differences between the two sessions.

Trunk muscle strength in voluntary movements was then investigated in 9 stroke patients and 23 controls. Reaction forces were simultaneously recorded at the points of contact of the lower body. This is the first study that demonstrated the relationship between trunk and lower body movements affected by stroke. This was achieved by studying how the forces are transferred from trunk to pelvis and legs during trunk movements. This approach has shown that stroke impairs both prime mover and spine stabilizer trunk muscle function which is further compounded by weakness of pelvis muscles resulting in impairment of co-ordination between trunk and pelvis and deficient stabilization of the whole axis.

These results contribute to our understanding of the physiological mechanisms that affect trunk movement and control after stroke.

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## **LIST OF ABBREVIATIONS**

ADL activities of daily living  
ANOVA Analysis of variance  
BBS Berg Balance Scale Fugl-Meyer balance test  
BG basal ganglia  
BL backwards left  
BR backwards right  
CMAP Compound Muscle Activated Potential  
CODA contactless measurement system  
COG Centre of gravity  
COP Centre of pressure  
CSA cross-sectional area  
CT Computed tomography  
EMG electromyography  
EO external oblique  
ES erector spinae  
F frontal  
FIM Functional Independence Measure  
FL forwards left  
FR forwards right  
GBS Guillan-Barré syndrome  
GPi globus pallidus interna  
HV haptic vertical  
IC insular cortex  
IRED infrared emitting diode  
L left  
LD latissimus dorsi  
MAS Motor Assessment Scale  
MCA middle cerebral artery  
MEP motor evoked potentials  
MRI magnetic resonance imaging  
MRP Motor Relearning Programme

NHP Nottingham Health Profile  
NIHHS National Institutes of Health Stroke Scale  
P parietal  
PASS Postural Assessment Scale for Stroke  
PD Parkinson's disease  
PPN pedunculo pontine nucleus  
PV postural vertical  
R right  
RA rectus abdominis  
RASP Rivermead Assessment of Somatosensory Performance  
SD standard deviation  
SEM standard error of the mean  
SHV subjective haptic vertical  
SMA supplementary motor area  
SMES Sødring Motor Evaluation Scale  
SNr substantia nigra  
SPV subjective postural vertical  
STN subthalamic nucleus  
SVV subjective visual vertical  
TCT Trunk Control Test  
TIS Trunk Impairment Scale  
TMS transcranial magnetic stimulation  
TPJ temporoparietal junction  
UPDRS Unified Parkinson's Disease Rating Scale  
VV visual vertical

## **CHAPTER 1: Introduction**

### **1.1 Overview**

This work investigated the normal static and dynamic function of trunk muscles in sitting, in health and after stroke. To do this, an apparatus was designed to measure trunk muscle strength and assess how this relates to trunk motor control and sitting balance. It can be used in normal man and in the context of disorders which impair trunk muscle strength, including stroke, at one time point and over time, and to assess the effects of interventions, including physiotherapy treatments, designed to improve trunk muscle strength, trunk control and sitting balance.

This chapter first addresses some aspects of the neuroanatomical and physiological organization of postural control in man, and how this control may be disordered after stroke. Next, the overall impact of stroke, the ways in which it may affect trunk control and sitting balance, how these motor skills can be assessed, and the importance and predictive value of trunk control and sitting balance after stroke, are reviewed. Lastly, a number of neurophysiotherapy treatment approaches and treatments will be described, and some theoretical questions that underpin these interventions discussed, including the mechanisms of their effect, their appropriate application and the assessment of their effectiveness.

### **1.2 Perception and neural representation of posture and postural control**

#### **1.2.1 Perception of verticality**

In order to understand posture and postural control it is important to understand how gravity is perceived. Healthy subjects are able to perceive their subjective vertical orientation very precisely. Visual, vestibular and proprioceptive systems are required for the correct orientation of the head and body to gravity. It is, however,

not entirely clear how the systems contribute and interact (Anastasopoulos et al 1999). It is possible to measure the contribution of different sensory systems to the perception of the subjective vertical. To assess the subjective visual vertical (SVV), the subject indicates the orientation of an illuminated line in complete darkness to exclude visual cues. Healthy controls are able to set a line upright within  $1^{\circ}$ - $2^{\circ}$  of true gravitational vertical when sitting (Anastasopoulos 1999). When measuring the subjective haptic vertical (SHV), using somatosensory perception, the subject uses touch to adjust the orientation of a rod in the absence of visual cues. Normal limits are within  $-4.5^{\circ}$  to  $4.5^{\circ}$ . Subjective postural vertical (SPV) is assessed in subjects sitting in darkness on an inclining surface. Subjects indicate when they perceive their body to be vertical. Normal limits range from  $-2.5^{\circ}$  to  $2.5^{\circ}$  (Pérennou 2005).

The perception of body verticality is often altered in patients with central or peripheral nervous system damage. Stroke is commonly associated with misperception of the body's subjective vertical, which has implications for the patient's clinical impairment. Bonan et al (2006) found a positive correlation between an abnormal SVV and balance. 20 out of their 30 stroke patients had an abnormal SVV and the scores were linked to abnormal balance scores as measured by the Postural Assessment Scale for Stroke (PASS) and centre of pressure (COP) displacement on a rocking chair. In their further study, Bonan et al (2007) demonstrated that abnormal SVV is also related to poor balance recovery and functional outcome after stroke. 28 patients with stroke were assessed within 3 months of stroke, and 23 of them also at 6 months. Both abnormal SVV tilt and abnormal range of uncertainty (defined as the standard deviation of mean absolute SVV deviation) were related to PASS and FIM scores (Functional Independence Measure) at the initial assessment and at 6 months. The relationship to balance recovery was independent of other factors contributing to imbalance such as poor motricity and visuospatial neglect.

A subgroup of about 10% of patients with stroke exhibit pushing behaviour, and show a particularly striking impairment of the sense of verticality. These patients push actively away from the ipsilesional (non-paralysed) side and tilt, and may fall, to the opposite, affected, side, and resist passive correction. It is however not clear how they perceive their subjective vertical as studies on this subject reached

different conclusions. Karnath et al (2000a) investigated contraversive pushing in 5 pusher stroke patients and 10 non-pusher controls, of whom 5 had brain damage, 4 with spatial neglect and hemianesthesia, and 5 were healthy. The subjective postural vertical and subjective visual vertical were determined in each case. Controls showed no tilt of SPV or SVV with or without visual input. Pushers exhibited tilt of SPV to ipsilesional side without visual clues whilst with visual input their point of subjective postural and visual verticality did not differ significantly of that of the controls. The authors argued that the normalisation of the perception of vertical with visual input stemmed from visually aligning body axis earth-vertical with upright orientation of surrounding objects. That was proved in additional experiments. When the subjects wore Frenzel glasses and faced a white plane, their subjective verticality tilt was comparable with that of the condition without visual input. When sitting in darkness and tilted to their perceived upright, their SVV judgment was comparable with SVV judgment when sitting physically upright. That indicated undisturbed processing of visual and vestibular inputs for the determination of visual vertical. When the subjects sat objectively upright, they perceived a mismatch between visual vertical based on vestibular and visual inputs and tilted orientation of subjective body verticality. Hence the authors speculated that pushing is an attempt to actively compensate for the mismatch since pushing was not present when patients were tilted to their subjective upright. As an alternative they proposed that pushing is a secondary response to patient's perception that they lose balance when trying to sit upright. The cause could be misperception of body orientation in relation to gravity. Visual input alone is not sufficient in pushers to control upright posture. They can use it sufficiently in laboratory experiments but continue to tilt and push in daily life even in presence of abundant visual clues. It is still useful for rehabilitation which aims to teach patients to transfer weight to the unaffected side. Pushers can align their body axis correctly to earth vertical when aided by visual input from their surroundings but must learn to use this ability (Karnath et al 2002).

Normal processing of visual and vestibular signals in pusher patients was also found by Pérennou et al (2002) but, in contrast to Karnath, they observed a contralesional tilt of postural vertical. They investigated 8 healthy subjects and 14 right-hemispheric stroke victims, 3 of who were pushers. The subjects were asked to maintain an upright posture whilst sitting on a rocking platform. Pushers showed a



contralesional tilt of the pelvis but a correct orientation of head. The authors argued that pushing results from a disrupted processing of somesthetic graviceptive orientation which is important for trunk orientation. The disrupted processing in pusher patients leads to a biased perception of their subjective vertical which is tilted away from the cerebral lesion. Pushers then try to align their posture with this contralesionally tilted subjective vertical. Visual input contributed little to upright posture in sitting pusher patients although their pushing behaviour was more pronounced in darkness. They relied mainly on graviceptive information. Still, visual inputs that provide additional clues from the surrounding environment can compensate for postural problems that patients with stroke experience.

Lafosse et al (2004) presented a similar argument. They studied 43 right hemispheric stroke patients with or without neglect. They found an ipsilesional tilt of the SPV and of the centre of gravity (COG) in mild and moderate neglect patients whilst patients with severe neglect showed a contralesional COG displacement. The authors explained the tendency to adjust the COG towards the contralesional side as an effort to align their disturbed body orientation with the gravitational frame of reference relying mainly on the proprioceptive information. In their study from 2008, Pérennou et al explored the impairment of postural (PV), haptic (HV) and visual (VV) vertical in 86 stroke patients, 6 of whom were pushers. In contrast to Karnath's and Lafosse's results and in keeping with their 2002 study, they found again a contralesional tilt of the PV, HV or VV or of two or all three modalities in 74% of 80 patients with a hemispheric stroke. No patient with a hemispheric stroke showed an ipsilesional HV or PV tilt. 44 patients showed a contralesional VV tilt and 9 showed a mild ipsilesional VV tilt. 18 patients demonstrated a transmodal contralesional tilt (i.e. of PV and HV and VV). All of them except one had a right hemisphere stroke and a more extensive lesion. They all also showed a severe contralesional lateropulsion and six were pushers (including the one patient with a left hemispheric stroke). Mild ipsilesional VV tilts were described previously in a small proportion of subjects with a hemispheric stroke (Brandt 1994, Dieterich 1993). In this study, the patients with the ipsilesional VV tilts had contralesional PV tilts. The authors could not explain the non-congruency of the tilts and it remains a topic for future study. With regard to the discrepancy with the studies of Karnath and Lafosse, the authors argued that the differing results of direction of the

subjective vertical tilt could be explained by movements of the head and legs that were not restrained in the other studies and could have provided biased cues to verticality perception. Also, the timing of the studies could have been an issue. The patients in the Lafosse study were tested 4-33 months after their stroke as opposed to after about 8 weeks in the Pérennou study. The time of testing could have influenced the results as the patients tested later after the stroke onset might have been improving. Karnath tested the patients within three weeks of onset of their stroke. Pérennou argued that whilst a transient change in perceptual function is possible, one would expect it to occur in parallel with the pushing behaviour. Also, some patients in the Pérennou study were studied within two to five weeks after stroke onset but it did not influence the direction of the SPV tilt. Hence they felt that the time difference in testing was unlikely to explain the differing results.

Work on perception of verticality in stroke patients with pushing behaviour or neglect thus revealed opposing conclusions. Karnath found that pusher patients had their SPV tilted ipsilesionally whilst being actually tilted contralesionally through active pushing and felt that the pushing was a compensatory mechanism to accommodate the abnormally perceived vertical orientation. Pérennou and Lafosse found a contralesional tilt of SPV along with contralesional pushing and argued that it was caused by disrupted processing of graviceptive input necessary for trunk orientation. The importance of somatosensory input from the trunk for verticality perception was demonstrated in several studies. This would support the results of Pérennou and Lafosse showing a contralesional tilt of the SPV in pushers. Mazibrada et al (2008) studied 1 patient with a nearly complete loss of peripheral sensation due to Guillan-Barré syndrome (GBS) and two patients with a thoracic spinal injury resulting in a sensory loss from T6-7 down. The subjects were assessed whilst seated. They were tilted to either left or right and immediately rolled back to the vertical position. They indicated when they started feeling upright and again when they started feeling tilted again (the entry and exit points indicated the cone of verticality). The GBS patient had a significant SPV bias towards the side of the preceding tilt in both directions and enlarged cone of verticality. Whilst his SPV bias resolved on retesting along with marked improvement in his limbs and trunk sensation, the enlargement of the cone of verticality persisted. The spinal injury patients did not have a significant SPV bias. The authors reckoned that the results

were due to the fact that the spinal injury patients had preserved somatosensory input from the upper trunk and shoulders which was important in the seated position. Afferent input from the lower limbs was relatively unimportant. Similar was suggested by Mittelstaedt (1998). He found no direct contribution of somatosensory input from legs to the perception of posture. He felt that leg proprioceptors were likely to only modulate the output of trunk graviceptors. Anastasopoulos et al (1999) found that variability of the SVV setting in a patient with left-sided hemihypaesthesia resulting from an infarct of the right thalamus increased considerably when lying sideways on her hypaesthetic side. They thought that somatosensory input increases the accuracy of the orientation of the body to the gravitational vector by providing additional information to that obtained from the visual input.

### **1.2.2 Neural representation of postural control**

It is not entirely clear how the perception of postural and visual vertical is represented in the brain. Mittelstaedt (1998) postulated that there are two different input sources of graviceptive information. Information about the visual vertical is obtained exclusively from the sensors in the head and neck whilst the posture of the trunk is sensed through unknown graviceptors in the trunk. He provided a proof of existence and localization of truncal graviception in experiments on the subjective horizontal posture on a tiltable board and a sled centrifuge. His subjects were able to set themselves horizontally on a tiltable board in a right-ear-down position with legs extended. When the same task was repeated on a sled centrifuge, normal subjects felt horizontal when the distance between the head and the rotation axis was below their head, i.e. negative. If the subjective horizontal posture was controlled by otoliths alone, they should feel tilted downwards. Subjects without vestibular function as a result of bilateral neurectomy then felt horizontal at even greater negative distance. Thus the distance found in normal subjects appeared to be the result of a compromise between the effect of otoliths and a trunk graviceptor. As candidates he proposed afferent input from the kidneys through the renal nerve and the inertia of the mass of blood in the large vessels or the mass of the abdominal viscera.

Others suggested two different pathways serving the perceptions of visual and postural verticals. Bisdorff et al (1996) investigated perception of body verticality in patients with various acute and chronic peripheral and central vestibular lesions. They did not find any significant directional bias of SPV in any patient group, neither was there any consistent tilt of the SPV after optokinetic stimulation in healthy controls. However, there was a significant ipsilesional tilt of SVV in patients who underwent neurectomy for Ménière's disease. In view of this dissociation the authors assumed that there would be different pathways to subserve these two perceptions. Karnath et al (2000) found in pusher patients that they had a severe ipsilesional tilt of the SPV with a normal perception of the SVV. The finding of a double dissociation led them to believe that there were indeed two different pathways, one that projects to the vestibular cortex and a separate pathway for sensing body orientation in relation to gravity. In order to identify this brain region, Karnath et al (2000b) examined magnetic resonance imaging (MRI) scans of infarcts in 23 patients with severe pushing and found that an overlapping area of the infarctions projected in the posterolateral thalamus. They speculated that the ventral and posterior and lateral nuclei of the posterolateral thalamus and possibly its cortical projections formed the neural representation of a second graviceptive system in humans. Pérennou et al (2008) found that postural vertical was more biased in strokes affecting the right hemisphere and that the area involved was located around the primary somatosensory cortex and thalamus. The temporoparietal junction (TPJ) of the sensory cortex was shown to be pivotal for the control of the body stability by Pérennou et al (2000). Of their 22 stroke patients tested with the rocking platform paradigm (trying to maintain an upright sitting posture whilst looking at a fixation point for 8 s), only those with the TPJ lesion had a marked increase in aborted trials and increased angular dispersion. Brandt et al (1994) identified posterior insula as the lesion that regularly affected the perception of subjective visual vertical in their stroke patients. They postulated that this area corresponded to the vestibular cortex found in the monkey. Johannsen et al (2006) studied 45 stroke patients with and without contraversive pushing with lesions sparing the thalamus. They too showed posterior insula, the postcentral gyrus and surrounding white matter to be the cortical areas contributing to processing the signals providing information about vertical body orientation.

Some argue that the right hemisphere is predominantly responsible for postural control. Pérennou et al (1999) assessed postural performance in 50 stroke patients (25 with right and 25 with left hemispheric lesion) as measured by Fugl-Myer scale modified by Lindmark and Hamrin. They found a lower performance in patients with right hemispheric damage. Rode et al (1997) evaluated postural sway in standing in 30 stroke patients (15 with right and 15 with left hemispheric lesion). Patients showed a significant ipsilesional shift of the centre of pressure which was more predominant in the left hemiparetic patients. Bonan et al (2007) found a higher frequency and of abnormal range of uncertainty but not of abnormal SVV tilt in patients with right hemispheric lesion than with left hemispheric lesion (71% vs 14%). The range of uncertainty was higher in patients with lesions in the temporoparietal junction and temporal cortex.

The tilt of SVV caused by brainstem vascular accidents depends on the site of the lesion. Dieterich et al (1993) found ipsilesional SVV tilt and ocular torsion in patients with stroke caudal to the upper pons and contralesional SVV tilt and ocular torsion in strokes rostral to this level.

The function of the cerebellum and its connections in postural control is well known. Functionally, cerebellum is divided into three parts, the vestibulocerebellum, spinocerebellum and the pontocerebellum. From the postural control view, the interesting parts are the first two. The vestibulocerebellum consists of the flocculonodular node with the adjacent areas of the vermis. It is phylogenetically the oldest cerebellar module and it exerts control over the orientation of the head and body in space via its afferents from the vestibular ganglion, the olivary nuclei and the basal pons and efferents to the vestibular nuclei, the fastigial nucleus and the lateral thalamus and from there to the trunk areas of the motor cortex. It controls balance through the descending vestibulospinal tracts. Damage to the flocculonodular node results in inability to stand steadily and to maintain balance when walking. The spinocerebellum consists of the vermis and the intermediate parts of the cerebellar hemispheres. It maintains the body posture against gravity by controlling the tone of axial muscles. It receives afferents from the spinocerebellar tracts and the accessory olivary nuclei and projects into the fastigial and interposed nuclei and to cerebral cortex via the thalamus (Greenstein et al 2000).

Basal ganglia also play an important role in balance control. The basal ganglia connections with brainstem nuclei are recognized to contribute to postural control (Visser et al 2005). There are only few studies documenting balance problems due to vascular basal ganglia lesion. Labadie et al (1989) described 9 patients with unilateral pallidal or putaminal haemorrhage or lacunar infarct who exhibited severe postural deficits. The patients fell in opposite direction from the lesion in a slow tilting motion whilst sitting, standing or walking. They were initially not aware of their propensity for falls which distinguished their postural instability from that caused by a cortical or cerebellar lesion. Later, having become aware of their impairment, they were not able to perform corrective movements fully. Masdeu et al (1994) presented a patient with gait failure consisting of inability to perform regular steps of coherent direction in absence of leg weakness. She had suffered a haemorrhage at the pontomesencephalic junction involving the right pedunclopontine area. The pedunclopontine nucleus (PPN) has, beside other brain structures, extensive connections to the basal ganglia. Its efferents connect to various thalamic nuclei, the subthalamic nucleus (STN), globus pallidus internus (GPi), ventral tegmental area, striatum and substantia nigra (SNr). The afferents project from the STN, GPi, SN, striatum and ventral tegmental area (Martinez-Gonzales et al, 2011). The dysfunctional projections via the pedunclopontine nucleus may account for the gait impairment.

Postural problems arising from degenerative disorders of basal ganglia and SNr have been studied more extensively. Martin (1965) described 14 patients with post-encephalitic parkinsonism. They developed lateral scoliosis usually accompanied by ipsilateral head tilt in absence of spinal disease. 8 of his patients were inclined to the side with lesser rigidity, 4 to the side of greater rigidity and in 2 patients both sides of the body were equally affected by the parkinsonian signs. Crawling was tested in 1 patient who deviated towards the concavity of the scoliosis when crawling, more so with the eyes closed. This was similar to findings in dogs (Delmas-Marsalet 1925) who developed flexion of the body and head with the concavity to the side of caudate nucleus lesion. They also circled to the side of the concavity. 3 of Martin's patients underwent unilateral pallidotomy. Operation ipsilateral to the concavity of the scoliosis relieved the postural deficit but failed to do so when it was contralateral to the concavity. Martin suggested that the scoliosis found in parkinsonian patients

was due to unbalanced activity of the caudate. Duvoisin et al (1975) confirmed that scoliosis is common in parkinsonism and its concavity correlates with the laterality of its symptoms and signs. They presented 21 patients with Parkinson's disease (PD) and post-encephalitic parkinsonism. 16 developed scoliosis contralateral to the side of initial parkinsonian symptoms and 3 had ipsilateral scoliosis. In 1 patient an initial contralateral scoliosis was replaced by an ipsilateral scoliosis with the progression of the disease. Several of the patients also deviated towards the concavity of their scoliosis when crawling. The patients' scoliosis also increased with the eyes closed indicating possible defective orientation of the body in space. Since the structure most affected in PD and post-encephalitic parkinsonism is SNr and since animal tests showed that lesions targeting nigrostriatal dopaminergic projections produce curvature of the body and head and circling towards the lesion, the authors concluded that it is a lesion of SNr that produces the postural deficit in PD. Stereotactic functional neurosurgery provides additional information on posture in PD. Su et al (2002) reported two patients who developed head and body tilt contralateral to unilateral subthalamotomy that later corrected itself with a contralateral procedure. Since previous reports have shown scoliosis towards dopamine-deficient hemisphere, the authors speculated that the tilt observed in their patients was due to dopaminergic imbalance between hemispheres. The operated side exerted more dopaminergic influence as the procedure interrupted inhibitory efferents from GPi and SNr. Two studies used gait analysis and posturography to assess the influence of bilateral GPi stimulation on gait and posture in PD. Defebvre et al (2002) studied 7 patients before and 3 months after the procedure. The gait analysis revealed improved stride and step length and reduced double support time off medication. Postural instability analysed with a lateral leg raising task showed improved preparatory postural adjustment parameters. Volkmann et al (1998) demonstrated increased gait velocity and cadence and reduced step time off medication on gait analysis of 9 patients 3 months after bilateral GPi stimulation. Unilateral ventroposterior pallidotomy also improved posture in PD patients as shown in two studies that used posturography to measure standing balance. Masterman et al (1998) measured static (with and without foam) and dynamic (on a moving platform) sway in 18 patients on medication. Their results showed a trend towards less sway in all conditions although the only significant result was in the foam test condition. Mandybur et al (1999) tested 14 patients off medication. Static and dynamic sway

and fall rate decreased in the patients' group after the procedure. Meyer (1997) demonstrated that ventroposterior pallidotomy improved gait and trunk movements in 26 patients. The subjects were assessed with Unified Parkinson's Disease Rating Scale (UPDRS) Motor Examination and 25 standardized motor tasks. The tests showed best results for standing and walking and tasks involving trunk movements.

