# POSTURAL STABILITY DURING STANDING AND WALKING AND THE EFFECTS OF AGEING

Deirdre Beth Birtles

A thesis submitted in partial fulfillment of the requirements for the degree of

Doctor of Philosophy in the University of London

1999

UCL Institute of Human Performance

Department of Physiology

University College London



ProQuest LLC.
789 East Eisenhower Parkway
P.O. Box 1346
Ann Arbor, MI 48106 – 1346

# Abstract

The postural stability during quiet stance and during walking was investigated in 22 elderly and 20 young subjects. A motion analysis system was used to simultaneously record movements of 14 markers on the body while a force plate recorded movement of the centre of pressure (COP) during stance (but not during walking). The movements of the body during stance could be well described (> 90 % of the variance explained) as a simple inverted pendulum moving about the ankles in the anterior-posterior and medial-lateral directions. This model was applicable to both young and elderly subjects and also predicted the records of COP movement well ($r > 0.90$). When account was taken of the ground reaction forces the prediction was further improved. The greater COP movements commonly observed in the elderly are shown to be due to increased pendulum sway in the medial-lateral direction, compared to young subjects. The inverted pendulum model also gave an adequate description of the deviations from the mean path ("sway") during walking which are larger than those during stance. The static measurement that best predicts sway during walking is medial-lateral movements of the COP when standing on a compliant surface with the eyes closed.

The relationship between muscle strength and COP displacement was examined in a larger group of elderly subjects (N = 56). Maximum voluntary force per cross-sectional area was found not to be correlated with COP movements during quiet stance. This suggests that muscle weakness and increased sway in the elderly have separate physiological causes.

A method was developed for inducing a trip-like perturbation of gait as subjects walked on a treadmill. Muscle activation patterns and body kinematics were recorded in 9 young subjects to establish the normative response to such a perturbation with a view to investigating these responses in the elderly.

# Acknowledgements

I extend my sincere gratitude to the following people:

Professor Roger Woledge, for all his guidance, patience and inspiration during the course of this degree; to Dr Martin Rosenberg for his encouragement and sound advice; Research into Ageing, for providing me with the studentship; the members of the Institute of Human Performance, particularly Tony Christopher who provided the technical expertise on the design and maintenance of the equipment and also provided the creative input on many of the figures presented in the thesis; to all the subjects, young and old, who so willingly participated in the experiments; to my parents and sister for their moral and financial support and to my husband John, whose support and understanding has helped enormously in the realisation of this work.

# Contents

# List of Tables

## Results

### *3.1 Postural Stability during standing*



### *3.2 Postural stability during walking*

### *3.3 Relationship between movements of COP and MVF/CSA*



### *3.4 Recovery responses to a trip*



### *Appendix A:* *Corresponds to Results 3.1*

# List of Figures

## Introduction



## Methods



## Results

### *3.1 Postural Stability during standing*

### *3.2 Postural Stability during walking*



### *3.3 The relationship between movements of COP and MVF/CSA*



### *3.4 The recovery responses to a trip*

## Discussion

### *4.1 The recovery responses to a trip*

# List of Abbreviations

| | | |
|---|---|---|
| 3-D | - | Three dimensional |
| ABR | - | Auditory Brain Response |
| A/P | - | Anterior–Posterior |
| BOS | - | Base of support |
| CNS | - | Central nervous system |
| COG | - | Centre of Gravity |
| COM | - | Centre of Mass |
| COP | - | Centre of Pressure |
| CSA | - | Cross sectional area |
| $D_j$ | - | Diffusion coefficient |
| DSP | - | Digital signal processor |
| EMG | - | Electromyogram |
| EC | - | Eyes closed |
| EO | - | Eyes open |
| FEC | - | Foam eyes closed |
| FEO | - | Foam eyes open |
| GABA | - | Gamma-Amino-Butyric acid |
| HRT | - | Hormone Replacement Therapy |
| LED | - | Light emitting diode |
| MGa | - | Medial gastrocnemius |
| M/L | - | Medial-Lateral |
| MVF | - | Maximum voluntary force |
| MVF/CSA | - | Maximum voluntary force per cross-sectional area of muscle |
| TA | - | Tibialis Anterior |