### **1.3 Definition, epidemiology and impact of stroke**

Stroke is defined as a rapidly developing episode of focal or global loss of cerebral function with symptoms, thought to be of vascular origin and lasting more than 24 h or leading to death. The main pathological types of stroke are ischaemic stroke, primary intracerebral haemorrhage, and subarachnoid haemorrhage. It is the third most common cause of death in the adult population (Ledingham et al 2000), and affects approximately 100,000 people a year in Britain. 30-60 % of survivors fail to recover completely and remain dependent to some degree (Smith et al 1999).

Mortality and morbidity associated with stroke presents a considerable financial burden. Evers et al (2004) reviewed 25 stroke economic cost studies from 8 developed countries including the UK. They found that the cost amounted to approximately 3% of total health expenditure. In the first year after stroke an average of 76% of the cost is spent on inpatient care. These studies largely did not take into consideration the financial burden incurred by the patient, family and other informal caregivers which can be significant. According to a study by Saka et al (2005), stroke costs about £7 billion to the economy in England annually, direct costs (providing acute and long-term health care to stroke sufferers) accounting for 40% of this total, informal care costs for 35% and productivity losses for 25%.

### **1.4 Impairment of trunk control and sitting balance after stroke**

In sitting, the pelvis and posterior thighs form the primary base of support, with additional stability provided by the feet in contact with the floor. In absence of spinal motion, the axis of anteroposterior movement rotates around the greater



trochanter, and forward/backward leans are achieved via pelvis and trunk movement. Anterior pelvic tilt with upper trunk extension allows forward reaching and begins the sit-to-stand transition. Lateral weight shifts with trunk rotation permit cross-midline reaching and begin the sit-to-supine progression. The use of the arms to prop in sitting is an extension of the base of support (Umphred 1995). Anti-gravity control in unsupported sitting is provided mainly through extensor activity at the pelvis, hips and lumbar spine (Edwards 2002).

Trunk control is necessary in order to change the body position, to control movements against gravity and to shift the weight to free the limbs for function. In sitting, normal trunk control maintains the stability of trunk and enables us to shift weight and balance and to reach with the arms. In the patient with stroke, poor trunk control results in poor sitting and standing balance and loss of ability to perform functional activities. Different levels of trunk control can be impaired, including the ability to achieve upright posture, to perform trunk movements, to adapt the trunk to arm movements and to generate power in the trunk and upper limbs (Umphred 1995).

After stroke, balance reactions can be inhibited, delayed or completely absent depending on the severity of the impairment. The unaffected side often lacks the ability to right itself, partially due to the inability of the affected side to bear weight. Also, the patient is often reluctant to carry weight on the affected side. This can be due to spasticity that prevents the automatic reactions from functioning. In the hemiparetic patient, muscle weakness, abnormal muscle tone, loss of trunk control and of the ability to coordinate trunk and limb movements result in development of abnormal postural reflexes (Bobath 1989). Trunk position sense is also impaired as shown by Ryerson et al (2008) in stroke patients who have a higher trunk repositioning error during forward flexion in sitting, an error which correlates negatively with the Berg Balance Score and PASS.

## **1.5 Clinical assessment of balance and postural control**

Evaluation of a patient with balance and postural control problems includes the

history and clinical examination to determine specific motor, sensory and cognitive deficits that impair balance control; functional balance scales; and quantitative posturography.

There are a number of functional balance assessment scales but the most commonly used in stroke patients are the Berg Balance Scale (BBS), the Postural Assessment Scale for Stroke Patients (PASS), the Fugl-Meyer balance test and the Trunk Control Test (TCT). The Berg Balance Scale, developed by Berg, uses 14 items rated from 0 to 4. It was originally developed to measure balance in the elderly and is not specifically dedicated to stroke patients (Berg et al 1995). PASS is aimed specifically at stroke patients. It was derived from the Fugl-Meyer assessment of balance and mobility but differs from it in that 2 items, which evaluate the postural response to a non-calibrated perturbation (examiner related), have been removed. PASS scans 3 fundamental postures: lying, sitting, and standing. It contains 12 items (Benaim et al 1999). The Fugl-Meyer balance test, described by Fugl-Meyer et al in 1975, consists of seven variables, three for sitting balance and four for standing balance, with a maximum score of 14. TCT examines the maintenance of the sitting position, the ability to roll from a supine position towards the affected and unaffected sides, and the transfer from supine to sitting position (Franchignoni et al 1997). All these assessment scales have been validated and shown to have a good test-retest and inter-rater reliability, internal consistency and excellent validity. The BBS and the Fugl-Meyer balance test do have a floor and a ceiling effect though. Hence they may not be suitable for patients who are initially either very mildly or very severely affected as they may not detect meaningful changes in these patients. It may be useful to use these balance measures in conjunction with the PASS as it does not show a significant floor or ceiling effect. TCT is a short and simple test and is useful in situations where trunk-specific balance impairments need to be assessed (Blum 2008, Franchignoni 1997).

Quantitative posturography uses a force-measuring platform to analyse stance, sitting, reactions to surface displacements, voluntary movement and gait. The parameters commonly measured are the standard deviation of the horizontal ground reaction force, the standard deviation of the centre of pressure, the mean velocity of the centre of pressure, the horizontal acceleration of centre of mass and the standard

deviation of the vertical ground reaction force (Karlsson et al 2000). It provides a detailed and objective analysis of postural responses, and is a useful research tool to investigate the pathophysiology of balance disorders. It is widely used clinically to assess patients with instability and falls, and to evaluate physiotherapeutic treatment of balance problems of various aetiologies (Visser et al 2008).

### **1.6 The importance of trunk control in the prediction of functional recovery after stroke**

In view of the high incidence of stroke and its wide-ranging and long-lasting impact, the ability to predict outcome is of paramount importance. Sitting balance and trunk control are well recognized to be reliable predictors of functional recovery in subacute stroke sufferers, since they are necessary for the performance of a number of activities of daily living including eating, dressing, transferring and walking.

Thus trunk control, measured by the Trunk Control Test (TCT) at admission to a rehabilitation unit, accounted for 71% of the variance of the motor component of the Functional Independence Measure (FIM) at discharge 3 to 10 weeks post-stroke (Franchignoni et al, 1997). Hsieh et al (2002) analyzed the relationship between trunk control, as assessed by trunk control items of the Postural Assessment Scale for Stroke Patients (PASS-TC) and the Fugl-Meyer balance test, at 14 days after stroke and functional outcome measures, consisting of the Barthel Index and the Frenchay Activities Index, at 6 month after stroke. They found a strong positive correlation between trunk control and functional outcome after stroke with PASS-TC having the highest power to predict the scores in activities of daily living. Feigin et al (1996) demonstrated a correlation between sitting balance during the first 3 weeks after stroke and gait at 6 months. Sandin et al (1990) found a strong positive correlation between weekly sitting balance score and the Barthel Index score at weeks 4 and 12 during rehabilitation. Recently, Di Monaco et al (2010) examined the correlation between trunk control in sitting at 20-22 weeks post-stroke, as assessed by the Trunk Impairment Scale (TIS) and balance as assessed by the PASS, and functional independence at discharge, as measured by FIM and discharge

destination (home or an institution), in 68 stroke patients. Both TIS and PASS scores correlated with the FIM score at discharge, change in FIM score during rehabilitation and destination at discharge. Similarly, after traumatic brain injury, Black et al (2000) showed in a cohort of 237 cases that the predictive power of sitting balance was exceeded only by age when measuring the Discharge FIM-Total score and the selected elements from the Discharge FIM-Motor score that relied most on sitting or standing balance.

## **1.7 Physiotherapy of balance**

Despite recent advances in thrombolytic therapy for acute ischaemic stroke physiotherapy still plays the main part of stroke treatment. There is evidence that rehabilitation is beneficial in stroke treatment. Little else in rehabilitation of stroke has a solid evidence base. This section outlines current evidence on physiotherapy in stroke and commonly used approaches. Also, there is no consistent means of assessing the effectiveness of commonly used rehabilitation interventions for treatment of impaired sitting balance and trunk control. This study proposes a new method of assessing trunk function after physiotherapy that would help expand the incomplete evidence base for stroke rehabilitation.

Langhorne et al (1996) examined seven randomised trials involving 597 stroke patients. They found that more intensive physiotherapy input was associated with a reduction in the combined outcome of death or deterioration and may enhance the rate of recovery. Kwakkel et al (1997) studied the effects of different intensities of rehabilitation in nine controlled studies involving 1051 stroke patients and found a small but statistically significant intensity-effect relationship in the outcome. Similar was reported by De Wit et al (2007). 532 stroke patients were followed in four European rehabilitation centres in the UK, Germany, Switzerland and Belgium. Whilst the content of therapy was similar in all centres, the daily therapy time showed an average of 1 hour in the UK, 2 hours in Belgium, 2 hours 20 minutes in Germany and 2 hours 46 minutes in Switzerland. Differences in therapy time were not attributable to difference in patients x staff ratio but the proportion of time spent on direct patient care was the highest in Germany and the lowest in the UK. Patients

in the Swiss and German centres achieved a significantly better motor and functional recovery when measured by the Rivermead Motor Assessment of Gross Function scale and the Nottingham Extended Activities of Daily Living scale. Patients in the British centre achieved a better personal care recovery as measured by Barthel Index.

Restoration of sitting balance and good trunk control is one of the main goals in stroke rehabilitation. The emphasis is on correcting alignment of body segments with a normal base of support during the performance of tasks, teaching the patient to make appropriate adjustments of posture during movement or displacement of any segment of the body, and retraining of balance in sitting and standing (Carr et al 1987). A number of different overall approaches are used, most commonly the Bobath and Movement science (motor relearning) approaches, incorporating a variety of interventions including exercise-based interventions, task-related relearning feedback training and constraint-induced movement therapy.

### **1.7.1 The Bobath approach**

The Bobath based approach is the method most commonly practised in the UK and in Europe. It is used by approximately 88% of physiotherapists in the UK (Davidson et al 2000), although understanding of the concept is rather diverse and the application tends to vary widely among the therapists. Despite having been practiced for over 50 years now, a debate concerning the underlying theory and effectiveness of the method is still ongoing.

The Bobath concept was devised in 1943 to re-educate children with cerebral palsy. It is based on the observations of Bertha Bobath, a physiotherapist. She noticed that it was possible to influence the tone and movement patterns of these children with specific manual handling techniques. Dr Karl Bobath then developed the theoretical framework based on a hierarchical model of the central nervous system (CNS). The main problems of the hemiplegic patient were considered to be abnormal coordination of movement patterns combined with abnormal posture tone caused by neurophysiological dysfunction; the muscle strength deficit was seen as

secondary. Great emphasis was placed on the role of postural reflexes. They were thought to be developmentally primitive. Re-education through the developmental sequence was applied to children with cerebral palsy and adults with stroke. The last updated theory saw postural reflexes more as postural reactions that are essential to support movement. The main interventions serve to inhibit abnormal tone and to facilitate automatic postural reactions and normal movement. It is achieved by handling the patient manually at key points (trunk, shoulder girdle, pelvis, knee) to manipulate mainly proprioceptive inputs in order to change the patterns of spasticity and guide recovery of function. The patient participates on an automatic or a volitional basis. The therapist works on patient's ability to recover sitting and standing balance against gravity (Bobath 1989).

Some of the assumptions the concept is based on are no longer compatible with current scientific knowledge. Much criticism of the method concerns its reliance on the hierarchical model of motor control over other concepts to account for movement dysfunction, failure to integrate principles of motor learning into its framework, too much emphasis on abnormal tone, and opposition to muscle strength training (Lennon 1996, Mayston 2000).

Incorporation of new theories into the Bobath concept has been advocated since the eighties. Systems model for motor control, as opposed to the neurodevelopmental theory as described in the writings of the Bobaths, was offered as an alternative explanation of functioning of the central nervous system. Kershner (1981) described the systems theory as a model where the organism is a circular network of interacting yet autonomous subsystems, rather than a vertical structure of descending controls. Bobath's hierarchical model of the CNS is a unidirectional model in which postural and voluntary motion become two separate and distinct entities. Bly (1991) also interpreted the Bobath theory in view of the systems control model of the CNS. Motor programmes were proposed to explain how automatic postural adjustments occurred in anticipation of movement. She recommended that new motor programmes were facilitated in therapy and recognised that spasticity could be a compensatory strategy. Neuroplasticity as the main rationale underlying Bobath's concept for treatment of brain damage has been suggested by Valvano et al (1991). Also, Bobath therapy focuses on preparing and practicing components of movement in order to improve tone and re-educate normal movement patterns. This

fails to acknowledge that the CNS is task oriented in its organization (Flament et al 1993), therefore, practicing functional tasks in the correct context may achieve the same and help acquire new skills, which is in keeping with the theory of motor learning. Motor learning was defined by Schmidt (1988) as a set of processes associated with practice or experience leading to relatively permanent changes in the capability for responding. As patients practice a task in a variety of situations and also outside therapy, they improve their understanding of the relationship between a movement outcome and their control of the movement's parameters. Lastly, Miller et al (1997) and Brown et al (1998) showed that strength training is beneficial in stroke patients and does not increase spasticity, in contrast to the Bobaths' suggestion.

### **1.7.2 Trials examining the effectiveness of different physiotherapeutic approaches**

The Bobath technique remains the most popular one although it has not been proven conclusively to be better than other methods or indeed effective. A meta-analysis that looked at studies concerning the effectiveness of the Bobath approach was carried out by Paci (2003). He evaluated 15 clinical trials involving a total of 726 subjects. Only 6 of them were randomised controlled trials, 6 were non-randomised controlled trials and 3 were case series. Results showed no definitive proof of effectiveness of the Bobath method nor did they suggest it to be the optimal type of treatment, however, methodological limitations of the studies did not allow for conclusion of non-efficacy either.

Of note is the trial by Langhammer et al (2000) which compared outcomes of Bobath and Motor Relearning Programme (MRP) methods administered to groups of 28 and 33 patients, respectively. Importantly, the sample size was determined by power calculation, the trial used a blind assessor and, with regard to this thesis, the outcome measures included functions of postural control. The groups were evaluated in the acute stage of their stroke and at three months follow-up. The main outcome measures were the Motor Assessment Scale (MAS), the Sødring Motor Evaluation Scale (SMES), the Barthel Index and the Nottingham Health Profile (NHP) which assesses the quality of life. MAS is a test of motor function and measures eight

activities such as turning in bed, sitting, standing up, walking, balance in sitting and arm function. SMES, unlike MAS, measures only the unassisted performance of the patient. It has three subscales, measuring leg function (SMES 1), arm function (SMES 2) and functions concerning trunk, balance and gait (SMES 3). Barthel Index tests activities of daily living. The authors found that the Motor Relearning Programme group improved more than the Bobath group both in MAS and SMES 2, in bladder and bowel function items of the Barthel Index and had a shorter hospital stay. However, in their follow-up study (2003) at one and four years after stroke the authors found that the initial physiotherapy programmes did not result in a significant difference in long-term motor function and disability. In view of the insufficient evidence, Van Vliet et al (2005) randomised 120 patients into their comparison of efficacy of the two methods. The primary outcome measures were the Rivermead Motor Assessment and the Motor Assessment Scale (MAS). The secondary outcome measures evaluated functional ability, sensory impairment and cognitive impairment. As in the previous study, the sample size was determined by power calculation and the assessments were completed by a blind assessor at baseline and at 1, 3 and 6 months. In contrast to Langhammer's study, the trial showed equivalent outcome and length of hospital stay in both groups. The main differences between the studies that may explain the discrepancy were early and more intensive treatment in the previous study and also possible differences in the content of treatments since the latter trial used more recent publications on the therapeutic methods.

The most up-to-date meta-analysis by Pollock et al (2007) still failed to determine whether any one physiotherapeutic approach was superior in facilitating recovery of leg strength or balance following stroke than any other approach. They reviewed 21 randomised or quasi-randomised trials, 5 of which were included in two comparisons. Treatment interventions were included if they focused on recovery of postural control, leg function or general functional ability; treatments that concentrated on arm function recovery were excluded. 8 of the trials compared neurophysiological approach with another approach; 8 compared a motor learning approach with another approach; and 8 compared a mixed approach with another approach. They found limited evidence that rehabilitation using a mixture of different approaches was significantly more effective than no treatment or placebo



for improving functional independence following stroke. Van Peppen et al (2004) conducted a metaanalysis of 151 controlled studies to determine the evidence for physical therapy interventions in stroke treatment. Task oriented exercise training was found to have strong evidence for restoration of balance and gait, for strengthening the leg and for improving symmetry when moving from sitting to standing. Interventions such as training of the arm with constraint-induced movement therapy, treadmill training, aerobics, external auditory rhythms during gait and neuromuscular stimulation for glenohumeral subluxation also appear to have strong evidence.

Similarly, Langhorne et al (2009) found that interventions that focus on high intensity and repetitive task-specific practice aid motor recovery. Thus arm function improved with constraint-induced movement therapy, electromyographic (EMG) biofeedback, mental practice with motor imagery and robotics. Transfers and balance improved with repetitive task training, biofeedback and training with a moving platform. Walking speed increased with fitness training, high-intensity physiotherapy and repetitive task training.

### **1.7.3 Retraining of seated weight distribution**

Another contentious issue in physiotherapy of balance is the problem of weight distribution. Ability to distribute weight evenly and to shift it as needed during tasks is important for normal balance. Healthy people load symmetrically at the point of body contact with a support both in sitting and standing (Drummond 1982, Sackley 1991). Stroke patients, on the contrary, bear more weight through the stronger leg in standing (Sackley 1991, Goldie 1996). It is less conclusive whether the same happens in sitting.

Au-Yeung (2003) tried to answer this question. She investigated buttock-seat interface loading in healthy and stroke subjects sitting without back or feet support on a seat pressure measurement system device. Stroke patients tended to load to the side contralateral to their hemiplegia. Manually guided shifting exercise regime had no effect on their loading asymmetry. Nichols et al (1996) used a force plate system

to measure the vertical force in symmetrical sitting and leaning to either side and forward in stroke victims. On longitudinal follow-up they found that only the lean forward task showed improvement over time. In addition, lean forward and to the paretic side correlated with Functional Independence Measure (FIM) on discharge. However, they did not record the feet forces and so it was unclear how much they contributed to the results. Kerr et al (2002) addressed that issue in their study of healthy elderly volunteers. They looked at the effect of foot support and the direction of trunk movement on the net centre of pressure (COP) derived from forceplates under the buttocks and each foot. The forward lean with foot support showed the greatest stability presumably because the feet enabled the subjects to extend their base of support. They also found that COP was reliable and clinically useful in forward and lateral leans but not in backward lean. They felt that the reason for that might have been the fact that leaning backward was a relatively unfamiliar task for the subjects. Yelnik et al (2006) investigated the effect of visual input on still sitting balance. The measure used was frontal plane displacement of the centre of pressure (COP) and total length of centre of pressure displacement under optokinetic stimulation in stroke patients and controls. The patients showed more dependency on visual input, on the whole, although individual reactions were more important than mean group reactions. Also, the visual dependence did not originate exclusively from the neurological impairment, implying that premorbid physiological behaviour might have an influence. Another study that explored the effect of visual deprivation on quiet sitting was conducted by van Nes et al (2008). Three consecutive assessments six weeks apart were evaluated. COP velocities were recorded in sitting with eyes open and closed, on both a stable and unstable support. They found that lateral balance control was more affected by stroke, more so during visual deprivation. It improved the most on subsequent testing and showed the strongest correlation with clinical impairment as measured by the Berg Balance Scale. Importantly, unstable support was necessary to obtain significant effects. It might therefore be effective to use unstable support in rehabilitation. Dean et al (1997) suggested in their experiment with 20 patients that early training to improve sitting balance which involves emphasis on appropriate loading of the affected leg while practicing reaching tasks prepares patients for standing. In view of this evidence Mudie et al (2002) attempted to determine which of three different treatment approaches was the most effective in retraining of seated weight distribution. They

compared task-related reach, Bobath and feedback training techniques with a control group that received standard physiotherapy and occupational therapy. Their group sizes were small containing only ten subjects each which meant that their findings had to be interpreted with caution. They found that all groups including the control group showed improvement in weight redistribution. The biggest effect was seen in the Bobath group at two weeks follow-up but the improvement was maintained only by a smaller number of subjects at twelve weeks. The group using the feedback also improved significantly but continued to improve long-term. Also, this was the only group where improvements in seated weight distribution translated into significantly improved standing symmetry. In contrast, the short-term benefits gained by the control group seemed to reverse long-term. Interestingly, task-specific reaching group failed to change seated weight distribution significantly, unlike in the Dean's study. The subjects in Dean's study were given performance feedback which might have influenced the results, though. Howe et al (2004) carried out a trial with 17 patients examining the effect of training aimed at improving lateral weight transference in sitting and standing. The treatment group received additional physiotherapy based on the work of Davies which loosely derives from neurophysiological principles and incorporates elements of motor learning. The results did not show any significant differences in lateral weight displacement in sitting or standing at four and eight weeks follow-up.

## **1.8 Conclusions**

Despite the well recognized importance of recovery of sitting balance after stroke, the current knowledge of postural control of balance in general, and trunk control and sitting balance impairment in stroke in particular, remains incomplete and inconsistent.

Evidence shows that physiotherapy is an effective treatment for stroke, but there is still a lack of agreement on which rehabilitation techniques should be used in balance re-training after stroke (Dean et al 1997, Harrison 1995), how much physiotherapy should be given, and whether physiotherapy should target weight distribution in sitting and use trunk muscle strengthening exercises.

Good trunk muscle strength seems a logical prerequisite for good trunk control. However, the interplay between trunk muscle strength and trunk motor control in health and after stroke is not well understood. The studies presented in this thesis aim to address this issue.

## **CHAPTER 2: A method for quantifying directional strength and motor control of the trunk**

### **2.1. Introduction**

Good trunk control is necessary to balance the body against gravity, to perform whole-body voluntary movements, and to allow the upper limbs to move freely from a stable base. It is impaired in a number of neurological conditions and its recovery can reliably predict functional outcome. However, trunk function is difficult to measure and quantify. Thus the pathophysiology of trunk motor control following neurological damage is not well understood. Furthermore, the physiotherapeutic treatment of impaired trunk control lacks effective tools to measure the efficacy of any intervention techniques that are used.

An important component of trunk control is the ability to activate the appropriate muscles either to accelerate the trunk, or to resist external forces, in any direction. This is a complicated motor task in free sitting or standing in that it entails transfer of forces from the upper body via the lower body to its contact with a support surface. Thus inadequate trunk control can arise from upper or lower body deficits or a problem in coordinating the two.

Currently available methods for measuring trunk muscle activation have a number of limitations. Bohannon et al (1995) used a hand-held dynamometer to investigate trunk muscle strength in sitting after stroke. High inter-rater reliability can be obtained with this method, but its validity is operator dependent because: 1) accuracy of measurement can be compromised if operator strength is low relative to the forces being measured (Bohannon 1999); 2) the accuracy of the direction of movement is subjective and therefore difficult to reproduce; 3) the validity of the data is compromised if the point of force application differs between measurements (Delitto 1990). Essendrop et al (2001) tested the reliability of isometric strain gauge dynamometer commonly used in occupational settings. They showed a modest but significant increase in back extension and back flexion force measurement from test to retest in their group of 19 healthy volunteers. The test was reliable on a group

level but a large variability was found on individual level. Isokinetic dynamometers have been used in evaluation of patients with low back pain and for studying stroke patients (Tanaka 1998). However, several technical problems may be encountered including adjustment for gravity, patient stabilization, placement of the axis of movement and reliability of measurements of lateral flexion of trunk. There is also little information on the relationship between isokinetic measures and physical impairment (Newton *et al* 1993). Both of these techniques test only the static component of trunk force development, are limited in the spatial aspect of trunk control, and are not suitable for evaluation of the contribution of the lower body to trunk control. These deficiencies argue for a new approach to the measurement of trunk motor control.

In this chapter we describe an apparatus that overcomes these problems. Our primary objectives were to develop a system that would: (1) evaluate voluntary trunk activation in multiple directions while seated to allow measurement of trunk strength, spatial accuracy and movement dynamics; (2) enable stabilisation of the subject's pelvis for the investigation of trunk control with and without the need for coordination with the lower body; (3) provide reliable test-retest measurements for application to longitudinal studies. Our apparatus incorporates a novel force-transducing system to measure upper-body quasi-isometric forces and a system of force plates to measure the lower-body reaction forces at the three contact surfaces of the feet and buttocks.

## **2.2. Methods**

### **2.2.1 The apparatus**

The frame (Fig. 2.1) is constructed from square-section metal bars and is 1.5 m high, 1 m wide and 1 m deep. The seat rests on a construction of four horizontal bars. The frame stands on screw-in legs that allow the height of the seat to be adjusted. The frame is easily accessible. The front top bar can be pivoted upwards to enable better access to the seat.

The seat is made of 100 cm wide and 60 cm deep wooden board with a 60 by 40 cm force plate (Kistler type 9286A, Kistler Instrumente AG, CH-8408 Winterthur, Switzerland) embedded in it to measure the reaction force between the buttocks and the seat. Its top is covered by 10 mm thick foam. Three 5 cm wide length-adjustable belts are attached to the frame and seat. They can be strapped around the subject's pelvis and across the thighs in order to stabilise the lower body and fix the pelvis to the seat. One belt passes from the back of the seat and around the front of the pelvis to prevent forward sliding. Another passes from the front framework and around the back of the pelvis to prevent backward sliding. The third passes over the upper thighs and is anchored on either side of the seat to prevent lifting.

The platform is positioned over ground force plates so that the subject's feet rest on separate force plates (Kistler types 9281B (left leg) and 9287 (right leg), Kistler Instrumente AG, CH-8408 Winterthur, Switzerland). These plates measure the reaction forces generated between each foot and the ground.

The system for transducing trunk forces comprises 4 force transducers, each anchored to a corner of the frame via steel wires. The transducers consist of strain gauges mounted on custom-built metal rings that are attached via length-adjustable steel wires to a harness worn by the subject. The harness has pass-thru buckles on the chest strap and the shoulder straps for easy adjustment. Each wire is attached to a loop on the chest strap; there are a sufficient number of loops to allow the wire to be connected at different positions depending on the subject's size. The corner anchors for the transducer wires can slide vertically in the metal bar for adjustment. One of the features of this force transducing system is that each of the four transducers can be attached anywhere to the subject and orientated at any angle with respect to the frame. A 3-D contactless measurement system (CODA) is used to measure the 3-D position in space of each transducer using pairs of infrared emitting diode (IRED) markers attached to the ends of each transducer, collinear with the transducer axis.



**Figure 2.1.** The apparatus in use.



### 2.2.2 Trunk force vector calculation

The force direction of each transducer was measured from the position of the markers placed on them (Figs. 2.1 and 2.2). The direction unit vector  $\mathbf{d}_i$  ( $i = 1,2,3,4$ ) of each transducer was calculated using the equation:

$$\mathbf{d}_i = \mathbf{P}_{i2} - \mathbf{P}_{i1} / \|\mathbf{P}_{i2} - \mathbf{P}_{i1}\|$$

where  $\mathbf{P}_{i1}$  and  $\mathbf{P}_{i2}$  are the magnitudes of the marker coordinates and bold denotes vector.

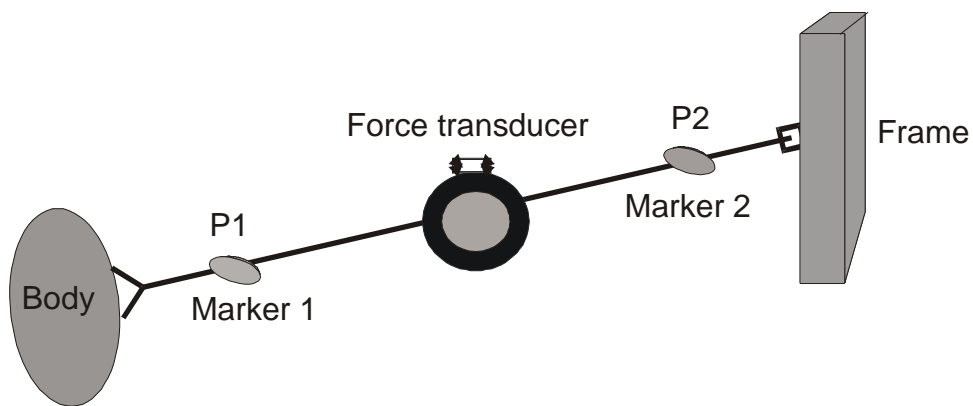


Figure 2.2. Diagram of a force transducer with the markers indicating its position in space.

The force vector,  $\mathbf{F}_i$ , from each transducer was obtained by multiplying the unit direction vector  $\mathbf{d}_i$  by the transducer output voltage,  $f_i$ , and a calibration constant,  $k_i$  (N/V).

$$\mathbf{F}_i = f_i k_i \mathbf{d}_i$$

The resultant trunk force vector,  $\mathbf{F}$ , was then obtained by vector summation

$$\mathbf{F} = \mathbf{F}_1 + \mathbf{F}_2 + \mathbf{F}_3 + \mathbf{F}_4.$$

### 2.2.2.1 Magnitude and direction of force in the horizontal plane

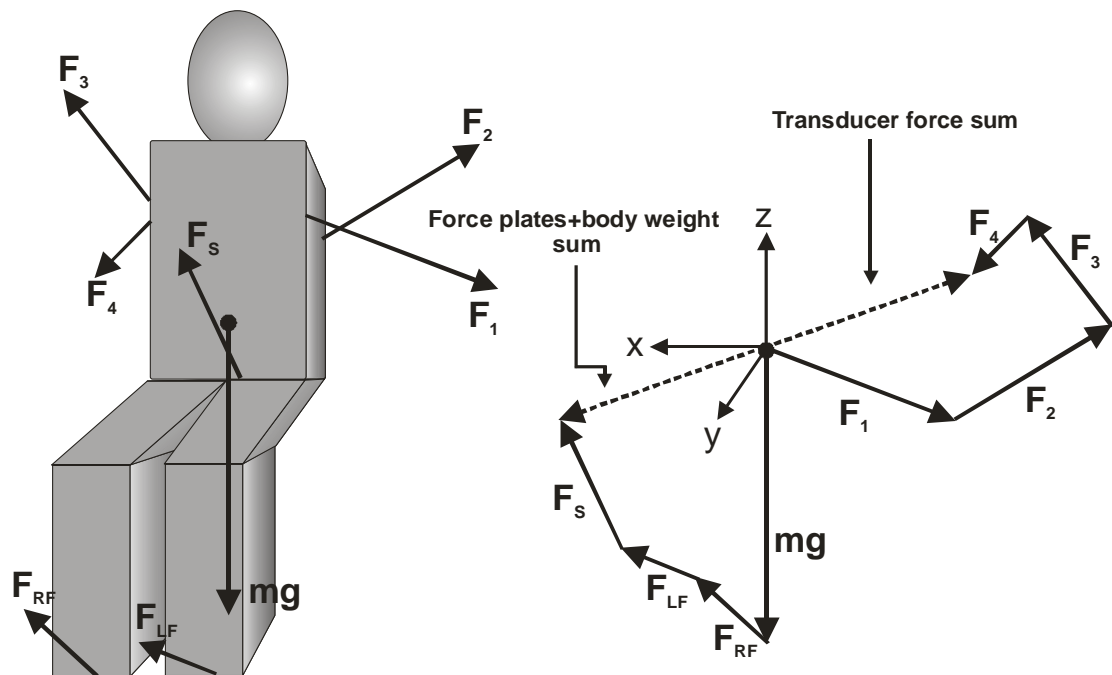
The magnitude,  $F$ , of the trunk force vector in the horizontal plane was calculated as:

$$F = \sqrt{F_x^2 + F_y^2}$$

where  $F_x$  and  $F_y$  are the magnitudes of the anteroposterior and mediolateral components of the resultant force vector  $\mathbf{F}$ .

The angle,  $\theta$ , of the force direction in the horizontal plane was calculated as:

$$\theta = \arctan(F_y / F_x)$$



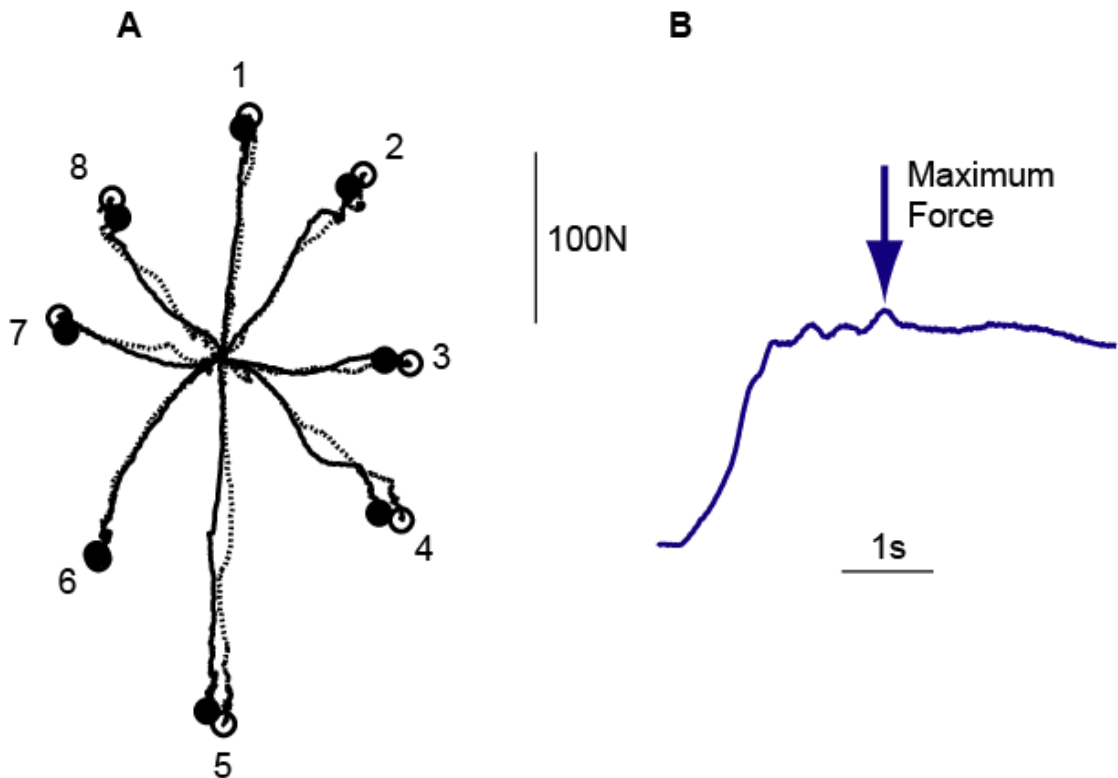
**Figure 2.3.** Forces acting on the body.  $\mathbf{F}_{1-4}$  indicate trunk forces from each transducer.  $\mathbf{F}_S$ ,  $\mathbf{F}_{LF}$  and  $\mathbf{F}_{RF}$  denote seat, left foot and right foot force respectively.  $Mg$  is body weight. Force vector diagram is shown on right of figure. Dashed lines denote the resultant force from the transducers and the resultant force from the force plates plus body weight.

### **2.2.3 Subjects**

Experiments were performed on 24 healthy volunteers (9 males, 15 females; age 44-80 years, mean 61.4, SD 8.2 years) with ethics committee approval. 18 of the 24 subjects were studied twice in a 6 to 8 week interval. 1 subject participated only in the without-pelvis-fixation condition. In 1 subject the force plate recordings were corrupted and therefore did not contribute to the analysis of agreement between methods. Subjects participated with informed consent and the approval of the local ethics committee according to the guidelines of the Declaration of Helsinki. In both experiments practice trials were given and rests were provided after every 8 trials.

### **2.2.4 Experimental procedure**

Subjects were instructed to move their trunk with maximum effort in each of eight different directions whilst sitting on the seat. The four taut steel wires passing from the subject's harness to the frame corners minimized actual body movement, although there was always a small amount of displacement due to elasticity of the body-harness-wires-frame arrangement and because the lower trunk was not restrained. Therefore, we refer to the test as quasi-isometric. The directions were forwards (1), backwards (5), laterally to the left (7) and right (3) and diagonally to the front left (8), front right (2), rear left (6) and rear right (4) (Fig 2.4A). A computer program chose the direction pseudo-randomly for each trial. The subject was told the direction and instructed to pull with maximum effort in the given direction at the sound of a tone and to relax at the sound of a second tone. The programmed sequence was started by the experimenter via a handheld switch that triggered data collection. Data were sampled at 100 Hz and the total recording time was 6s with the audible tones occurring 1s and 6s after the start of a trial. Each set of eight movement directions was repeated three times.



**Figure 2.4.** Typical raw horizontal force trajectories from single trials of one subject. A. Trunk forces (solid lines) and reaction forces (dotted lines) in each of the eight directions. Trunk and reaction forces are plotted in the same direction to illustrate agreement between the two. Circles show maximum force magnitude. B. The development of trunk force magnitude with time for the same subject in direction 1.

#### 2.2.4.1 Validating the method

We assessed the accuracy and validity of our new method of trunk force measurement by comparing its estimate of the trunk force vector with the reaction force vector measured by the three force plates. In accordance with Newton's third law, when the body is stationary and without pelvis fixation straps, the trunk force, reaction force and body weight should sum to zero (Fig. 2.3). If we consider only the components of force in the horizontal plane it is clear that the trunk horizontal force should be equal and opposite to the horizontal ground and seat reaction force.

#### **2.2.4.2 Effect of pelvis fixation**

To assess the effect of active lower body stabilization on the forces developed by the trunk, the experimental procedure was repeated with the pelvis passively fixed to the platform by the seat straps (see apparatus).

#### **2.2.4.3 Test-retest reproducibility**

In order to test the stability over time of our trunk force measurement method, we looked at the reproducibility of results on two measurements separated in time. Eighteen subjects were tested on two separate occasions, 6-8 weeks apart.

#### **2.2.5 Data and statistical analysis**

Data were stored on-line and converted to text files for analysis. Calculations were performed off-line using Matlab (The MathWorks Inc, Natick, Massachusetts). Measurements were made of force magnitude and direction at the time of maximum force development (Fig. 2.4B) in single trials. All analyses were performed on the mean values of the three trials performed in each direction for each condition. For mean force direction, the angle of the force direction was calculated for each trial and then averaged over the 3 trials using the circular statistics procedures according to Batschelet (1981).

We assessed the accuracy of the magnitude and the direction of the trunk horizontal force using the Altman-Bland method (1999) in which the agreement between trunk force measured by the force-transducing system – the “new” method - and reaction force, measured by the force plates – the “gold- standard” method - are expressed as bias plots. This was done separately for force magnitude and force direction using data only from the no-fixation condition.

The effects of movement direction, pelvis fixation and retesting on force parameters were assessed using repeated measures ANOVA design (SPSS version 11). Post hoc analysis was performed using either Tukey's test or two-tailed paired samples t-test as appropriate. Inter-session reliability of the method was assessed using the Pearson correlation coefficient on paired (session 1 versus session 2) data of each subject's mean force magnitudes averaged across movement direction. Significance was taken as  $p < 0.05$  for all tests.

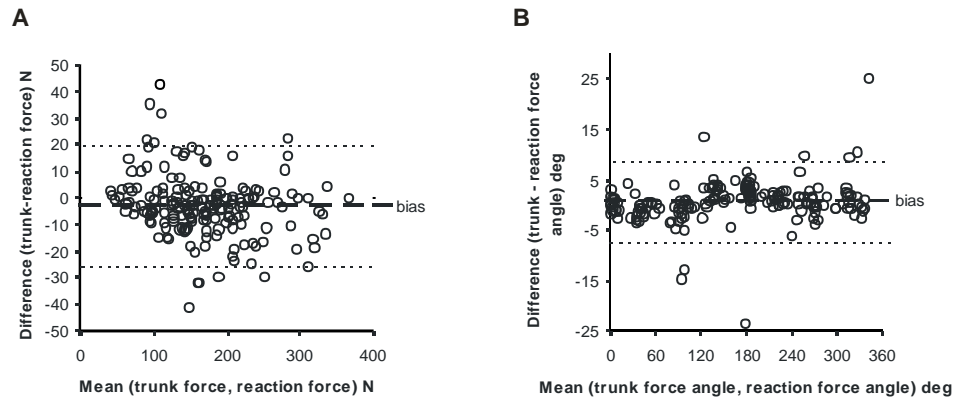
### **2.3. Results**

Subjects were able to comply with the instructions and generate forces in the directions specified. Typical force trajectories in each of the 8 directions are shown for one subject in Fig. 2.4A. The solid lines show the trunk force measurements and the dashed lines show the reaction force measurements from the force plates. The circles in Fig. 2.4A denote the maximum forces achieved in the eight directions. The development of force magnitude with time is shown in Fig. 2.4B for movement in one of these directions. The vertical arrow indicates the time of maximum force development.

#### **2.3.1 Validation of trunk force measurements**

Using the Altman-Bland method, the bias between trunk force magnitude and reaction force magnitude was -3.0 N (95% CI -4.7 to -1.3) with a lower limit of agreement of -25.6 N (95% CI -28.5 to -22.8) and an upper limit of agreement of 19.7 N (95% CI 16.8 to 22.5) (Fig. 2.5A).

Bias between trunk movement direction and reaction force direction was 0.72 deg (95% CI 0.13 to 1.31) with a lower limit of agreement of -7.18 deg (95% CI -8.18 to -6.19) and an upper limit of agreement of 8.63 deg (95% CI 7.63 to 9.62) (Fig. 2.5B).



**Figure 2.5.** Agreement bias plots. Each point represents mean data from one direction per subject. The limits of agreement are shown by the dotted lines and the bias is shown by the middle dashed line. A. Agreement between trunk force and reaction force magnitudes. B. Agreement between trunk force and reaction force directions.

### 2.3.2 Effect of movement direction on trunk force magnitude

Fig. 2.6A shows the group mean maximum trunk force generated in each of the eight directions, with and without pelvis fixation. We used 1-factor repeated measures ANOVA separately for the fixation and no-fixation conditions to determine whether the trunk force magnitude depended on the direction of trunk movement. There was a significant effect of movement direction on force magnitude both with and without pelvis fixation ( $F(1,7) = 3.2$  and  $2.8$  respectively;  $p < 0.05$ ). Post hoc analysis (Tukey's test) showed that subjects generated the biggest force in the backwards direction. The force exerted in direction 5 with pelvis fixation was significantly greater than for directions 2, 3 and 8 ( $p < 0.05$ ) and borderline for direction 7 ( $p = 0.052$ ). Similarly without pelvis fixation, the force in the backwards direction 5 was significantly larger than in directions 3, 7 and 8 ( $p < 0.05$ ) and borderline for direction 2 ( $p = 0.054$ ).