# *Chapter 1: Introduction*

# 1.0 Falls in the elderly

It has been estimated that approximately 30% of people over the age of 65, living in the community, fall each year (Campbell, Borrie, Spears, Jackson, Brown, & Fitzgerald, 1990;. Tinetti, Speechley, & Ginter, 1988). Although only 5% of the falls result in a fracture and only 5-10% cause serious injuries that require medical attention (Gryfe, Amies & Ashley, 1977; Tinetti, 1987; Tinetti, Speechley & Ginter, 1988) the fear of future falls can lead to a progressive restriction of lifestyle and mobility (Prudham & Evans, 1981). Repeated falls and fear of falling are common reasons for the admission of previously independent elderly persons to an institution (Smallegan, 1983; Lilley, Arie & Chilvers, 1995). In some cases it is possible to identify a single aetiology for the fall such as syncope, but the majority of falls do not have a clear cause and are the result of a combination of factors. These may include the ageing process, disease, prescription medication, the activity being undertaken and environmental hazards (Campbell, Reinken, Allan & Martinez, 1981). Failure to successfully negotiate an environmental hazard accounts for 30 - 50% of reported falls (Sheldon, 1960; Brocklehurst, Exton-Smith, Lempert-Barber, Hunt & Palmer, 1978; Lipsitz, Jonsson, Kelley & Koestner, 1991). However, there is likely to be an interaction between the environmental hazard (extrinsic factor) and intrinsic factors such as age and disease that lead to an increased susceptibility to falling.

*Intrinsic factors that contribute to fall risk*

Loss of postural control in the elderly may be due to a decreased sensory input (e.g. poor vision, peripheral neuropathy), impaired central nervous system processing (e.g. dementia, Parkinson's Disease), impaired muscle response (e.g. muscle weakness, osteoarthritis) or some combination of these factors. Acute illnesses such as pneumonia may also increase fall risk temporarily (Tinetti & Speechley, 1989).

*Decreased sensory input*

Age-related declines in all the major sensory systems important for balance and mobility (visual, proprioceptive and vestibular) have been well documented. Even a mild impairment in any two of these systems could combine synergistically to reduce postural stability during both standing and walking. Visual impairment is common in the elderly population as a result of age-related changes in acuity, visual field, contrast sensitivity and dark adaption (McFarland and Fisher, 1955; Sekuler & Hutman, 1980; Klein, Klein & Linton, 1992; Mitchell, Smith, Attebo & Wang, 1995; Attebo, Mitchell & Smith, 1996). Conditions such as cataracts, macular degeneration and glaucoma are also serious threats to vision in the elderly (Kornzweig, 1977; Ivers, Cumming, Mitchell & Attebo, 1998). A cross-sectional survey of eye disease with retrospective fall history found that poor visual acuity, reduced visual field, impaired contrast sensitivity and the presence of cataract were associated with recurrent falls (Ivers *et al.* 1998).

The proprioceptive system is particularly important for maintaining postural stability during changes of position, walking on uneven surfaces and when other senses are impaired. In addition, during normal standing, proprioceptive inputs from the legs rather than visual or vestibular inputs provide the most sensitive means of perceiving postural sway (Fitzpatrick & McCloskey, 1994). The power of input from muscle spindles for providing information about body sway has been demonstrated by the production of postural sway in unrestrained subjects in response to vibratory stimulation of soleus or tibialis distal tendons and illusionary movements in restrained, standing subjects (Roll, Hay, Quoniam & Roll, 1992). Quoniam and colleagues (1995) used tendon vibration to investigate the effects of ageing on segmental reflexes in the upper limb and on postural responses in a group of young subjects (20 - 44 years old) and a group of elderly subjects (60 to 86 years old). They recorded tonic vibration reflexes from biceps and triceps brachii and showed no age-related changes in the latency or amplitude of the EMG. Similarly there was no age-related change in the latency