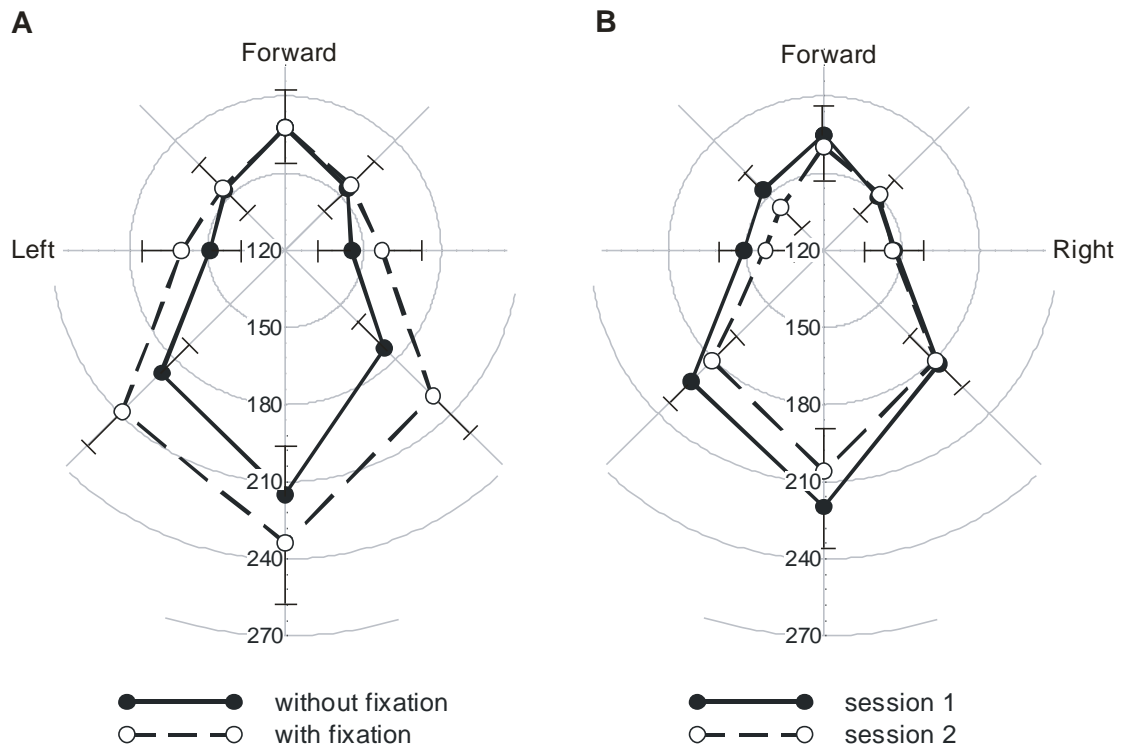
### 2.3.3 Effect of pelvis fixation on trunk force magnitude

To examine the effect of pelvis fixation we used a 2-factor (factors: *direction* and *fixation*) repeated measures ANOVA on the data of Fig. 2.6A. This revealed a significant *fixation* x *direction* interaction ( $F(7,154) = 3.6$ ;  $p < 0.05$ ) indicating that pelvis fixation influenced the maximum force depending upon movement direction. Post hoc analysis showed that the maximum trunk force generated was greater with pelvis fixation than without for the two backward diagonal directions 4 and 6 (two-tailed paired samples *t* test,  $t(22) = 2.9$  and  $2.7$  respectively;  $p < 0.05$ ).

### 2.3.4 Test-retest reproducibility

Fig. 2.6B shows the mean data obtained from the two sessions. Repeated measures ANOVA with three factors (factors: *repetition*, *fixation* and *direction*) showed no significant main effect of *repetition* ( $F(1,17) = 0.47$ ,  $p > 0.05$ ) and no significant interactions involving *repetition* (*repetition* x *fixation*  $F(1,17) = 1.27$ ,  $p > 0.05$ ; *repetition* x *direction*  $F(1,7) = 0.52$ ,  $p > 0.05$ ; *repetition* x *fixation* x *direction*  $F(1,7) = 0.14$ ,  $p > 0.05$ ). Correlation of data from session 1 with session 2 showed good inter-session reliability ( $r = 0.85$ ,  $p < 0.001$ ).





**Figure 2.6.** Polar plots of group mean force magnitude as a function of intended direction. Error bars denote standard error of the mean. A. Effect of movement direction and pelvis fixation. Forces obtained with the pelvis fixed to the seat using belts (dashed line) compared with the pelvis free (solid line). B. Test-retest reproducibility. Forces obtained in the first session (solid line) compared with a second session performed 6-8 weeks later (dashed line). Forces have been averaged across the 2 conditions of +/- pelvis fixation. Note that the radial arms denoting force magnitude start from 120N.

## 2.4. Discussion

The development of this new apparatus arose from the need for a reliable method to quantify trunk motor control and thus assess the effectiveness of therapeutic intervention in patients after neurological damage. From a practical viewpoint the apparatus is suitable for studying neurological patients; the platform is easily accessible and transfers of a subject from wheelchair to platform are simple. The subject's safety is maintained throughout testing by the support provided by the wires that attach the harness to the frame via the force-transducing system.

The accuracy of our method of trunk force measurement seems good. The mean measurement error (bias) between the trunk force magnitude and reaction force

magnitude was -3.0 N. This means that the trunk force-transducing system method slightly underestimates the force measured by the force plates. The spatial accuracy measurement agreement was close with a bias of 0.72 degrees. These apparent inaccuracies could arise from inaccuracies in the calibration of each transducer; the transducers were calibrated on a force equivalent to 200 N whereas trunk forces could reach around 350 N. Another source of inaccuracy could be a failure to completely eradicate the voltage offsets that were present in each force transducer output. A final source of error lies in the measurement of the orientation of each transducer in 3-d space. However, it should be pointed out that the accuracy of the force plates, our “gold-standard” method, is also limited, being of the order of  $\pm 1.5\%$ .

The limits of agreement for trunk force measurement between the two methods were -25.6 to 19.7 N which means that we can reliably detect asymmetries in trunk force generation above about 10% of the measured force. There are no data in the literature to indicate whether this is acceptably sensitive, but it seems likely that the asymmetry in trunk force would have to be more than 10% to be clinically important and worth targeting with specific physiotherapeutic techniques.

The sensitivity of the apparatus was good in that it revealed direction-specific effects on trunk control as well as the effects of lower-body stabilisation that were consistent across our sample of healthy subjects. In addition, the measurements were stable since similar results were obtained from the same subjects when they were retested at a later date. These findings indicate that the apparatus should be valuable for detecting even subtle impairments in a subject’s trunk control and to quantify change in those impairments over time.

The effect of direction on maximum force magnitude presumably reflects differences in strength between the major muscle groups of the trunk. For example, the trunk extensor muscles typically are stronger than the flexors, hence backward forces tend to be greater than forward forces. The effects of pelvis fixation are more complex. Without fixation the forces developed at the top of the trunk have to be balanced at joints lower down, for example between the trunk and the pelvis, in order for the force to be transmitted to the seat surface. Even if the pelvis and lower spine can be adequately actively stabilised there could be further stabilisation

problems if the trunk horizontal forces overcome the limiting frictional force between the bottom and the seat. This could occur if the coefficient of friction between the clothing and the seat materials were not high enough or if the bottom were to partially rise off the seat, for example through inappropriate pelvis stabilisation or leg action. Then the leg muscles must be used to stabilise the leg joints to transmit part or all of the reaction force to the floor. If the leg muscles can be adequately activated to resist these forces then the limiting factor would be the frictional force between the feet and the floor. Thus, the motor control problem is greater and potentially involves the coordination of many parts of the body when the pelvis is free to move. For the healthy subjects and materials used in the present experiments, the pelvis fixation made a difference only for movements in the backward diagonal directions.

The force plates, which measured the reaction forces between the body and the ground, were necessary for the present study because they provided the gold standard against which the new system could be compared. It is clear that the trunk measuring system, or perhaps even a simplified version of it, could be used without the expensive force plates and provide most of the information required to assess trunk motor control. However, the force plates do provide some extra information. For example, without the pelvis fixation belts the force plates show how the reaction forces are distributed at the three points of contact of the lower body (feet and buttocks), which potentially could illuminate difficulties of coordination between the upper and lower body. With the fixation belts, which theoretically are capable of providing all the required reaction force, the force plates measure unnecessary “overflow” of motor activity that may represent pathological motor behaviour.

Overall, the results suggest that the apparatus satisfies our three primary aims. First, it provides a tool for measuring a seated subject’s ability to voluntarily activate trunk muscles in multiple directions over the whole workspace. Although we have concentrated on measures of maximum force magnitude and direction there is potential for extracting other parameters of temporal and spatial aspects of motor control. For example, measurements could be made of the time to achieve peak force, or the force path and its variability. Second, with simple fixation devices we are able to investigate trunk behaviour with and without the requirement for lower

body coordination. Third, because of the consistent test-retest performance, the apparatus is suitable for longitudinal studies that investigate changes in motor behaviour over time, perhaps as a result of physiotherapeutic intervention or drug treatment. In conclusion, our new method is a reliable tool for studying trunk motor control and should prove useful for investigating the impact of neurological damage and recovery on that control process.

## **CHAPTER 3. Trunk motor control after acute right hemispheric stroke**

### **3.1. Introduction**

Sitting and standing balance problems occur commonly after acute stroke and can persevere as a chronic deficit. Trunk comprises a major part of the body mass which explains why good trunk control is essential when maintaining balance. It is not entirely clear to which extent trunk muscle weakness, lower body weakness or disrupted coordination between the two contribute to balance disturbance in stroke patients. The effects of a unilateral neurological insult such as stroke on trunk function are harder to understand and quantify since the strength and function of trunk muscles could be damaged on both sides of the body due to their bihemispheric innervation.

This was observed by Beevor in his seminal article from 1909. He described a patient with left-sided hemiparesis two months after the onset of his stroke resulting from an anterior circulation infarct secondary to syphilitic arteritis. The strength of the lateral movements of his trunk in sitting with his thighs fixed by an assistant was measured. The starting positions tested were a straight vertical and an inclination at 45 degrees. A traction dynamometer was used and the timing with which the trunk muscles contracted was noted. The patient had difficulty moving the trunk from the extreme left to the mid-line but could move from the mid-line to extreme right against resistance and, conversely, he could adduct the spine from extreme right to the mid-line but struggled to abduct the spine from the mid-line to the left against resistance. Beevor interpreted this as the trunk muscles acting normally in movements with the trunk situated on the right side of midline (lateral flexion of the spine to the right and adduction from the right to the midline) and weakly in movements with the trunk situated on the left side of midline, on the side of patient's paralysis (lateral flexion of the spine to the left and adduction from the left to the midline). He also noted that flexion and extension movements showed no difference in strength of the rectus abdominis or erector spinae muscles, only that the muscles

of the right side started the movement sooner. He coupled this clinical observation with his and Horsley's experiments with electrical stimulation of primates's cortex and internal capsule that they performed in 1890 and concluded that the muscles of each side of trunk were represented in each opposite hemisphere and closely associated through the corpus callosum. While this view is no longer accepted, Beevor's strength of clinical observation and reasoning is timeless.

Experiments carried out by Carr et al (1994) showed that axial muscles are innervated from both hemispheres. They recorded surface electromyography (EMG) from the left and right diaphragm, rectus abdominis, masseter and upper limb muscles during voluntary contraction and EMG responses evoked by transcranial magnetic stimulation (TMS) from the same muscles. On cross-correlation of the EMG responses during voluntary contraction from each pair of muscles they obtained a central peak of EMG discharges from the diaphragm, rectus abdominis and masseter muscles but not from the upper limb muscles. That was suggestive of a common drive to the motoneurons from co-contracting muscle pairs but not to the co-activated muscle pairs of the arms. TMS of the dominant motor cortex whilst recording the response from the studied muscles evoked a response from both left and right axial muscles but only a contralateral response from the limb muscles. The bilateral axial muscles responses were consistent even when the stimulation site was moved laterally from the optimal point excluding the possibility of stimulus spread to the opposite hemisphere. The authors suggested that the bilateral innervation arises from a common drive from activity in corticospinal tract which is synchronized at the cortical level. Bilateral cortical drive to axial muscles was also proposed by Marsden et al (1999) who found low frequency coherence when measuring bilateral needle and surface EMG responses from paraspinal muscles but not from first dorsal interosseus muscles. Similarly, Tunstill et al (2001) recorded motor evoked potentials (MEP) from both left and right rectus abdominis muscle when applying TMS to the right cortex only which was suggestive of ipsilateral corticospinal innervation along with the contralateral innervation.

Given the complicated nature of trunk muscle innervation and action, it is not that surprising that data on trunk muscle strength and sitting balance post-stroke are scarce and somewhat contradictory. Bohannon et al (1995) found that trunk muscle

performance measured with hand-held dynamometer during forward and lateral trunk movements was impaired in stroke sufferers in forward and lateral flexion on both sides of the trunk, more so on the paretic side, and particularly in the forward flexion. Karatas et al (2004) measured trunk strength in flexion and extension with an isokinetic dynamometer and reported isokinetic weakness in both directions in patients and weaker trunk extension than flexion both in controls and patients. Tanaka et al (1997, 1999) investigated trunk muscle function in flexion, extension and rotation using an isokinetic dynamometer. They too reported isokinetic trunk weakness in all directions in patients and stronger trunk flexion than extension in both groups, however, they did not find any effect of direction in trunk rotation.

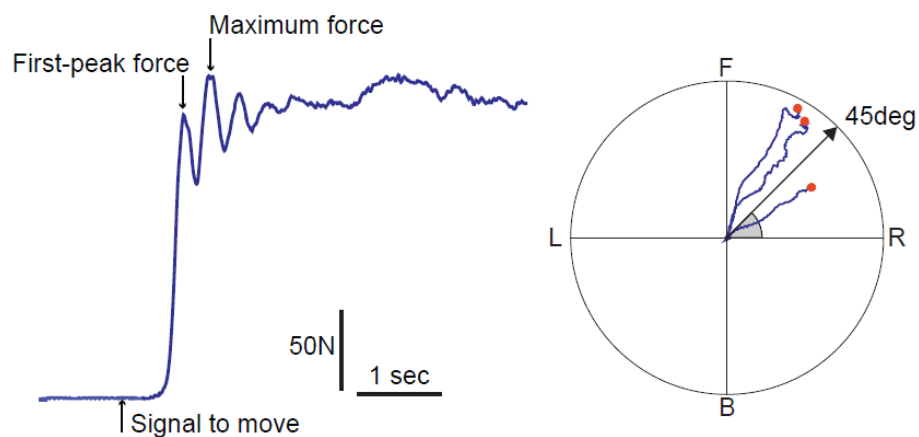
EMG studies on trunk muscle function post-stroke do not clarify the issue either. Whilst Dickstein et al (2004) did not find evidence to support the notion of unilateral erector spinae muscle deficit in hemiparetic patients despite finding reduced and delayed activity of the rectus abdominis and latissimus dorsi on the paretic side, Winzeler-Mercay et al (2002) recorded consistent asymmetrical activity in erector spinae and also in rectus abdominis in a dynamic task.

It is not clear how trunk muscle strength and trunk motor control are linked. The new method to examine the impairment of trunk muscle function in seated subjects described in the previous chapter was used to investigate acute stroke sufferers in order to expand our understanding of this issue. Trunk strength, trunk movement accuracy and movement dynamics were measured and the contribution of the lower body was manipulated by fixating the pelvis in order to answer the following questions: (i) How is trunk muscle strength impaired after stroke and is there a directional asymmetry? (ii) How does pelvis fixation influence trunk force? (iii) How does the lower body behave during trunk movements and is that influenced by pelvis fixation? (iv) Is the dynamic aspect of trunk force development affected by stroke? (v) Does stroke influence the accuracy of direction of trunk movement? and (vi) Is there any correlation between trunk force and clinical impairment?

## 3.2. Methods

### 3.2.1 First peak force magnitude calculation

The magnitude and direction of the trunk force vector in the horizontal plane was calculated as described in Chapter 2 Section 2.2.2.1. Therefore, an additional measure was assessed to control for the possibility that the maximum force was corrupted by a high degree of noise due to subjects trying to maintain the maximum force till the end of the trial. The force plots were evaluated visually and the time point at which the subject reached the first peak of force was established. The force magnitude at this time point was then calculated using Matlab (The MathWorks Inc., Natick, Massachusetts) and called first peak force (Fig. 3.1). The outcome measures did not show any significant difference between maximum and 1<sup>st</sup> peak force (Chapter 3 Section 3.3.3).



**Figure 3.1.** Typical raw trunk force trajectories. Trunk maximum and 1<sup>st</sup> peak forces shown on the left. Actual trunk force trajectory in one direction, 3 trials, shown on the right. Desired angle of movement indicated by arrow at 45 degrees. Red circles show maximum force magnitude.



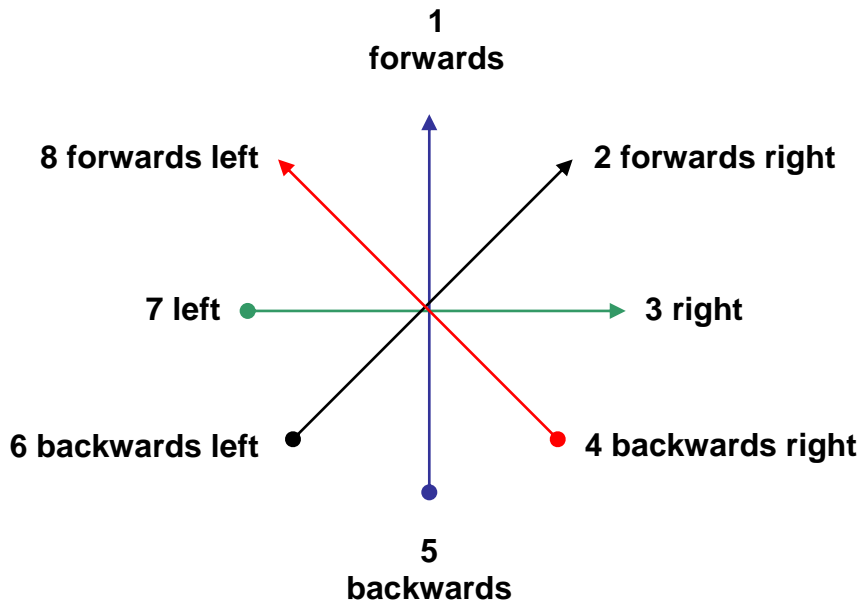
### 3.2.1.2 Absolute and normalised forces

Both maximum and 1<sup>st</sup> peak absolute forces showed considerable variability among individuals. Two approaches were taken to control for the wide spread of force magnitude values. Firstly, absolute forces were analysed in patients and gender matched controls, since subjects' gender influenced the trunk force to a major degree (Table 3.3).

Secondly, data was normalized to reduce the inter-individual variability. It allowed use the whole control group for analysis and so the statistical power was retained. The data was calculated as the normalised force difference between trunk flexion and extension, the paretic and non-paretic sides and the diagonal directions. The normalisation was carried out as follows. Forces were averaged across 3 trials for each subject for each direction. The normalised force asymmetry  $F_A$  between two directions was calculated using the following formula:

$$F_A = F_{da} - F_{db} / F_{da} + F_{db}$$

where  $F_{da}$  (force direction a) and  $F_{db}$  (force direction b) represent backwards (B, direction 5) and forwards (F, direction 1) trunk movement directions giving B-F asymmetry; right (R, direction 3) and left (L, direction 7) giving R-L asymmetry; backwards right (BR, direction 4) and forwards left (FL, direction 8) giving BR-FL asymmetry; and backwards left (BL, direction 6) and forwards right (FR, direction 2) giving BL-FR asymmetry respectively (Fig. 3.2). Thus the normalised data ranged from -1 to 1, the result being 0 if there was no difference in force between the two directions.



**Figure 3.2.** Trunk movement directions and normalised asymmetry calculation.

### 3.2.1.3 Magnitude of seat and feet force in the horizontal plane

The magnitude of the force vector in the horizontal plane generated under the subjects' bottom was calculated from the data obtained from the force plate measuring the reaction forces between the buttocks and the seat (Figs. 2.1 and 3.3).

The magnitude,  $\mathbf{F}$ , of the seat force vector in the horizontal plane was calculated as:

$$\mathbf{F} = \text{sqrt} ( F_x^2 + F_y^2 )$$

where  $F_x$  and  $F_y$  are the magnitudes of the anteroposterior and mediolateral components of the resultant force vector  $\mathbf{F}$ .

The magnitude of the feet force vector in the horizontal plane was calculated from the data obtained from two individual force plates measuring the reaction force between each foot and the ground (Figs. 2.1 and 3.3).

The magnitude,  $\mathbf{F}$ , of each feet force vector in the horizontal plane was calculated as:

$$\mathbf{F}_R = \text{sqrt} ( F_{xR}^2 + F_{yR}^2 ) \text{ and}$$

$$\mathbf{F}_L = \text{sqrt} ( F_{xL}^2 + F_{yL}^2 ) \text{ respectively.}$$

where  $F_x$  and  $F_y$  are the magnitudes of the anteroposterior and mediolateral components of the resultant force vector  $\mathbf{F}_R$  and  $\mathbf{F}_L$  (right and left foot).

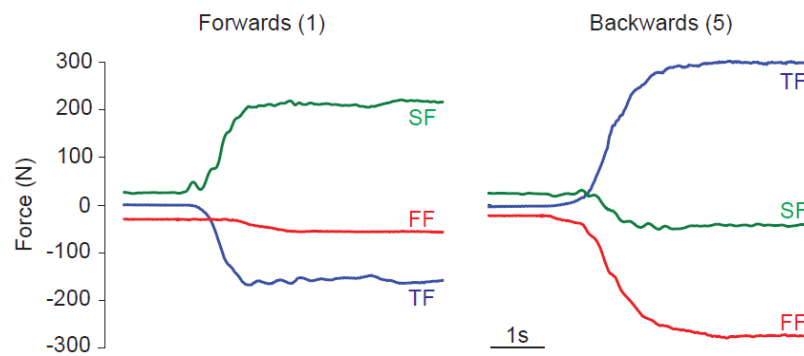
The magnitude of the combined feet force was calculated as:

$$F_{x\text{comb}} = F_{xR} + F_{xL}$$

$$F_{y\text{comb}} = F_{yR} + F_{yL}$$

$$\mathbf{F}_{\text{comb}} = \text{sqrt} ( F_{x\text{comb}}^2 + F_{y\text{comb}}^2 )$$

where  $F_{x\text{comb}}$  and  $F_{y\text{comb}}$  are the magnitudes of the anteroposterior and mediolateral components of the resultant combined force vector  $\mathbf{F}_{\text{comb}}$ .



**Figure 3.3.** Typical raw anteroposterior force trajectories. TF indicates trunk force, SF indicates seat force, FF indicated feet force. Forces shown without pelvis fixation.

### **3.2.1.4 Adjusted seat and feet force calculation**

It was conceivable that the lower body movement did not always follow in the direction of the upper body movement during the force task. In order to control for this possible bias, adjusted seat and feet force was calculated at the maximum effort time point of the trial for each direction of trunk movement as follows:

$$F_a = F_{s/f} * \cos(\theta_t - \theta_{s/f})$$

where  $F_a$  is adjusted force,  $F_{s/f}$  is seat or feet force respectively,  $\theta_t$  is trunk force angle and  $\theta_{s/f}$  is seat or feet force angle respectively.

### **3.2.1.5 Directional accuracy of trunk movement**

The angle,  $\theta$ , of the force direction in the horizontal plane was calculated as  $\theta = \arctan(F_y / F_x)$ .

## **3.2.2 Subjects**

Patients with a cortical or subcortical stroke were recruited from the National Hospital for Neurology and Neurosurgery, London, UK and from the Homerton University Hospital, London, UK. Subjects were recruited if they were able to sit unaided for a short period of time. Exclusion criteria were the presence of a previous stroke or other neurological or orthopaedic condition and severe cognitive or language impairment that would impact the ability to give informed consent and participate in the experiment. 9 patients with left-sided hemiparesis participated in the experiment (6 males, 3 females, mean age of 57.3, SD 17.9 years). An ischaemic stroke occurred in 8 patients and a haemorrhagic stroke occurred in 1 patient. The localisation of the lesions was defined on computed tomography or magnetic resonance imaging (MRI) scans (table 1). Further 3 patients with right-sided hemiparesis and 2 patients with cerebellar stroke participated in the experiment. They were excluded from the analysis due to their low numbers.

23 healthy volunteers with no history of neurological or orthopaedic conditions were recruited as controls. The group comprised of 8 males and 15 females. The age range was 50 to 80 years. There was no difference between the stroke and control groups with regard to age (two-tailed independent *t* test,  $t(30) = 1.09$ ,  $p > 0.05$ ). In one subject the ground force plate recordings were corrupted and therefore did not contribute to the analysis of feet force.

All subjects participated with informed consent and the approval of the local ethics committee according to the Declaration of Helsinki.

### **3.2.3 Clinical measures**

The upper and lower limb strength was measured using The Motricity Index for Motor Impairment after Stroke. The maximum score is 100 with a higher score indicating greater strength. The bodily mobility was assessed with the Rivermead Motor Index. It assesses the patient's ability to move his body in 15 tasks. The score ranges from 0 to 15 with 15 being the best possible score. Impairment of activities of daily living was evaluated using the Barthel Index. The score ranges from 0 to 100. The higher is the score, the greater is the degree of personal independence. Postural function was measured with the Postural Assessment Scale for Stroke Patients (PASS). It evaluates sitting and standing balance and the ability to change posture. Its score ranges from 0 to 36. A higher score indicates better postural stability. Spasticity was measured using the Modified Ashworth Scale. The maximum score is 4 indicating severe spasticity. Personal neglect was assessed with the comb and razor test and visuospatial neglect with stars cancellation and line bisection tests. The score increases with severity of neglect. Behavioural neglect was tested with the Catherine Bergego Scale. This scale rates the patient's functioning in ten life situations. Each item is scored from 1 to 4 with higher score equalling more severe neglect. Recovery of motor function was evaluated with the Modified Rankin Scale that ranges from 1 to 6. Higher score equals greater impairment. Neurological deficit was documented using The National Institutes of Health Stroke Scale (NIHSS). The score ranges from no deficit of 0 points to maximum deficit of 34 points. Sensory impairment was tested using the subtests 1, 2 and 7 of the Rivermead Assessment of Somatosensory Performance (RASP) score. A higher score indicates better

sensation. The patients' neurological impairment ranged from very mild (subjects number 2 and 9) to very severe (subjects number 4, 5, 6, and 8). Three had significant neglect (subjects number 5, 8 and 9) and four showed serious balance problems as tested by the PASS scale (subjects number 1, 4, 5 and 6) (table 2).

### **3.2.4 Experimental procedure**

Two experiments were carried out in this study. The details of the experimental procedure were given in Chapter 2 section 2.2.4.

Briefly, in experiment 1, subjects were instructed to move their trunk with maximum effort in each of eight different directions whilst sitting on the seat. The directions were forwards (1), backwards (5), laterally to the left (7) and right (3) and diagonally to the front left (8), front right (2), rear left (6) and rear right (4) (Fig 2.3A). A computer program chose the direction pseudo-randomly for each trial. The subject was told the direction and instructed to pull with maximum effort in the given direction at the sound of a tone and to relax at the sound of a second tone. The programmed sequence was started by the experimenter via a handheld switch that triggered data collection. Data were sampled at 100 Hz and the total recording time was 6s with the audible tones occurring 1s and 6s after the start of a trial. Each set of eight movement directions was repeated three times. The experiment was conducted first with the pelvis fixated to the platform with the seat straps (see Fig. 2.1) and then repeated in the same fashion without pelvis fixation.

In experiment 2, an additional study was conducted with six healthy volunteers from the control group. This was done in order to answer the question of how integral was the lower body action to upper body movement. It would seem intuitive that normal lower body strength and coordination between the two would generate a bigger upper body force but, it is possible that the body segments act in a synergy that does not necessarily impact on the ultimate magnitude of the generated force and serves a different purpose. The study investigated the effect of ground support on trunk force development. Trunk forces were measured: i) with the feet resting on the ground, and: ii) with the ground support removed by raising the seat so that the

feet did not touch the ground. The experimental paradigm remained the same as in experiment 1. There were 2 males in the group (ages 69 and 51) and 4 females (ages 64, 65, 66, 55). The order of the trunk movement directions and the presence or removal of ground support conditions was set randomly.

### **3.2.5 Data and statistical analysis**

Data were stored on-line and converted to text files for analysis. Calculations were performed off-line using Matlab (The MathWorks Inc., Natick, Massachusetts). Measurements were made of force magnitude and direction at the time of first peak force and the time of maximum force development (Fig. 3.1) in single trials. All analyses were performed on the mean values of the three trials performed in each direction for each condition. The effects of movement direction and pelvis fixation on force parameters were assessed using repeated measures ANOVA design (SPSS version 14). Hyunh-Feldt correction was used when assumption of sphericity was not met. Post hoc analysis was performed using Tukey's test or 2-tailed paired or unpaired samples t-test as appropriate. Correlation of force measures with gender and clinical impairment scales was analysed using Pearson correlation coefficient. Significance was taken as  $p < 0.05$  for all tests. The distribution of trunk movement direction was tested using the circular statistics procedures by Zar (2010).

### **3.3 Results**

In total, 9 stroke patients were included in the analysis. Their characteristics are shown in Tables 3.1 and 3.2.

**Table 3.1.**

Individual data of the 9 patients with left hemiparetic stroke

Subject	Age (yrs)	Gender	Time from CVA (wks)	Lesion
1	68	M	2	R thalamic haemorrhage
2	19	F	3	R FP lobe infarct
3	78	F	2	R IC, adjacent FP lobe infarct
4	64	F	4	R F lobe infarct
5	67	M	6	R FP lobe and BG infarct
6	55	M	14	R FP lobe infarct
7	55	M	4	R FP lobe and BG infarct
8	41	M	20	R P lobe infarct
9	69	M	3	R P lobe infarct

F, female; M, male; R, right; F, frontal; P, parietal; IC insular cortex; BG, basal ganglia

**Table 3.2**

Individual clinical scales scores of the 9 patients with left hemiparetic stroke

Subject	1	2	3	4	5	6	7	8	9
<b>Test</b>									
Motricity	76.5	50.5	100	44	27	48.5	69.5	24.5	100
Mobility	5	12	15	1	2	8	13	9	13
Barthel	55	100	100	45	45	85	95	75	100
PASS	23	31	30	11	9	26	35	31	35
NIHHS	3	5	0	5	8	6	2	11	2
Spasticity	0	0	0	0	2	1	0	1	0
Stars	0	0	0	0	6	1	0	22	11
Lines	0	0	0	0	0	0	0	4	2
Comb+razor	0	0	0	0	20	0	0	100	11
Behavioural	10	10	10	10	13	13	10	16	18
Rankin	4	2	1	4	4	3	1	3	1
RASP	154	203	240	231	202	240	240	186	235



### 3.3.1 Correlation of trunk force with gender

It was shown by Newton et al (1993) that trunk strength correlates with gender. This was replicated in our study. Table 3.3 shows the gender, age and weight characteristics of the controls. Table 3.4 shows the statistical analysis of the effect of age, weight and gender on maximum and 1<sup>st</sup> peak trunk force. The effect of age and weight was analysed using Pearson correlation test. The effect of gender was analysed using unpaired t-test. Significance value p is shown as 2-tailed.

<b>Table 3.3.</b>				
Control group characteristics. Controls matched to patients by age and gender in bold italics.				
<b>Females</b>			<b>Males</b>	
<b>Subject</b>	<b>Age (yrs)</b>	<b>Weight (kg)</b>	<b>Age</b>	<b>Weight</b>
1	60	76	<i>70</i>	88
2	66	84	<i>61</i>	70
3	62	102	<i>53</i>	69
4	55	78	80	77
5	62	68	51	66
6	<i>74</i>	52	<i>50</i>	89
7	69	63	<i>69</i>	65
8	55	58	<i>55</i>	69
9	65	62		
10	63	52		
11	<i>64</i>	83		
12	<i>55</i>	78		
13	66	77		
14	62	83		
15	62	54		

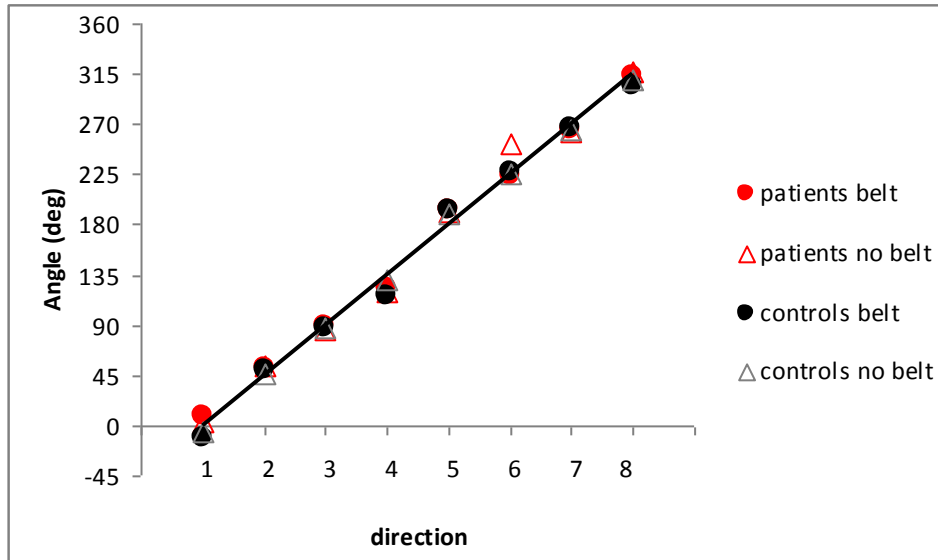
**Table 3.4.**

Correlation between trunk force and age and weight shown as Pearson correlation coefficient r. Correlation between trunk force and gender shown as unpaired t-test t. Significant results in bold.

	<b>Maximum force fixation</b>		<b>Maximum force no fixation</b>		<b>1<sup>st</sup> peak force fixation</b>		<b>1<sup>st</sup> peak force no fixation</b>	
	r	p	r	p	r	p	r	p
Age	-0.24	>0.05	-0.30	>0.05	-0.25	>0.05	-0.30	>0.05
Weight	0.16	>0.05	0.24	>0.05	0.12	>0.05	0.27	>0.05
	t	p	t	p	t	p	t	p
Gender	<b>-3.11</b>	<b>0.005</b>	<b>-3.62</b>	<b>0.002</b>	<b>-3.15</b>	<b>0.005</b>	<b>-3.40</b>	<b>0.003</b>

### 3.3.2 Accuracy of direction of trunk movement

The accuracy of direction of trunk movement was assessed as the extent to which the subjects deviated from the given direction when moving their trunk. The mean actual achieved angle of trunk movement was compared to the desired angle of movement. Fig. 3.4 shows the group mean actual achieved angle of movement direction in relation to the desired angle. The subjects were able to perform the task. The ideal angle lies within the 95% confidence interval meaning that the mean actual achieved angle were not significantly different from the ideal angle (Rayleigh test for circular uniformity,  $p < 0.001$ , Tables 3.5 and 3.6).



**Figure 3.4.** Plot of group mean actual angle of movement in controls and patients in maximum trunk force with and without fixation. Error bars denote 95% confidence interval. Black line joins the ideal angles of movement.

**Table 3.5**

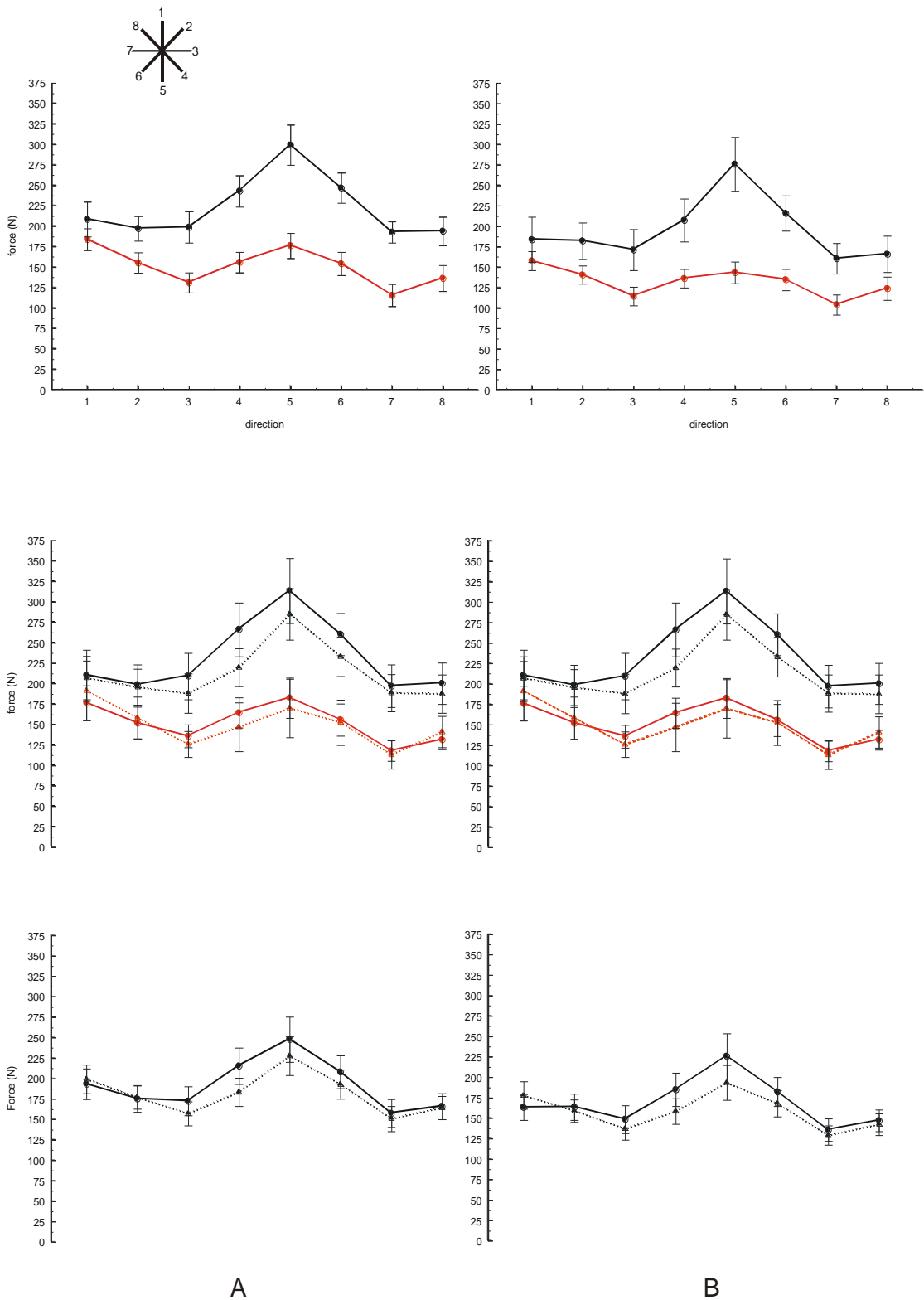
Statistical analysis of circular distribution of maximum force mean angle in left hemiparesis patients shown as 95% CI.

Direction	Ideal angle	Fixation	No fixation
1	90°	85.6-91.7°	80-91.2°
2	45°	47.8-55.2°	48.3-59.3°
3	0°	356.9-20.7°	353.3-14.2°
4	315°	300.3-326.7°	307.1-327°
5	270°	258-272.4°	254.4-271.2°
6	225°	208.6-237.8°	196.1-231.2°
7	180°	164.7-193°	169.8-191.7°
8	135°	116.2-139°	113.2-126.7°

<b>Table 3.6</b>			
Statistical analysis of circular distribution of maximum force mean angle in controls shown as 95% CI.			
<b>Direction</b>	<b>Ideal angle</b>	<b>Fixation</b>	<b>No fixation</b>
1	90°	85.4-90.8°	85.9-88.8°
2	45°	46.1-54.3°	42.1-52.3°
3	0°	344.4-355.4°	349.4-0.04°
4	315°	299.6-309.4°	305.6-315°
5	270°	265.5-268.2°	264.6-267.2°
6	225°	222.3-233.5°	220.4-232.3°
7	180°	182.4-194.8°	180.1-190.7°
8	135°	125.5-139.6°	124.9-137°

### 3.3.3 Effect of stroke, movement direction and pelvis fixation on trunk force magnitude

We used a mixed design (factors: *group*, *fixation*, *direction*) repeated measures ANOVA on the data shown in Fig. 3.5 to examine the effect of stroke on trunk force. Four significant effects were found: 1) a main effect of *group* demonstrating a difference in overall trunk force magnitude between controls and patients both in maximum and 1<sup>st</sup> peak forces; 2) a main effect of *direction* indicating that the trunk force magnitude depended on the direction of trunk movement; 3) a significant *group* x *direction* interaction showing that the effect of direction differed between the groups; 4) a significant *fixation* x *direction* interaction. There was no 3-way interaction between *group*, *direction* and *fixation* (Table 3.7).



**Figure 3.5.** Plot of group mean trunk force magnitude as a function of intended direction. Black line denotes controls, red line denotes patients. Error bars denote standard error of the mean (SEM). Solid line denotes pelvis fixation, dotted line denotes pelvis free. A. Maximum force. B. 1st peak force.

Top panels: Forces obtained with fixed pelvis combined with force obtained with

pelvis free. Middle panels: Force obtained with and without pelvis fixation shown separately. Bottom panels: Forces obtained from controls combined with forces obtained from patients separately for pelvis fixation and pelvis free conditions.

**Table 3.7.**

Statistical analysis of trunk force in left hemiparesis patients and matched controls. Significant results in bold.

<b>Effect/Interaction</b>	<b>Maximum force</b>		<b>1<sup>st</sup> peak force</b>	
	F value	p value	F value	p value
group	<b>6.2 (1,16)</b>	<b>0.024</b>	<b>6 (1,16)</b>	<b>0.026</b>
fixation	2.5 (1,16)	>0.05	3.9 (1,16)	>0.05
direction	<b>14.9 (3.8,61.4)</b>	<b>&lt;0.001</b>	<b>12.8 (3.3,53.5)</b>	<b>&lt;0.001</b>
<i>group x fixation</i>	1.6 (1,16)	>0.05	2.9 (1,16)	>0.05
<i>group x direction</i>	<b>4.8 (3.8,61.4)</b>	<b>0.002</b>	<b>5.8 (3.3,53.5)</b>	<b>0.001</b>
<i>fixation x direction</i>	<b>4 (3.5,56.5)</b>	<b>0.008</b>	<b>5 (3.9,63)</b>	<b>0.001</b>
<i>group x fixation x direction</i>	0.4 (3.5,56.5)	>0.05	0.6 (3.9,63)	>0.05

The effect of direction was analysed further in order to establish whether the trunk force was bigger in controls over the whole workspace or only in some directions (Fig. 3.5 top panel) and to show where the effect of direction was in the *group x direction* interaction. The data from the pelvis fixation and no fixation conditions were combined together separately for controls and left hemiparesis groups. Fig. 3.5, top panels, show the group mean maximum and 1<sup>st</sup> peak trunk force generated in each of the eight directions with the pelvis fixation and without fixation conditions combined. Independent samples t-test showed that the trunk force generated by controls was bigger across most of the workspace. In maximum force, controls generated greater force in all directions except directions 1 and 2. For 1<sup>st</sup> peak force there was a significant difference in all directions except 1, 2 and 8 and the difference in direction 3 was borderline significant (Table 3.8).

**Table 3.8.**

Statistical analysis in matched controls versus left hemiparesis patients. Effect of individual directions on trunk force. Significant results in bold.

Direction	Maximum force		1 <sup>st</sup> peak force	
	t value	p value	t value	p value
1	0.68	>0.05	0.83	>0.05
2	1.42	>0.05	1.47	>0.05
3	<b>2.4</b>	<b>0.029</b>	2.09	0.053
4	<b>2.68</b>	<b>0.016</b>	<b>2.3</b>	<b>0.035</b>
5	<b>3.08</b>	<b>0.007</b>	<b>3.63</b>	<b>0.002</b>
6	<b>3.02</b>	<b>0.008</b>	<b>2.9</b>	<b>0.01</b>
7	<b>2.92</b>	<b>0.01</b>	<b>2.66</b>	<b>0.017</b>
8	<b>2.15</b>	<b>0.047</b>	1.7	>0.05

The significant *fixation x direction* interaction revealed that the fixation had a differential effect depending on direction. In order to analyse this interaction the data from the matched controls and patients for maximum trunk force were combined together separately for fixation and no fixation conditions. Paired t-test analysis on the data shown in Fig. 3.5, bottom panel A, showed that force exerted in directions 3, 4 and 6 with fixation was significantly bigger than without fixation (Table 3.9). Fixating the pelvis helped generate a greater trunk force in movement directions to the right and diagonal backwards and did not improve trunk force exerted to the left direction.

**Table 3.9.**

Statistical analysis in matched controls combined with left hemiparesis patients for fixation and without fixation conditions separately. Effect of fixation on maximum trunk force. Significant results in bold.