of the postural sway response elicited by applying vibration to the ankles. However, there was a decrease in the mean velocity, acceleration and maximum amplitude of the sway with age. The authors concluded that as no major changes are observed at the lower reflex level it is at the higher integrative level that proprioceptive messages are affected by ageing. There have been reports of absent ankle tendon reflexes in the elderly (MacLennan, Timothy & Hall, 1980), particularly in elderly fallers (Pack, Wolfson, Amerman, Whipple & Kaplan, 1985). However, this loss rather than being an inevitable consequence of ageing, is suggestive of large-fibre peripheral neuropathy (Sabin, 1997). Peripheral neuropathy is associated with diseases such as diabetes, which is prevalent in the elderly, as well as conditions such as vitamin B12 deficiency and some medications. The increased cutaneous vibratory thresholds frequently observed in the feet of the elderly (MacLennan *et al.* 1980; Brocklehurst, Robertson & James-Groom, 1982) are probably also due to peripheral neuropathy. In addition, degeneration of the cervical spine from cervical spondylosis, injury or arthritis may disturb postural control in the elderly because of damage to mechanoreceptors in the apophyseal joints (Tinetti & Speechley, 1989).

Over 60% of the elderly population over the age of 70 experience symptoms commonly associated with vestibular disorders such as dizziness or vertigo, (Sixt & Landahl, 1987). The vestibular system provides information about the movement of the head, via the velocity sensitive semicircular canals and about the position of the head relative to the effective direction of gravity via the otolith organs (utricle and saccule). Sensory loss in the semicircular canals can reach up to 40% by the age of 70 (Bergstrom, 1973; Rosenhall & Rubin, 1975) and a loss of neurofibers and hair cells in the utricle and saccule amount to a loss of sensitivity of between 21 - 24% (Bergstrom, 1973). The visual and prorioceptive systems can generally compensate for the loss of both types of vestibular input (Paulus, Straube & Brandt, 1987). However, when visual and proprioceptive inputs are reduced, absent or inappropriate, the vestibular system is crucial for the maintenance of upright

stance, as studies on patients with vestibular loss have shown (Nashner, Black & Wall, 1982). A combination of reduced vestibular information and age-related changes in the visual and proprioceptive systems will increase the risk of falling in the elderly.

*Impaired CNS processing*

Changes in the elderly brain may affect the selection and initiation of corrective postural responses and cause gait irregularities that predispose an elderly person to falling. For example, loss of dendritic spines in the Betz cells of the cerebral cortex (Area 4) may be related to timing problems and slower responses in the elderly (Scheibel, 1985). Parkinson's disease is a common degenerative disease of the nervous system with a mean age of onset of 62 years (Hoehn & Yahr, 1967). Gait abnormalities are the presenting complaint in 12-18% of Parkinson's Disease patients (Hoehn & Yahr, 1967; Martin, Loewenson, Resch & Baker, 1973) while postural instability and falls are often seen in the late stages of the disease. The gait irregularities observed in Parkinson's Disease may be due to a loss of neurones in the pedunculopontine nucleus, as this region of brainstem plays an important role in the supraspinal control of locomotion (Mori, 1987; Armstrong, 1988, Austin & Kalivas, 1991). Lesions in the thalamus and basal ganglia can produce abnormalities in gait and balance (Masdeu, and Gorelick 1988; Labadie, Awerbuch, Hamilton & Rapesak, 1989; Masdeu *et al.* 1994) and gait disturbances caused by cerebellar or vestibular cerebrovascular lesions are also well documented (Kase *et al.* 1993). White matter changes, which often occur as a result of arteriolar disease, have been correlated with impaired gait and balance and a tendency to fall (Masdeu *et al.* 1989). Fibres in the periventricular region are particularly susceptible to ischaemic damage (De Reuck, Crevits, De Coster, Sieben & Vander-Eccken 1980; Harrison & Marshall, 1984) and travelling through this region are the fibres that mediate the long-loop reflexes essential for control of balance and gait (Thompson and Marsden, 1987). Cognitive impairment, depression and anxiety have been associated with falls in a large number of studies (Campbell *et al.*, 1981; Nevitt, Cummings, Kidd, & Black,

1989; Tinetti, Williams & Mayewski, 1986) providing indirect evidence for abnormal higher-level neurological function in postural instability.

*Impaired motor response*

Any disease or disability that affects the muscles, bones, and joints will result in some degree of immobility and contribute to the risk of falling. Adequate muscle strength and joint range of motion, especially in the lower extremities, are essential for generating an effective response to a perturbation of balance (Whipple, Wolfson & Amerman, 1987). Muscles atrophy with age, by 60-70 years of age muscle mass decreases by 25-30% (Grimby & Saltin, 1983) which results in loss of strength. In addition to atrophy, the remaining muscle has a reduced maximum force for a given cross-sectional area (Young, Stokes & Crowe, 1985; Bruce, Newton & Woledge, 1989). Age-related changes in the connective tissue (Alnaqeeb, Zaid & Goldspink, 1984) and/or degeneration of cartilage will reduce flexibility and range of motion of the joints. Unstable, deformed or painful joints may also impair function and increase fall risk. Even deformities of the foot such as calluses and bunions, may disturb gait and balance, resulting in falls (Tinetti, Speechley & Ginter, 1988; Rubenstein, Robbins, Shulman, Rosado, Osterweil, & Josephson, 1988; Tinetti & Speechley, 1989).

*Medications*

Certain prescription medications and changes in dose have previously been associated with falls (Prudham & Evans 1981; Wild, Nayak & Isaacs, 1981; Granek, Baker, Abbey, Robinson, Myers, Samkoff, & Klein, 1987; Larson, Kukull, Buchner, Reifler, 1987) and fractures (Ray, Griffin, Schaffner, Baugh & Melton, 1987). In particular medications that cause instability, fatigue, hypotension and impair mental ability such as psychotropics, sedatives and anti-hypertensives will increase an elderly persons fall risk (Granek, *et al.* 1987; Sorok & Shimkin, 1988). Over-the-counter medications may also increase fall risk if

their side effects include drowsiness or postural hypotension. In addition, studies have shown a direct relation between the total number of drugs used and the risk of falling (Tinetti, Williams & Mayewski, 1986; Campbell, Borrie & Spears, 1989, Robbins, Rubenstein, Josephson, Schulman, Osterweil & Fine 1989). The total number of drugs may be an indicator of frailty but psychotropic and hypotensive drugs are likely to be direct contributors to falls.

*Extrinsic factors that contribute to fall risk*

Environmental hazards include obstacles such as uneven paving and stairs as well as circumstances where sensory input is diminished, for example, areas with poor lighting or thick carpeting. The likelihood of being seriously injured is increased when a fall results from an activity displacing the centre of gravity, such as a slip or a trip (Tinetti *et al.* 1995). The degree of hazard associated with environmental factors depends upon the individual. Elderly persons with a shuffling gait and decreased step height will be more vulnerable to tripping while those with decreased mobility or postural control may find even routine movements such as bed transfers, bending and turning are sufficiently challenging to cause a fall (Nevitt, Cummings & Hudes, 1991). Experience can also modify the degree of hazard. Older adults who use stairs regularly are at lower risk/use than those who use them infrequently.