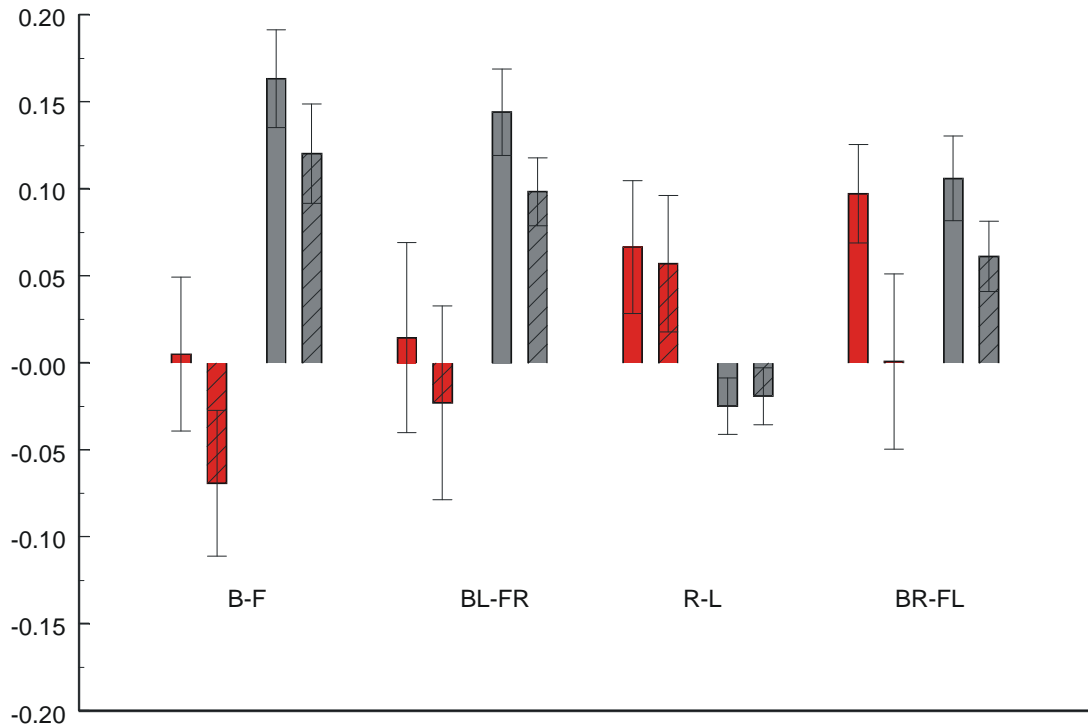
Direction	Maximum force	
	t value	p value
1	-0.85	>0.05
2	-0.35	>0.05
3	<b>2.14</b>	<b>0.047</b>
4	<b>2.88</b>	<b>0.01</b>
5	1.27	>0.05
6	<b>2.26</b>	<b>0.037</b>
7	1.00	>0.05
8	0.27	>0.05

### 3.3.3.1 Effect of stroke and movement direction on trunk force magnitude normalised asymmetry

Normalised force asymmetry (see Section 3.2.1.2) was analysed in the 9 patients and the whole control group. 2-factor mixed design ANOVA (factors: *group*, *fixation*) revealed that trunk force magnitude normalised asymmetry did depend on movement direction in patients (Table 3.10). A significant effect of *group* was found in maximum force in B-F asymmetry, R-L asymmetry and BL-FR asymmetry indicating that the patients generated smaller trunk force in the directions with backwards and left component. In 1<sup>st</sup> peak force, the effect of *group* was significant in B-F and BL-FR asymmetry.

There was a significant effect of *fixation* in B-F, BL-FR and BR-FL asymmetry in maximum force and in B-F and BL-FR asymmetry in 1<sup>st</sup> peak force (Fig. 3.6 and Table 3.9) meaning that pelvis fixation exacerbated the force asymmetry. There was no significant *group* x *fixation* interaction.





**Figure 3.6.** Plot of individual mean normalised maximum trunk force magnitude as a function of B-F, BL-FR, R-L and BR-FL asymmetry. Controls (grey) compared with L hemiparesis patients (red). Error bars denote standard error of the mean. Forces obtained with the pelvis fixed to the seat using belts (solid bar) compared with the pelvis free (crossed bar). Forces have been averaged across 3 trials, forwards side subtracted from backwards side and normalised for B-F asymmetry, left side subtracted from right side and normalised for R-L asymmetry, forwards right side subtracted from backwards left side and normalised for BL-FR asymmetry and forwards left side subtracted from backwards right side and normalised for BR-FL asymmetry respectively.

**Table 3.10.**

Statistical analysis of trunk force asymmetries in left hemiparesis patients and controls. Significant results in bold.

Difference	Effect/ <i>Interaction</i>	Maximum force		1 <sup>st</sup> peak force	
		F value	p value	F value	p value
B-F	group	<b>14 (1,30)</b>	<b>0.001</b>	<b>11.4(1,30)</b>	<b>0.002</b>
	fixation	<b>5.6 (1,30)</b>	<b>0.024</b>	<b>10.9(1,30)</b>	<b>0.002</b>
	<i>group x fixation</i>	0.4 (1,30)	>0.05	1.3(1,30)	>0.05
L-R	group	<b>7.8(1,30)</b>	<b>0.009</b>	3.4(1,30)	>0.05
	fixation	0.01(1,30)	>0.05	0(1,30)	>0.05
	<i>group x fixation</i>	0.2(1,30)	>0.05	0.2(1,30)	>0.05
BL-FR	group	<b>7.4(1,30)</b>	<b>0.011</b>	<b>8(1,30)</b>	<b>0.008</b>
	fixation	<b>5.6(1,30)</b>	<b>0.024</b>	1.7(1,30)	>0.05
	<i>group x fixation</i>	0.6(1,30)	>0.05	0.1(1,30)	>0.05
BR-FL	group	0.7(1,30)	>0.05	1.1(1,30)	>0.05
	fixation	<b>17.9(1,30)</b>	<b>0.003</b>	<b>9.6(1,30)</b>	<b>0.004</b>
	<i>group x fixation</i>	2.4(1,30)	>0.05	0.1(1,30)	>0.05

### 3.3.4 Force development over time

One could expect that stroke would have an effect on the dynamic aspect of trunk force development as well as on the static trunk force magnitude. We analysed the time the subjects took to reach 1<sup>st</sup> peak force and maximum force as a dynamic measure of trunk force generation. Surprisingly, the groups did not differ significantly (3-factor mixed design ANOVA, factors: *group*, *fixation*, *direction*). Patients took on average 363 msec (SEM 9) to reach maximum force and 236 msec to reach 1<sup>st</sup> peak force (SEM 3.2) whilst matched controls took 376 msec and 193 msec respectively (SEM 8, data collapsed across fixation and direction). Fixation of pelvis had significant effect on time to reach a certain force magnitude only in time to reach 1<sup>st</sup> peak force but there was no significant *group x fixation* interaction. The speed of force development did depend on the trunk movement direction but again there was no significant *group x direction* interaction (Table 3.11). Despite the trunk weakness patients were able to activate their trunk muscles with a speed similar to that of the controls.

**Table 3.11.**

Statistical analysis of time to reach maximum and 1<sup>st</sup> peak force in left hemiparesis patients and matched controls. Significant results in bold.

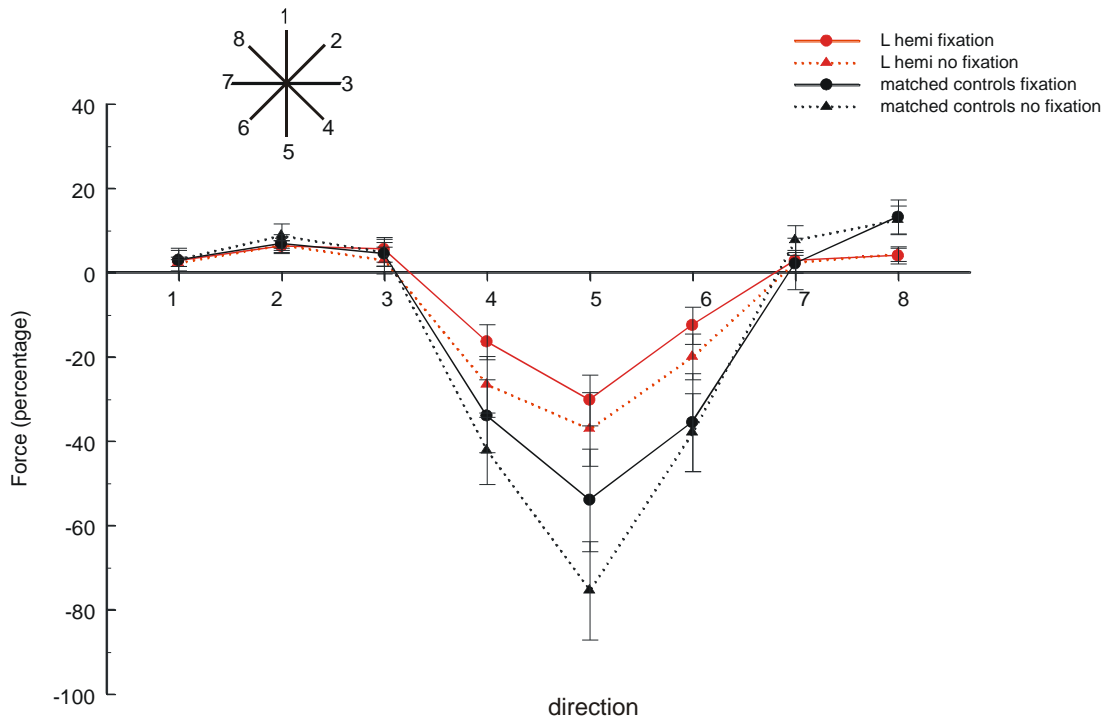
<b>Effect/Interaction</b>	<b>Time to maximum force</b>		<b>Time to 1<sup>st</sup> peak force</b>	
	F value	p value	F value	p value
group	0.1 (1,16)	>0.05	0.4 (1,16)	>0.05
fixation	1.4 (1,16)	>0.05	<b>8.9 (1,16)</b>	<b>0.009</b>
direction	<b>3.9 (5.3,85.1)</b>	<b>0.003</b>	<b>3.7 (3.2,51.7)</b>	<b>0.014</b>
<i>group x fixation</i>	0.04 (1,16)	>0.05	0.7 (1,16)	>0.05
<i>group x direction</i>	1.0 (5.3,85.1)	>0.05	1.0 (3.2,51.7)	>0.05
<i>fixation x direction</i>	1.9 (4.6,74.3)	>0.05	0.4 (3.5,56.7)	>0.05
<i>group x fixation x direction</i>	0.7 (4.6,74.3)	>0.05	0.5 (3.5,56.7)	>0.05

### 3.3.5 Lower body behaviour

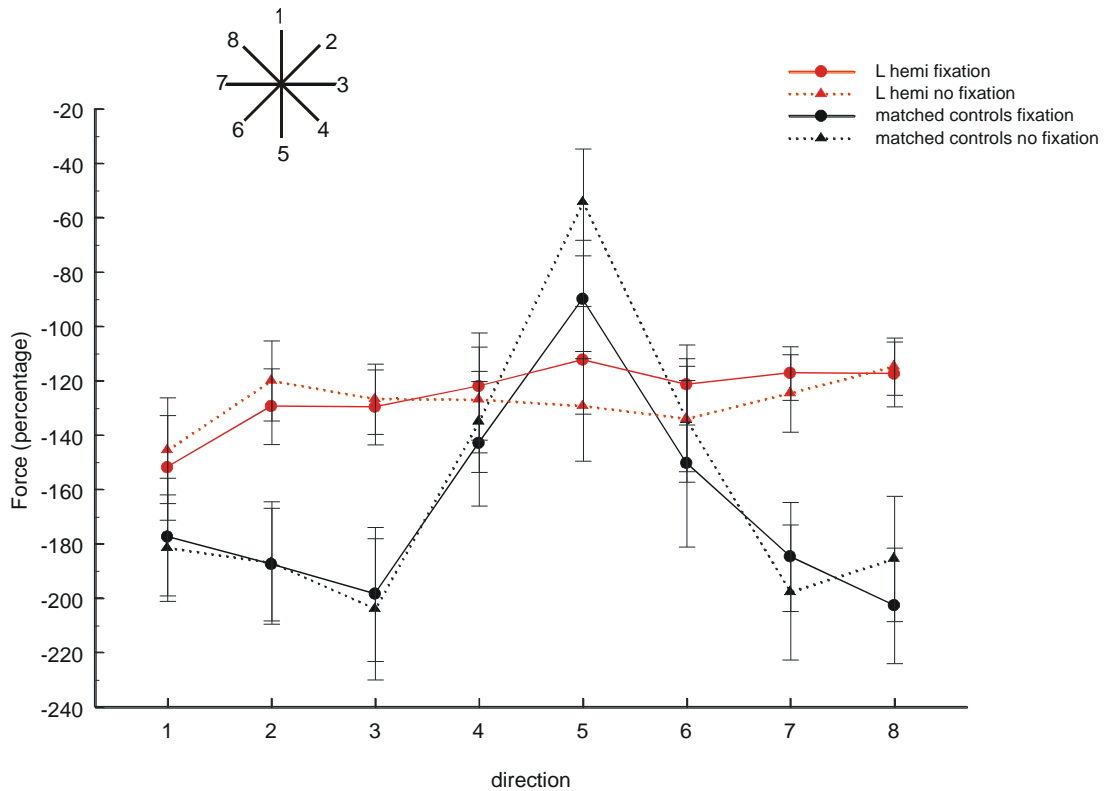
It is not entirely clear how upper and lower body co-operate during trunk movement. One would presume that in stroke patients with their impaired trunk control and balance and trunk and limb weakness this co-ordination would be affected as well. To describe the behaviour of the lower body during voluntary trunk movement we measured the forces generated through the buttocks (seat force) and the feet. In order to control for the possibility that the lower body movement did not follow exactly in the direction of the upper body movement, adjusted seat and feet force was calculated (see Section 3.2.1.4). The adjusted force was then expressed as percentage of the trunk force as a form of normalisation of the data. This was done in order to eliminate the difference in absolute trunk force development between subjects and groups.

Figs. 3.7 and 3.8 demonstrate the normalised adjusted forces exerted through the feet and seat. There was no effect of *group* on the force magnitude. Movement *direction* had a significant effect on the force magnitude and there was a significant *group x direction* interaction indicating that the effect of direction differed between

the groups. There was also a significant *fixation x direction* interaction in feet force only (mixed design repeated measures ANOVA (factors: *group, fixation, direction*, Table 3.12).



**Figure 3.7.** Plot of group mean maximum normalised adjusted feet force magnitude as a function of intended direction. Black line denotes controls, red line denotes patients. Error bars denote standard error of the mean (SEM). Solid line denotes pelvis fixation, dotted line denotes pelvis free.



**Figure 3.8.** Plot of group mean maximum normalised adjusted seat force magnitude as a function of intended direction. Black line denotes controls, red line denotes patients. Error bars denote standard error of the mean (SEM). Solid line denotes pelvis fixation, dotted line denotes pelvis free.

**Table 3.12.**

Statistical analysis of normalised adjusted seat and feet maximum force in left hemiparesis patients and matched controls. Significant results in bold.

<b>Effect/Interaction</b>	<b>Seat force</b>		<b>Feet force</b>	
	F value	p value	F value	p value
group	1.1 (1,16)	>0.05	3.6 (1,15)	0.078
fixation	1 (1,16)	>0.05	3.6 (1,15)	>0.05
direction	<b>16.9 (2.5,40.2)</b>	<b>&lt;0.001</b>	<b>43.4 (2.4,36.8)</b>	<b>&lt;0.001</b>
<i>group x fixation</i>	0.4 (1,16)	>0.05	0.01 (1,16)	>0.05
<i>group x direction</i>	<b>5.4 (2.5,40.2)</b>	<b>0.005</b>	<b>4.4 (2.4,36.8)</b>	<b>0.015</b>
<i>fixation x direction</i>	2.3 (3.6,57.2)	0.073	<b>5 (2.3,33.9)</b>	<b>0.01</b>
<i>group x fixation x direction</i>	1.7 (3.6,57.2)	>0.05	1.5 (2.3,33.9)	>0.05

The data from the pelvis fixation and without fixation conditions were combined together separately for controls and left hemiparesis groups and analysed further in order to investigate the effect of direction and the *group x direction* interaction. Post hoc analysis of the seat force (independent samples t-test) revealed that patients did not activate their lower body in the same pattern as the controls. There was a significant drop in seat force in direction 5 reciprocal to the increase in force generated through the feet in the control group. The seat force generated by controls in the backwards direction (5) was significantly smaller than the force produced by the patients (Table 3.13) and the feet force generated in directions with backward component (5 and 8) was bigger in controls than in patients (Table 3.14).

**Table 3.13.**

Statistical analysis in matched controls versus left hemiparesis patients. Effect of individual directions on seat force. Significant results in bold.

Direction	Maximum force	
	t value	p value
1	0.86	>0.05
2	1.33	>0.05
3	0.51	>0.05
4	-2.09	0.07
5	<b>-2.44</b>	<b>0.04</b>
6	-2.13	>0.05
7	-0.65	>0.05
8	2.06	>0.05

**Table 3.14.**

Statistical analysis in matched controls versus left hemiparesis patients. Effect of individual directions on feet force. Significant results in bold.

Direction	Maximum force	
	t value	p value
1	-0.21	>0.05
2	-0.56	>0.05
3	- 0.92	>0.05
4	1.85	>0.05
5	<b>2.41</b>	<b>0.029</b>
6	2.04	>0.05
7	-0.45	>0.05
8	<b>-2.23</b>	<b>0.041</b>

There was a significant *fixation x direction* interaction in feet but not seat force showing that the fixation had a differential effect depending on direction. Data from the matched controls and patients were combined together separately for fixation and no fixation conditions in order to analyse this interaction further. Paired samples t-test analysis on the data shown in Fig. 3.7 showed that force exerted in directions 4 and 5 with fixation was significantly smaller than without fixation (Table 3.15).

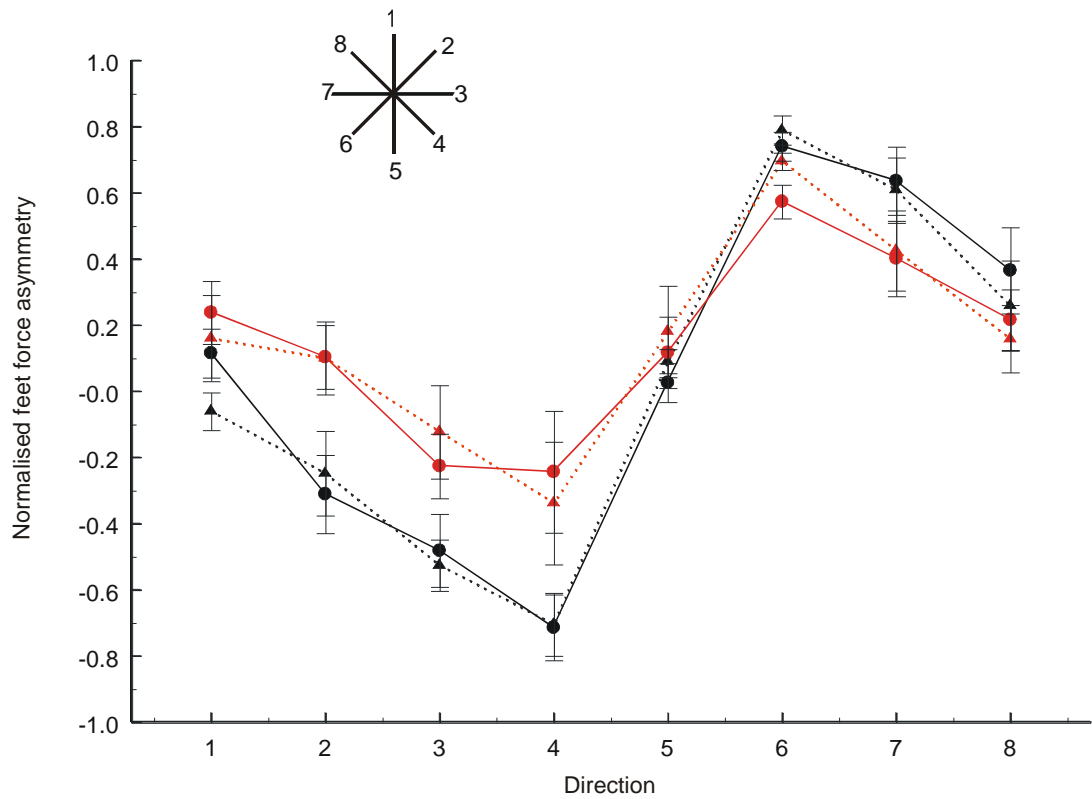
**Table 3.15.**

Statistical analysis in matched controls combined with left hemiparesis patients for fixation and without fixation conditions separately. Effect of fixation on maximum feet force. Significant results in bold.

Direction	Maximum force	
	t value	p value
1	0.68	>0.05
2	-0.4	>0.05
3	1.51	>0.05
4	<b>2.62</b>	<b>0.018</b>
5	<b>2.81</b>	<b>0.012</b>
6	1.17	>0.05
7	-0.9	>0.05
8	0.58	>0.05

We thought that the patients would distribute less force through the left, paretic leg during trunk movement. We measured the force generated through each leg and calculated the contribution of each leg to the total leg force. We expressed the data as normalised right-left leg force asymmetry in order to eliminate the difference in absolute leg force development between subjects and groups. Figure 3.9 shows the distribution of force through each leg during trunk movement. We found a significant difference between the groups in the way each leg was used during trunk movements and the difference depended on direction of movement (mixed design repeated measures ANOVA; factors: *group*, *fixation*, *direction*, Table 3.16). The force was loaded fairly equally between each foot in straight forwards and backwards directions, predominantly through the right leg when the trunk was moved in directions with left sided component and predominantly through the left leg when the trunk was moved in directions with right sided component. However, the patient group distributed less force through the left leg than the control group in movement directions with right sided component but also through the right leg in the backwards left direction (independent samples t-test, Table 3.17).





**Figure 3.9.** Plot of group mean left and right foot normalised force asymmetry as a function of intended direction in matched controls in with and without pelvis fixation conditions. Black line denotes controls, red line denotes patients. Error bars denote standard error of the mean (SEM). Solid line denotes pelvis fixation, dotted line denotes pelvis free.

**Table 3.16.**

Statistical analysis of normalised left and right foot force asymmetry in left hemiparesis patients and matched controls. Significant results in bold.