*Reducing fall risk*

Several studies have shown that risk of falling increases dramatically as the number of risk factors increase (Tinetti *et al.* 1986, 1988; Nevitt *et al.* 1989; Robbins *et al.* 1989). In their survey of community-living elderly persons Tinetti *et al.* (1988) reported that the percentage of persons falling increased from 27% among those with no or one risk factor to 78% among those with 4 or more risk factors. (Risk factors included sedative use, decreased cognition, leg and foot disabilities, gait and balance impairments and the presence of a palmomental

reflex). Many of the contributing factors are amenable to treatment and therefore by reducing the number of risk factors an individual's risk of experiencing a fall and its devastating consequences will also be reduced. Tinetti and colleagues (1994) conducted a randomised control intervention study involving behavioural and medical changes, education and exercise in a group of community dwelling elderly (≥ 70 years). At the one year follow-up, the intervention group had fewer risk factors, a reduced fear of falling and there was a significant (~30%) reduction in falls and fall risk. These results highlight how regular screening for fall risk factors and targeted intervention programs can reduce the incidence of falls in the community dwelling elderly and help to maintain their independence and quality of life.

## 1.1 Postural stability during standing

The postural sway which invariably accompanies the upright stance has been the object of study for well over 100 years (Romberg, 1853). Several studies have shown that sway increases in older people (Sheldon, J.H., 1963; Peterka and Black, 1990; Wolfson *et al.* 1992) and that the frequency of falls increases as sway increases (Overstall, Exton-Smith, Imms & Johnson, 1977; Brocklehurst, Robertson & James-Groom, 1982; Fernie, Gryfe, Holliday & Llewellyn, 1982; Lichtenstein, Sheilds, Shiavi & Burger, 1988). Therefore, quantitative measures of sway could be an important clinical tool for identifying older people at risk for falling. In many of the investigations of sway a force plate is used to obtain records of movement of the centre of pressure under the feet during quiet stance. Older adults show larger centre of pressure (COP) excursions compared to young adults, particularly under conditions where sensory information is absent or conflicting. For example, Woollacott, Shumway-Cook & Nashner (1986) demonstrated that older adults had significantly more sway than young adults under conditions in which ankle proprioception was limited (platform moving in proportion to the subjects sway) and visual input was either occluded or rotated to follow body sway. The COP is an indirect measure of body sway and does not reveal what kind of postural movements have been utilised during the balance test. One of the aims of this thesis is to investigate more fully the movements of the body responsible for this age-related increase in COP excursion.

*Movements of the COP versus movements of the body's centre of gravity*

The regulation of posture and balance during quiet standing requires the control of the body's centre of gravity (COG) about an equilibrium point within the base of support (BOS). If the COG moves outside the BOS potential instability arises which may necessitate a corrective movement to return the COG to within the confines of the BOS. Movements of the COG are not the same as movements of the COP. It is well established that movements

of the COG and COP can differ in amplitude, phase and frequency (Thomas & Whitney, 1959; Gurfinkel, 1973; Kapteyn, 1973; Murray, Siereg, & Sepic, 1975; Ruder, MacKinnon, & Winter, 1989; Benda, Riley, & Krebs, 1994). The low frequency content of the COP and COG movements is similar but movements of the COP have higher frequency components absent in the movements of the COG. The COG of the body is the point in three-dimensional space about which the sum of the torque produced by the weights of the body parts is equal to zero. For the purpose of investigating the relationship between the COP and the COG, the location of the vertical vector from the COG projected onto the support surface is sometimes called the COG as vertical movements of the COG are very small during quiet standing. The COP is the location of the net force vector on the support surface. Movements of the COP reflect both the movement of the COG and the actions of the neuromuscular system to maintain the COG within the BOS. A change in the level of activity of a relevant muscle will instantaneously produce a movement of the COP but there is a delay before the same change in muscle activity will produce a movement of the COG. The relationship between the COP and COG is illustrated by Figure 1.1.1. Here we see a diagrammatic representation of a subject swaying back and forth while standing erect on a force plate. Each figure shows the changing situation at six different points over time. At time (A) the COG is directly above the COP, the body is perfectly balanced and the ankle dorsiflexors and plantarflexes are electrically silent. At (B) a large backward sway of the body has occurred which stretches the ankle dorsiflexors. When the CNS detects that this backward sway needs correcting there is an activation of the dorsiflexors (C), which produces a torque about the ankles, moving the COP backwards and accelerating the body forwards. Having corrected the backward sway, the dorsiflexor activation ceases and at (D) the body is swaying forward at a constant velocity. At (E) the COP has moved anteriorly to the COG so in order to halt the forward sway the subject activates the plantar flexors until the COP once again lies close to the COG. To assess the ability of the central nervous system to control postural sway movements, both COP and COG should be measured.

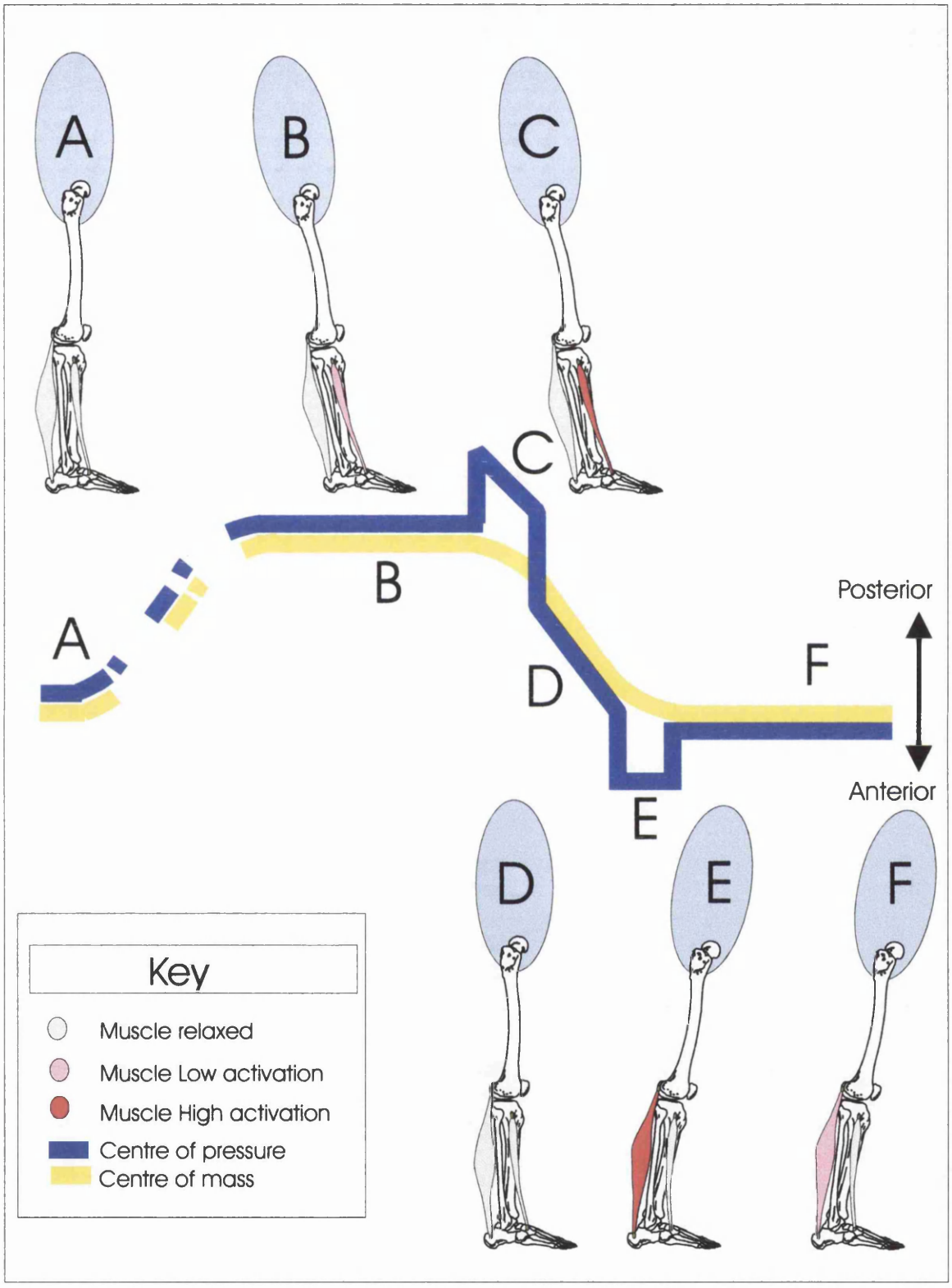



**Figure 1.1.1:** Diagrammatic representation of a subject standing on a force plate illustrating the actions of the neuromuscular system to control a backward sway of the body. A – F show the changing situation at six different points over time. See text for a full description.

*Determining the body's COG*

A limitation of assessing posture and balance by movements of the COG and COP is that while the COP is easily measured and quantified using a force plate, the COG is not directly accessible. Several investigators have attempted to compute the COG from force plate data by filtering out the higher frequency components present in the COP record (Shimba, 1984; Karlsson & Lanshammar, 1997; King & Zatsiorsky, 1997). Although these techniques may have some merit in certain clinical situations one drawback is that the COG and COP are not obtained independently. Other shortcomings include an inability to accommodate any phase difference between the COG and COP time histories and the problem of minimising differences in the COP based estimates of COG motion (Benda, Riley & Krebs, 1994). In addition, as all the methods of estimating COG position from COP data employ a filtering technique to eliminate the high frequency components these methods may not be suitable for analysing COP records from unstable subjects such as cerebellar patients for whom analysis of high frequency sway components can be important diagnostic tool (Diener, Dichgans, Guschlbauer & Mau, 1984).

With the development and more widespread use of commercial motion analysis systems it has become more common to measure the movements of the COG independently from those of the COP (Riley, Mann & Hodge, 1990; Benda *et al.