**Effect/Interaction**

	F value	p value
group	<b>5 (1,30)</b>	<b>0.032</b>
fixation	0.01 (1,30)	>0.05
direction	<b>62.4 (4.2,126.2)</b>	<b>&lt;0.001</b>
<i>group x fixation</i>	0.1 (1,30)	>0.05
<i>group x direction</i>	<b>5.9 (4.2,126.2)</b>	<b>&lt;0.001</b>
<i>fixation x direction</i>	0.5 (4.2,126.2)	>0.05
<i>group x fixation x direction</i>	0.2 (4.2,126.2)	>0.05

**Table 3.17.**

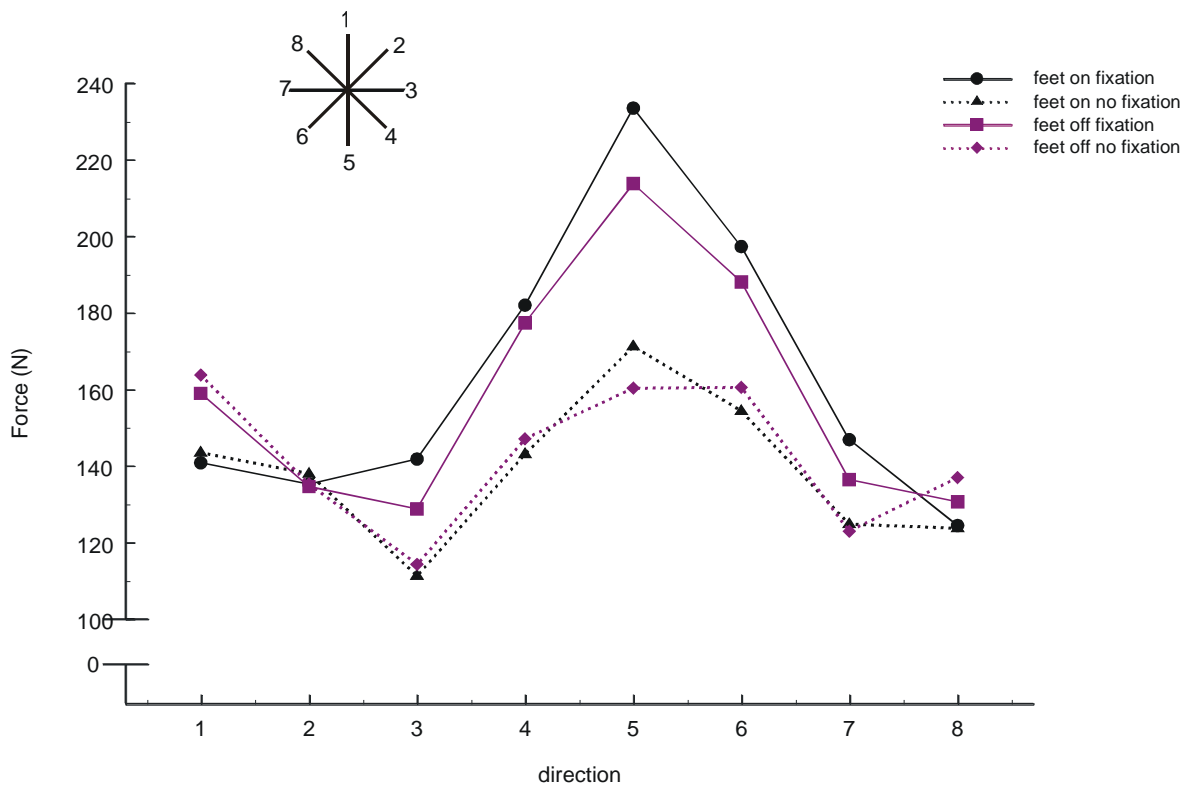
Statistical analysis in matched controls versus left hemiparesis patients. Pelvis fixation and without fixation conditions combined. Effect of direction on maximum feet force normalised left-right asymmetry. Significant results in bold.

Direction	Maximum force	
	t value	p value
1	1.36	>0.05
2	<b>2.75</b>	<b>0.015</b>
3	<b>2.27</b>	<b>0.038</b>
4	1.99	>0.05
5	0.781	>0.05
6	<b>-2.66</b>	<b>0.018</b>
7	-1.42	>0.05
8	-0.83	>0.05

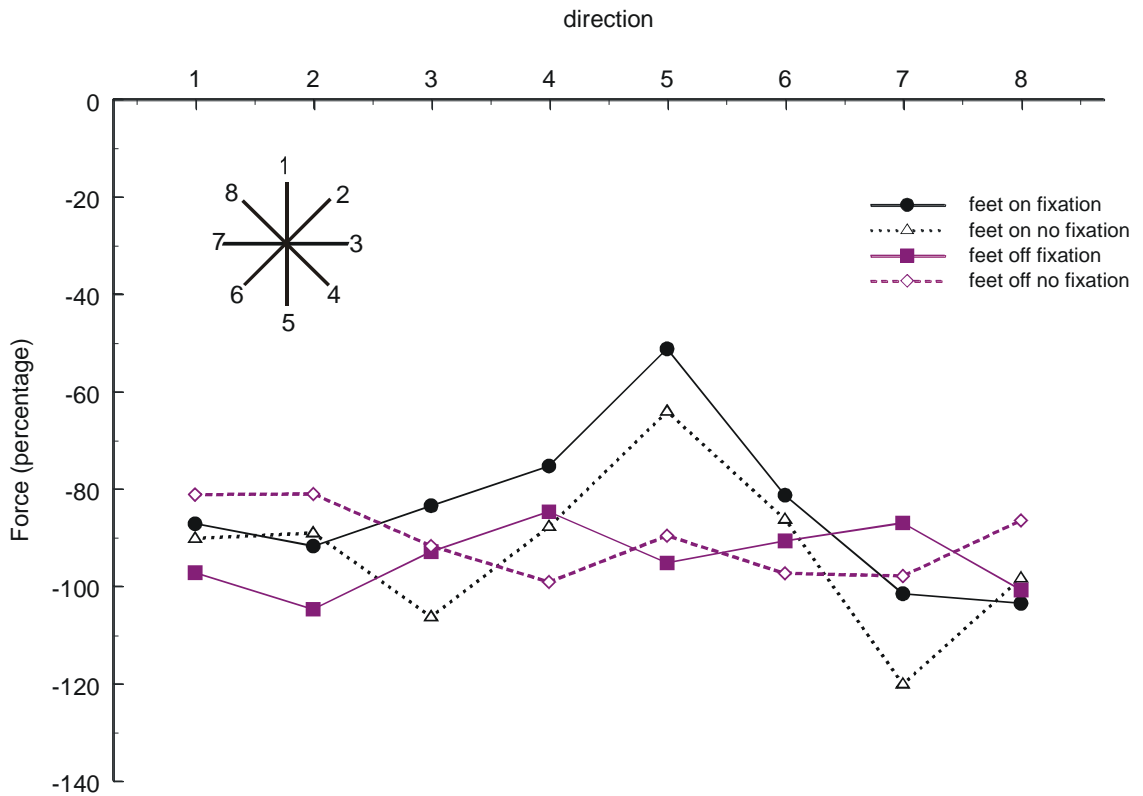
### 3.3.5.1 Effect of removal of ground support in healthy subjects

The behaviour of the lower body during trunk movements was investigated further with an additional experiment where the ground support was removed from under the feet. By removing the ground support and thus the contribution of the lower extremities we hoped to shed more light on the question of how is the lower body action linked to the upper body action during trunk movement (see Section 3.2.4). We used a repeated measure design ANOVA (factors: *ground support*, *fixation*, *direction*) on the data in Figs. 3.10 and 3.11. The seat force was adjusted to the direction of trunk force and normalised as in the main experiment (see Sections 3.2.1.4 and 3.3.4). This showed that the subjects generated a similar trunk force regardless of whether their feet rested on the ground or not indicating that the lower body action contributed only marginally to the overall trunk force magnitude. Ground support did not significantly alter the overall normalised seat force magnitude either (Table 3.18). There was a significant effect of direction on trunk and normalised seat force magnitude in keeping with the results of the main experiment. In normalised seat force a significant *support x direction* interaction was shown. This effect was examined further with post hoc analysis. The pelvis fixation and without fixation conditions were combined for with ground support and without

ground support conditions separately and analysed with paired samples t-test (Fig. 3.12, table 3.19). It showed that with ground support, similarly as in the main experiment, there was a significant decrease in the seat force generated in the backwards direction with borderline significant decrease in directions 4 and 7 as well. This did not happen without ground support when the subjects could not use their legs to move the lower body. Hence without ground support the control subjects' lower body behaviour was similar to that of the patient group in the main experiment. There was no statistical effect of pelvis fixation, perhaps due to the fact that the group size was small with fair amount of between-subject variability.



**Figure 3.10.** Plot of group mean maximum trunk force magnitude as a function of intended direction. Black line denotes feet on ground, magenta line denotes feet off ground. Solid line denotes pelvis fixation, dotted line denotes pelvis free.



**Figure 3.11.** Plot of group mean normalised maximum seat force magnitude as a function of intended direction. Black line denotes feet on ground, magenta line denotes feet off ground. Solid line denotes pelvis fixation, dotted line denotes pelvis free.

**Table 3.18.**

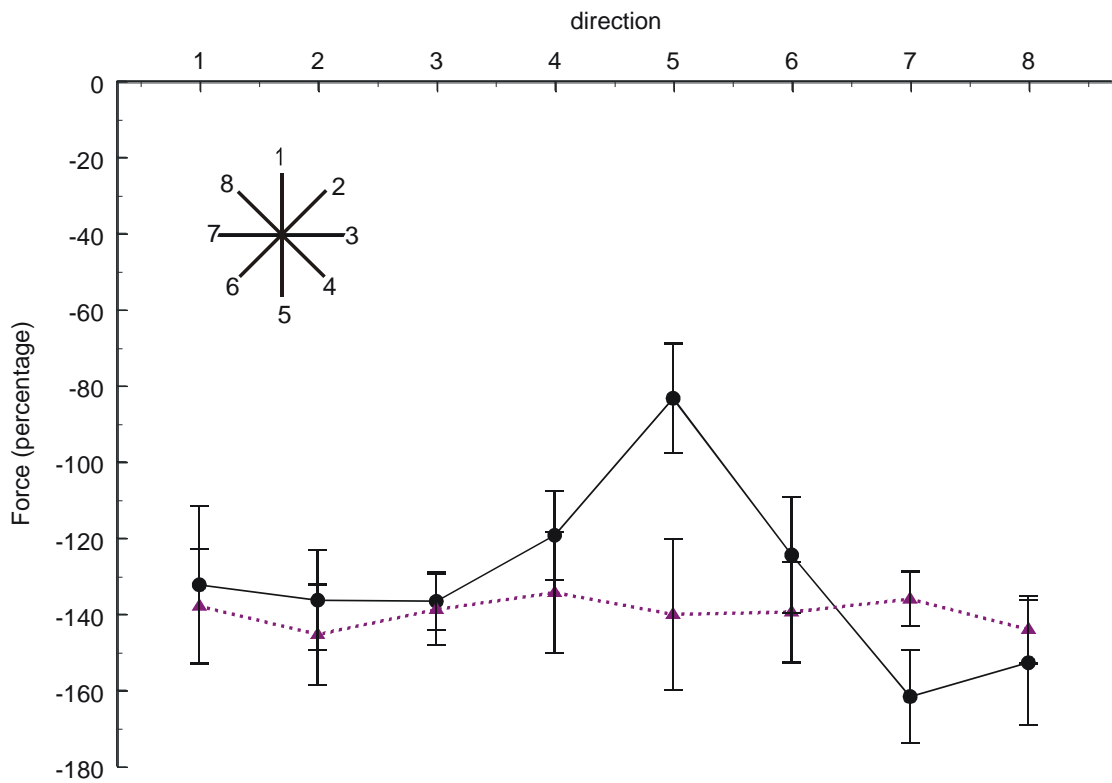
Statistical analysis of maximum trunk and normalised seat forces with and without ground support. Significant results in bold.

**Effect/Interaction**

**Trunk force**

**Seat force**

	F value	p value	F value	p value
ground support	0.001 (1,5)	>0.05	5.2 (1,5)	>0.05
fixation	1.3 (1,5)	>0.05	0.1 (1,5)	>0.05
direction	<b>5.5 (1.6,8.2)</b>	<b>0.035</b>	<b>3.6 (4.6,23.1)</b>	<b>0.016</b>
<i>support x fixation</i>	0.8 (1,5)	>0.05	1.4 (1,5)	>0.05
<i>support x direction</i>	1.2 (3.2,16)	>0.05	<b>6.9 (7,35)</b>	<b>&lt;0.001</b>
<i>fixation x direction</i>	1.8 (1.3,6.3)	>0.05	2.5 (4.2,20.8)	>0.05
<i>support x fixation x direction</i>	0.3 (2.3,11.5)	>0.05	0.8 (6,30.2)	>0.05



**Figure 3.12.** Plot of group mean normalised maximum seat force magnitude as a function of intended direction. Forces obtained with fixed pelvis combined with force obtained with pelvis free. Solid black line denotes feet on ground, dotted magenta line denotes feet off ground. Error bars denote standard error of the mean (SEM).

**Table 3.19.**

Statistical analysis in ground support versus without ground support conditions. Pelvis fixation and without fixation conditions combined. Effect of direction on normalised maximum seat force. Significant results in bold.

Direction	Maximum force	
	t value	p value
1	0.48	>0.05
2	0.81	>0.05
3	0.25	>0.05
4	<b>2.66</b>	<b>0.045</b>
5	<b>4.58</b>	<b>0.006</b>
6	1.2	>0.05
7	<b>-2.66</b>	<b>0.045</b>
8	-0.87	>0.05

### **3.3.6 Correlation of kinematic measures with clinical impairment**

We compared the clinical impairment scales scores of our patients with the laboratory measures that were most affected by the stroke. These were the normalized trunk force asymmetry B-F, R-L and BL-FR measures, overall trunk force in directions 5 and 7, normalized seat force in direction 5, normalized feet force in direction 5 and right-left feet asymmetry in directions 2, 3 and 6. Fixation and without fixation conditions were analysed separately using Pearson correlation coefficient  $r$ . Motricity, Mobility, Barthel index and PASS are scales where a higher score indicates less neurological deficit. The  $r$  is therefore positive when correlating with seat and feet force and BL-FR asymmetry and negative when correlating with R-L asymmetry. A higher score in NIHSS, spasticity, comb-razor test of personal neglect and Rankin score indicates more severe neurological deficit, hence the  $r$  is negative when correlating with seat and feet force and BL-FR asymmetry and positive when correlating with R-L asymmetry. Only the normalized trunk force L-R and BL-FR asymmetries and normalized seat and feet forces in backwards direction showed some correlation with clinical impairment. The clinical measures that most commonly correlated with the kinetic measures were the NIHHS, spasticity and the comb-razor test of neglect. The correlation was more pronounced in the with-pelvis-fixation condition (Tables 3.20 and 3.21). Overall, there was little correlation of trunk movement deficit as measured by our method and neurological deficit as measured by clinical scales.

**Table 3.20.**  
Correlation between clinical scales and kinematic measures in with fixation condition shown as Pearson correlation coefficient r. Significance shown as p 2-tailed in brackets. Only significant results presented.

	RLa	BLFRA	Seat 5	Feet 5	R-L feet asymmetry direction 2	R-L feet asymmetry direction 3	R-L feet asymmetry direction 6
Motricity	-0.65(>0.05)	0.49(>0.05)	0.39(>0.05)	<b>0.71 (0.032)</b>	0.44 (>0.05)	-0.38(>0.05)	0.46 (>0.05)
Mobility	-0.19(>0.05)	0.07(>0.05)	0.51(>0.05)	<b>0.77 (0.015)</b>	-0.04(>0.05)	-0.65(>0.05)	-0.02(>0.05)
Barthel	-0.23(>0.05)	0.05(>0.05)	0.59(>0.05)	<b>0.77 (0.015)</b>	-0.09(>0.05)	<b>-0.75 (0.02)</b>	-0.16(>0.05)
PASS	-0.17(>0.05)	-0.10(>0.05)	0.30(>0.05)	<b>0.78 (0.012)</b>	-0.04(>0.05)	-0.66(0.053)	-0.2 (>0.05)
NIHHS	<b>0.77 (0.015)</b>	<b>-0.73(0.025)</b>	-0.47(>0.05)	<b>-0.7 (0.037)</b>	-0.5 (>0.05)	0.29(>0.05)	-0.41(>0.05)
Spasticity	<b>0.85 (0.003)</b>	-0.30(>0.05)	-0.51(>0.05)	-0.64(>0.05)	<b>-0.7 (0.036)</b>	0.09(>0.05)	0.07 (>0.05)
Comhazor	<b>0.75 (0.02)</b>	<b>-0.86(0.003)</b>	-0.40(>0.05)	-0.44(>0.05)	-0.3 (>0.05)	0.28(>0.05)	-0.18(>0.05)
Rankin	0.22 (>0.05)	-0.17(>0.05)	-0.67 (0.05)	<b>-0.77 (0.015)</b>	-0.15(>0.05)	0.56(>0.05)	-0.01(>0.05)

**Table 3.21.**  
Correlation between clinical scales and kinematic measures in without fixation condition shown as Pearson correlation coefficient  $r$ . Significance shown as  $p < 0.05$ -tailed in brackets. Significant results in bold.

	RLa	BLFRa	Seat 5	Feet 5	R-L feet asymmetry direction 2	R-L feet asymmetry direction 3	R-L feet asymmetry direction 6
Motricity	-0.42(>0.05)	0.50(>0.05)	0.37(>0.05)	0.34(>0.05)	0.03(>0.05)	-0.54(>0.05)	0.18(>0.05)
Mobility	-0.02(>0.05)	-0.11(>0.05)	0.42(>0.05)	0.46(>0.05)	0.14(>0.05)	-0.49(>0.05)	0.18(>0.05)
Barthel	-0.23(>0.05)	-0.10(>0.05)	0.41(>0.05)	0.45(>0.05)	0.05(>0.05)	-0.59(>0.05)	0.05(>0.05)
PASS	-0.41(>0.05)	-0.26(>0.05)	0.59(>0.05)	0.20(>0.05)	0.28(>0.05)	-0.32(>0.05)	-0.01(>0.05)
NIHHS	0.46(>0.05)	-0.65(>0.05)	-0.55(>0.05)	-0.48(>0.05)	-0.02(>0.05)	<b>0.67(0.048)</b>	-0.13(>0.05)
Spasticity	<b>0.82(0.007)</b>	-0.17(>0.05)	<b>-0.68(0.045)</b>	-0.49(>0.05)	-0.59(>0.05)	0.33(>0.05)	-0.16(>0.05)
Combrazor	0.31(>0.05)	<b>-0.81(0.008)</b>	-0.46(>0.05)	-0.45(>0.05)	0.34 (>0.05)	0.75 (0.02)	0.12(>0.05)
Rankin	0.24(>0.05)	0.002(>0.05)	-0.53(>0.05)	-0.35(>0.05)	-0.13(>0.05)	0.54(>0.05)	-0.07(>0.05)



### 3.4 Discussion

We have shown that patients with left hemiparetic stroke have impaired trunk strength across most of the workspace with the trunk extension and movement to the paretic side being most affected in comparison with normal controls. Also, by measuring force generated through the seat and feet and manipulating the way forces are transmitted from the spine and pelvis to the seat and ground, we were able to investigate for the first time how lower body is activated during trunk movements in sitting and how is this behaviour affected by pelvis stabilization and by stroke. We found that patients did not increase force generated through the feet with a reciprocal drop in force generated through the buttocks in trunk extension in contrast to healthy subjects and that they loaded each leg differently from the controls during trunk movements distributing less force through the left leg in movement directions with right sided component but also through the right leg in the backwards left direction. These findings enable us to discuss the issue of how trunk muscle weakness, difficulties of coordination between upper and lower body, lower body weakness and problems of stabilization by the lower body affect trunk movement after stroke.

In order to understand the relationship between trunk muscle strength problem and upper and lower body stabilization problem we need to consider how trunk and lower body muscles act during trunk movement in sitting and how forces are transmitted from the trunk to the ground in our experiment. The transmission of forces happens through the skeleton of the spine and the joints between the trunk and pelvis to the seat to the frame and the ground. To achieve the transmission the spine and pelvis are stabilized by the deep fibers of the lumbar multifidus and transverse abdominis muscles at spinal level and the gluteal, psoas and iliacus muscles at pelvis level. At the seat surface the forces are resisted by friction between the clothing worn by the subject and the high friction foam that covers the seat. If the frictional force between the bottom and the seat is overcome through insufficient pelvis stabilization or leg action, the force is transmitted through the leg joints to the ground. The frictional force between the feet and the ground is low compared to that between the bottom and the seat as the floor force plate surface is low friction steel, hence more stabilization is required by the leg muscles executed principally by the

soleus muscle and biceps femoris muscle. In our study, we fixated the pelvis to the seat with belts. In so doing we have in theory provided the required reaction force normally generated by the lower body and removed the need of the trunk forces to be transmitted to the seat and feet. However, despite pelvis fixation forces were still generated through seat and feet. The forces not taken by the belts did not appear to have a mechanical function but could be a result of axial muscle synergies.

We have demonstrated that stroke patients generated smaller upper body forces principally in extension and lateral flexion to the left and smaller lower body forces in extension and left backward movement. We found that stabilization of the lower body at the level of the pelvis did have a positive effect on the trunk force, enabling the patients and controls generate a bigger force in the right (i.e. non-paretic in patients), right backward and left backward directions. Yet the stabilization did not increase the force produced in the left, i.e. patients' paretic, direction nor did it allow patients to achieve the same force magnitude as controls. We suggest that this indicates that whilst true weakness of trunk prime movers was present, it was compounded by weakness of pelvis and hip extensors and impaired co-ordination between the muscle groups resulting in deficient stabilization of the spine and pelvis necessary for trunk movements. This hypothesis is supported by studies on muscle synergies, movements involving two or more joints that require muscle coordination and co-activation with a specific temporal distribution. They were most extensively studied in the upper limbs, to a lesser degree in the lower limbs and in axial muscles in standing but little is known about how forces are transferred from the upper to the lower body during trunk movements in sitting. Most information on trunk synergies concerns co-activation of trunk flexors and extensors and transfer of forces through the hands during lifting and pulling tasks which are very relevant to activities of daily living. Trunk extension has been studied in patients with low back pain, although the results may not be applicable to stroke patients. The differences in muscle activation in patients with low back pain might be due to limiting effect of pain itself rather than weakness and spasticity as in stroke victims. Findings by Leinonen et al (2000) could be relevant to our study. The authors recorded surface electromyography from back and hip flexors and extensors during a flexion-extension task in low back pain sufferers and found shorter activation of the hip extensor gluteus maximus muscle. One could argue that deficit in gluteus maximus

activation with resulting poor pelvic stabilization impairs trunk stabilization in extension and contributed to our findings of weak trunk extension in post-stroke patients. Several studies have shown that trunk extension strength improved with dynamic exercise significantly more when the pelvis was stabilized (Graves et al 1994, da Silva et al 2009, San Juan et al 2005).

Axial synergies (movements of the lower body segments opposite to trunk movements) stabilize antero-posterior displacements of centre of gravity during trunk movements in standing and thus maintain equilibrium (Crenna et al 1987, Odsson 1990, Alexandrov et al 1998). Data from sport medicine suggest that similar synergies play a role during trunk movements in sitting. The trunk extension task in our experiment resembles a portion of a rowing stroke. There the resulting force of the stroke is largely determined by extension of the legs. Obviously, forces are also transferred to the handle through the hands. Pollock (2007) collected kinematic and surface EMG data from nine healthy female rowers. The muscles studied comprised trunk flexors rectus abdominis, external and internal oblique and transversus abdominis muscles, trunk extensors erector spinae lumbar and thoracic and latissimus dorsi and hip extensors biceps femoris and gluteus maximus. The author found little co-contraction of the trunk flexors and extensors during extension of the trunk whilst the predominant activity occurred in the extensors of the pelvis and spine with a similar time of onset. She concluded that co-ordination of the back and pelvis extensors was crucial for stabilisation of the spine in trunk extension. Winzeler-Mercay et al (2002) collected EMG data in stroke patients from rectus abdominis and erector spinae during trunk flexion and extension, forward reaching and shoe donning. They suggested that trunk muscle weakness can become more pronounced when the trunk muscle is required to act as a pelvic stabilizer in order to provide support for limb action.

It is also noticeable that even healthy controls gained from external stabilization when moving diagonally backwards. That could suggest that these movements are more difficult to stabilize requiring stronger spine and pelvis stabilizing muscles. Force generated through the legs did not appear to contribute significantly to force generation in trunk extension. In our experiment where ground support was removed the control subjects generated a similar trunk force regardless of whether their feet

rested on the ground or not. There was also a trend towards bigger force in backward directions with pelvis fixation. The results did not reach statistical significance probably due to the small sample size. Our patients also generated relatively more force through the buttocks than the feet during trunk extension. This would point towards pelvis fixation and stabilization rather than transmission of forces through the legs being the important contributor to generation of high trunk forces in diagonal backwards directions.

To discuss the trunk muscle weakness found in our study we have to review advances in the understanding of neuroanatomy of the motor pathways and regions governing axial muscle function and the effect of stroke on these structures. The information available on cortical control of axial musculature is still considerably less detailed compared with knowledge of cortical projections to arm and hand muscles. Recent studies using transcranial magnetic stimulation with EMG recordings from individual axial muscles have established that axial muscles are innervated bilaterally. Distal limb muscles, on the other hand, are mainly innervated by contralateral corticospinal input (Palmer et al 1992).

Several pathways have been suggested to provide the bilateral axial input. Kuypers (1964) proposed corticobulbar, corticospinal and subcorticospinal pathways incorporating the vestibulospinal, reticulospinal and tectospinal fibers based on his studies on cats, Rhesus monkeys and chimpanzees. Studies on healthy volunteers proposed fast conducting corticospinal tracts (Carr et al 1994, Ferbert et al 1992). Ferbert et al (1992) found both short and long latency ipsilateral responses from the lumbar erector spinae suggestive of slow conducting pathways acting in parallel with the faster direct corticospinal route, possibly corticoreticulospinal or corticorubrospinal pathways. Similarly Fujiwara et al (2001) recorded longer latency ipsilateral than contralateral responses from the external oblique muscles in stroke group compared to controls. The authors suggested that this response might be mediated by polysynaptic pathways such as corticoreticulospinal pathway. Tunstall et al (2001) recorded longer latency ipsilateral responses from the rectus abdominis muscle and suggested that slower conducting corticospinal pathways were involved. Similar conclusions were made by Strutton et al (2004) who recorded longer latency ipsilateral responses from the internal oblique muscle.

It appears that the ipsilateral activity is increased after stroke and is associated with better recovery of axial muscles. On the contrary, the increased ipsilateral activity seems to correlate with poorer recovery of arm and hand function (Ward et al 2003, Schwerin et al 2008). It is possible that this increase in ipsilateral responses is due to loss of transcallosal inhibition after a hemispheric lesion (Meyer et al 1995). Misawa et al (2008) elicited ipsilateral motor evoked potentials (MEP) from the trapezius muscle more frequently in stroke patients than in healthy controls. The patients with recordable ipsilateral activity during voluntary contraction also had less severe paresis in the trapezius and deltoid muscles but not in the more distal arm muscles. Muellbacher et al (1999) found that recovery of unilateral lingual paralysis can be achieved without input from the affected hemisphere. They elicited contralateral and ipsilateral CMAPs from 40 controls but from none of their six stroke patients. All patients recovered their lingual function but in only one patient TMS managed to elicit CMAPs from the affected hemisphere. Fujiwara et al (2001) found bilateral fast responses in external oblique and erector spinae muscles when stimulating healthy controls and the unaffected hemisphere of stroke patients. Both responses in patients had an increased MEP ratio than the controls. Furthermore, the MEP ratio increased as the trunk muscle performance as measured by clinical assessment scores (Trunk Control Test and trunk control parts of the Stroke Impairment Assessment Set) improved. These findings showed that the ipsilateral pathways are hyperexcitable after stroke and that the ipsilateral activation can compensate for the loss of the crossed contralateral input caused by stroke. In particular the brainstem descending pathways identified by Kuypers (1964) project primarily to the spinal intermediate zone and the dorsomedial parts of the ventral horn and branch extensively over several spinal segments thus innervating motoneurons of axial and proximal limbs muscles. This provides the residual structure to depend on after the main input has been damaged. Yet the ipsilateral control could be suboptimal in view of the slower conduction.

The exact cortical representation of the trunk muscles and the origin of the pathways that mediate the ipsilateral trunk MEPs is not clear. MacKinnon et al (2004) have not shown any significant difference between centres of gravity of the scalp area from which ipsilateral and contralateral MEPs for the latissimus dorsi

muscle could be elicited. In contrast, O'Connell et al (2007) found that ipsilateral responses were obtained from sites lateral to the optimal site for evoking contralateral MEPs when mapping the cortical representation of the lumbar paravertebral muscles. They were also able to evoke contralateral MEPs from areas anterior to the optimal site indicating possible projections from the supplementary motor area (SMA). Thus MEPs in trunk muscles could be mediated from sites in primary motor cortex, premotor cortex and supplementary motor area.

Our findings of predominant left and backward trunk weakness could therefore be explained by loss of input from the affected right hemisphere that is only partly compensated by the increased activity in the unaffected ipsilateral left hemisphere. However, we found that the trunk extension was weaker than left lateral flexion. This cannot be explained by damage to the prime mover function of trunk muscles only but suggests an element of stabilization difficulty between the spine and pelvis and at spinal level. Rectus abdominis (RA) and external oblique (EO) muscles are the prime movers in trunk flexion and trunk lateral flexion and latissimus dorsi (LD) and erector spinae (ES) muscles in trunk extension. Studies exploring bilateral EMG activity in these muscles showed opposing results with regard to impairment of their prime mover function. Dickstein et al (2004) studied RA and EO muscles in trunk flexion and LD and ES muscles in trunk extension after stroke. They found reduced and delayed activity of RA and LD compared to the unaffected side in the patients and to the controls, EO function was impaired to a lesser degree and erector spinae showed normal activity. In contrast, Winzeler-Mercay et al (2002) found normal activity in RA during maintenance of trunk flexion and extension but demonstrated asymmetric RA weakness more profound on the paretic side during shoe donning task where the muscle was acting as a pelvic stabilizer as well as a prime mover. ES muscle showed asymmetric abnormal activity both during steady state postures and dynamic tasks. Even healthy subjects showed asymmetric EMG activation during lifting tasks significant in the external oblique, gluteus maximus, iliocostalis lumborum pars thoracis, and latissimus dorsi muscles reflecting their role as prime movers but also global stabilizers of the spine and pelvis (Danneels et al 2001).

We have demonstrated that the directions of trunk movement most affected by stroke were trunk extension and lateral flexion to the paretic side in comparison to

the opposite direction whilst forward flexion and forward right flexion were the only directions in which patients generated muscle force similar to that of the controls. These results agree with some of the findings of other studies but contradict their other findings. Bohannon et al (1995) found a difference in overall trunk strength between paretic and non-paretic sides and between patients and controls, but in contrast to our study they also demonstrated a trunk flexion weakness in stroke patients. This discrepancy in results is most likely due to the differences in methodology. These studies differ markedly in the way the trunk strength was measured and in the extent of the workspace the trunk movement measurements covered. Bohannon et al used a hand held dynamometer with its potentially limited accuracy of strength measurement and of direction of trunk movement and validity (Chapter 2.1). They also measured only forward flexion, lateral flexion to the paretic side and lateral flexion to the non-paretic side. In our study we were able to distinguish with good accuracy between forward and forward left (or right) trunk movement direction. Directions of movement with left (i.e. towards the paretic side) or backward component were the most affected in our study. It is possible that the forward direction measured in Bohannon's study contained some movements in forward right or forward left direction, i.e. in directions towards the subjects' paretic side, producing a lower strength measurement on average. Karatas et al (2004) and Tanaka et al (1998) have found that stroke patients were weak in trunk flexion and even more so in extension. Their studies tested only trunk flexion and trunk extension using an isokinetic dynamometer. In contrast, this study found that trunk flexion strength was preserved in patients with left hemiparesis, and indeed, they performed the best in this direction, whilst trunk extension was significantly impaired. Again, it is possible to explain the discrepancy by difference in the method used to measure trunk muscle strength. Isokinetic dynamometry has its limitations with regard to adjustment for gravity, patient stabilization and placement of the axis of movement which affect the accuracy of direction of movement and force measured (Chapter 2.1).

We have not found any difference in trunk movement velocity between patients and controls. However, Dickstein et al (2004) showed that stroke patients moved their trunk at lower velocities than controls but the methodology used was different. It involved testing extension from bending forwards touching a table with the

forehead and flexion from reclining backwards to 130° hip flexion. Also Dickstein's sample was much bigger comprising 50 stroke patients and 30 healthy volunteers.

Furthermore, all the studies tested patients with right and left hemiparesis whilst our study only focused on patients with right hemispheric lesion. This might have influenced our results in view of the findings that right hemispheric lesions have a greater negative impact on postural performance and sitting balance than left hemispheric lesions (Bohannon et al 1986, Pérennou et al 1999, Rode et al 1997, Bonan et al 2007).

Trunk strength correlated more frequently only with three out of the twelve clinical measures we used to quantify the neurological deficit in our patients. These were the NIHHS, spasticity and comb-razor test of neglect. NIHHS is a comprehensive scale and the correlation could be linked to the neglect item of the scale. Spatial neglect has been shown to be associated with postural instability after stroke (Pérennou 2006). Bohannon (1995) measured the relation between trunk muscle strength and clinical variables after stroke. He did not find any correlation with the Motricity Index that was also used in this experiment. He demonstrated a relationship with the Trunk Control Test (TCT) but that scale was not used in this study. Karatas et al (2004) found a correlation between trunk muscle strength and the locomotion-transfers subscore of the Functional Independence Measure scale (FIM) and the Berg Balance Scale. Again, those scales were not used in this experiment. Nevertheless, the PASS scale that was used overlaps with the TCT and Berg Balance Scale to some extent, yet no correlation was found with any of the measures studied. The reason for that might be the fact that this study used a smaller number of patients. Also, the TCT and Berg Balance Scales focus more on trunk balance and control alone and therefore could correlate better with trunk muscle weakness than the more comprehensive scales used in this study. Most of the scales we used measure leg function and not surprisingly, the feet force produced in backwards direction correlated with most scales in the with-pelvis fixation condition only. There was little correlation with the right-left feet asymmetry. This could be due to the different leg action being assessed in clinical examination and in our experiment.



We have investigated trunk muscle strength in voluntary movements covering the whole workspace whilst simultaneously recording reaction forces at the points of contact of the lower body after stroke. This is the first study that demonstrates the relationship between trunk and lower body movements affected by stroke and this was achieved by studying how the forces are transferred from trunk to pelvis and legs during trunk movements. This approach has shown that stroke impairs both prime mover and spine stabilizer trunk muscle function which is further compounded by weakness of pelvis muscles resulting in impairment of co-ordination between trunk and pelvis and deficient stabilization of the whole axis. These results contribute to our understanding of the physiological mechanisms that affect the functional impairment of trunk movement and control after stroke. In order to understand the dynamic process of trunk control recovery patients will have to be studied longitudinally during their recovery.

## **CHAPTER 4. Conclusion and general discussion**

### **4.1 Overview**

This chapter will summarise the main results of the experiments presented in this thesis. The results have already been discussed in Chapters 2 and 3. In this chapter, the important methodological issues of the study will be addressed. The implications of the results will then be discussed in the context of trunk strength recovery.

### **4.2 Summary of results**

#### **4.2.1 A new method for quantifying trunk control**

In chapter 2, a novel method of measuring trunk motor control was described. A new apparatus using a novel force-transducing system enables to study the time-course of development of trunk force magnitude and direction in sitting. The subject is able to exert quasi-isometric forces with the trunk in any horizontal direction, with or without the pelvis stabilised. In addition, reaction forces between the buttocks and the seat and between each foot and the ground are measured with three independent force plates. Reproducibility of the new method was demonstrated in the experiment. The agreement analysis showed a mean bias of the new system of only  $-3.0$  N for force magnitude and  $0.72$  degree for force direction. The trunk force measurement system was sensitive enough to detect effects of movement direction and pelvis fixation on maximum force magnitude. When subjects repeated the test at a later date there were no significant differences between the two sessions. In summary, the new device allows measurement of static and dynamic aspects of voluntary trunk activation in multiple directions while seated and provides reproducible measurements for application to longitudinal studies.

#### **4.2.2 Trunk motor control after acute right hemispheric stroke**

In Chapter 3, we have shown how acute right hemispheric stroke affects voluntary trunk movement and co-ordination between upper and lower body during trunk movement. We found that trunk muscle strength was impaired over most of the

whole workspace with a greater impairment of trunk extension and left lateral flexion. Trunk weakness greater on the side of hemiparesis was previously described (Bohannon 1995). Our results extend the findings of previous studies (Tanaka et al 1998, Karatas et al 2004) that found trunk flexion rather than extension weakness. Also, unlike previous studies, this experiment assessed aspects of trunk movement not investigated before. We found that the directional accuracy of trunk movement was not impaired over the entire workspace; that stroke patients exerted a smaller force through the lower body during trunk muscle activation and; when the need to coordinate the upper and lower body was abolished by stabilising the pelvis, all subjects were able to produce bigger trunk force in the right and diagonal backwards directions yet patients were not able to increase their trunk strength to match that of the controls. We have therefore hypothesized that the trunk weakness was a result of true prime trunk movers muscle weakness coupled with lack of coordination between the upper and lower body.

### **4.3 Methodological issues**

The main weakness of the present thesis is the small sample size. This was due mainly to the fact that a large proportion of patients admitted to the units from which subjects for the study were recruited had to be eliminated from recruitment based on exclusion criteria. Patients were largely willing to take part if approached. A significant number of patients had suffered from a previous stroke and hence could not be considered for recruitment in the study. Another common reason for patients to be excluded from recruitment was cognitive impairment or severe dysphasia that prevented patients from being able to give informed consent and understand commands during the experiment. Lastly, some patients could not be recruited due to the severity of their overall clinical condition. Our initial recruitment focused on patients within 2 weeks of stroke onset from Queen Square Hospital only. Given the low number of recruits, the inclusion criteria was expanded to incorporate all first ever stroke patients at any time of onset, also the pool of potential participants was enlarged by reaching out to Homerton Hospital Stroke Unit. In order to generalise the results, they should be confirmed in a larger more representative sample. In future studies multiple sites should be identified for recruitment in order to maximise the number of potential study subjects. The small sample size also made it

impossible to assess the impact of lesion site on trunk control. In addition, it was not possible to recruit a large enough number of patients with left hemispheric and cerebellar strokes which compromised the potential to generalise the findings further. Trunk motor control in patients with cerebellar lesions has not been studied extensively and we believe that our new method would be very useful for investigation of this group of patients.

We found that the interaction between upper and lower body in trunk movements was impaired in stroke patients but failed to demonstrate that they would benefit from fixation of their pelvis. There is no standardised way to stabilise the pelvis during trunk movements in sitting. It is possible that our pelvis fixation was not applied at the optimal position. On the other hand, the number of ways to fixate pelvis is limited and we believe that we had achieved a good stabilisation of the lower body. It is therefore possible that pelvis fixation is not sufficient means to compensate for impairment of coordination between upper and lower body in trunk movement and for loss of trunk muscle strength.

#### **4.4 Trunk motor recovery following stroke**

The method used in this thesis could be a very useful tool for longitudinal studies. 5 of the patients who participated in the experiments were also tested repeatedly. The results were not included in the thesis due to a high heterogeneity of the stroke type and length of follow-up assessments of the group. Of the 3 longitudinally assessed patients with right hemispheric stroke, 1 was tested twice over 6 weeks, 1 three times over 12 weeks and 1 four times over 12 weeks. 1 patient with left hemispheric stroke was tested twice over 6 weeks and 1 with cerebellar stroke was assessed four times over 6 weeks.

The functional and motor recovery potential of a stroke depends on a number of factors, such as its type, the degree of the initial deficit, age, prior stroke, persistent urinary and faecal incontinence, visual-spatial deficits, the level of social support, metabolic rate of glucose outside the infarct area in hypertensive patients and treatment at a dedicated stroke unit. The initial severity of motor dysfunction

appears to be the most important factor (Wade et al 1983, Skilbeck et al 1983, Jørgensen et al 1995, Feys et al 2000).

The effect of the initial stroke severity was investigated in The Copenhagen Stroke Study (Jørgensen et al 1995). The authors followed 1197 patients for 6 months. 21% of patients died whilst inpatient. After completed rehabilitation, 78% of patients had no or only mild deficit and 46% had no disability in activities of daily living. On the other hand, 11% had severe neurological deficits and 20% were severely disabled. The speed of functional recovery strongly correlated with the initial stroke severity. Thus patients with mild stroke achieved their best function in activities of daily living (ADL) within 8.5 weeks from onset, patients with moderate stroke within 13 weeks, patients with severe stroke within 17 weeks and patients with very severe stroke within 20 weeks.

With regard to subtypes of cerebral infarction, subjects with partial anterior circulation stroke, posterior circulation stroke and lacunar stroke achieve the 1-minute independent sitting, the 10-second independent standing and the 10-meter walk mobility milestones most rapidly. However, patients with intracranial haemorrhage and those with total anterior circulation infarcts were hospitalised two to three times as long and were most likely to fail to achieve independent ambulation (Smith et al 1999).

Hendricks' et al (2002) meta analysis of studies on motor recovery after stroke showed that in case of complete paralysis, only 15% of patients regain complete motor function, both for the upper and lower limb. Overall, 65% of patients with initial deficit of any severity in the leg achieve some degree of motor recovery. The data on arm recovery is less conclusive. Study of 92 patients by Wade et al (1983) showed that 14% of patients with initial complete paralysis of the arm regained full function and further 25% partial function (Wade et al 1983). Kong et al (2011) found that more than 1 year following a stroke, 28.3% of their 140-subject sample regained dexterity in the upper limb.

Most motor recovery occurs within the first month after acute stroke followed by further significant improvement three to six months later with less significant

improvement seen up to 1 year after the initial insult (Newman 1972, Wade et al 1983, Skilbeck et al 1983, Hendricks et al 2002, Kotila et al 1984). Horgan et al (2009) did see continuing significant improvement in motor disability and social participation between 6 to 12 months after the onset of stroke although they did observe a slowdown after 9 weeks. Feyes et al (2000) observed a higher level of arm motor recovery from 2 to 12 months in patients with subcortical lesions.

Whilst motor recovery of arm and leg function has been studied extensively, little is known about how the trunk muscles and movement recover from stroke. Bohannon (1995) showed in a sample of 28 patients that trunk muscle strength recovers over time after acute stroke. The increases were bigger in forward flexion than both paretic and non-paretic lateral side flexion at 5-day follow up. The forward flexion was initially the weakest direction tested. Fujiwara et al (2001) evaluated trunk muscle recovery with transcranial magnetic stimulation (TMS). They found that in most of their 20 stroke patients, TMS of the affected hemisphere did not evoke motor evoked potentials (MEP) in the external oblique and the erector spinae muscles but stimulation of the unaffected hemisphere evoked a bilateral response in both muscles. At the second study 3 months later, stimulation of the affected hemisphere evoked a response in only 1 patient. The MEP ratio of the ipsilateral external oblique muscle and the Trunk Control Test score increased significantly from the first assessment. Since the MEP ratio and clinical assessment score improved whilst the cortical projection from the affected hemisphere did not change, the authors concluded that the trunk muscle recovery was mediated by the unaffected hemisphere and probably by a compensatory activation of preexisting uncrossed pathways rather than cortical reorganization. A morphological study using CT imaging found that trunk muscle size increases on the side contralateral to the brain lesion with time. Tsuji et al (2003) measured cross-sectional area (CSA) and mean CT number of paravertebral and thigh muscles in 83 stroke patients after admission and before discharge from a stroke rehabilitation unit. Thigh muscle size increased also but on the ipsilesional side. This was interpreted as the result of different innervation of the tested muscles. Paravertebral muscles are predominantly innervated by uncrossed fibers from the unaffected hemisphere whilst limb muscles receive mostly unilateral innervation. The increase in CSA was bigger for the leg muscles than trunk muscles. This was possibly due to bilateral innervation of trunk

muscles preventing marked atrophy and weakness of trunk on the affected side. With regard to the time course of trunk recovery, Verheyden et al (2008) showed it to be similar to that of the arm, leg and functional ability. They assessed 32 patients with the Trunk Impairment Scale, Fugl-Meyer arm and leg test and Barthel Index at 1 week and 1, 3 and 6 months after stroke. The most significant improvement occurred from 1 week to 1 month with no significant improvement found from 3 to 6 months.

#### **4.5 Future directions**

The experiments in this thesis have contributed to our knowledge about what happens to trunk control after acute vascular brain injury. The novel method of measuring trunk movement used in the study allows to assess both static and dynamic aspects of trunk strength and to evaluate the contribution of the lower body to trunk movement. It provides means for an instrumented analysis of long-term effect of stroke on trunk muscle strength, trunk control and sitting balance which should be utilised in further studies. Future experiments should also aim to expand the scientific basis of therapeutic interventions for recovery of trunk function after stroke.

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