* 1994; Hasan, Robin, Szurkus, Ashmead, Peterson & Shiavi, 1996). The most sophisticated data collection methods involve optical tracking systems, which follow the movements of markers positioned at specific anatomical landmarks. Using the co-ordinates of the markers, the location of a subject's whole body COG position can be found by modelling the body as a number of rigid segments, estimating the mass of each segment, and determining the body segments' position and orientation in space. The body segment kinematics used for estimating the COG position are compiled either from studies of cadavers (Dempster, 1955) or from a combination of in-vivo measurements with relationships based on cadaver studies (Hasan *et*

*al.* 1996). Although several assumptions and simplifications are employed when using a motion analysis system, they are fewer than those inherent in using COP movement data to compute the COG.

*The inverted pendulum model of postural sway*

The inverted pendulum model of postural sway has been used by many researchers to describe the movements of the body during quiet stance in the sagittal plane (Smith; 1957, Gurfinkel & Osevets, 1972). In this type of model the body is assumed to be a single rigid segment which rotates about the ankle joint. If the COG of the body is located exactly above the pivot of the ankles, then the system is perfectly balanced (Figure 1.1.2a). However, if the COG rotates, θ degrees from this position, then the weight of the body creates a torque at the ankles 'T' which will cause the body to fall over unless a torque is applied at the ankles in the opposite direction (Figure 1.1.2b). The amount of torque needed is given by the formula $T = m\ g\ h\ \theta$, where $m$ is the mass of the body, $h$ is the height of the centre of gravity above the axis of rotation of the ankle, $g$ is the acceleration due to gravity, $\theta \sim \tan \theta$ = angle of forward lean of the body.

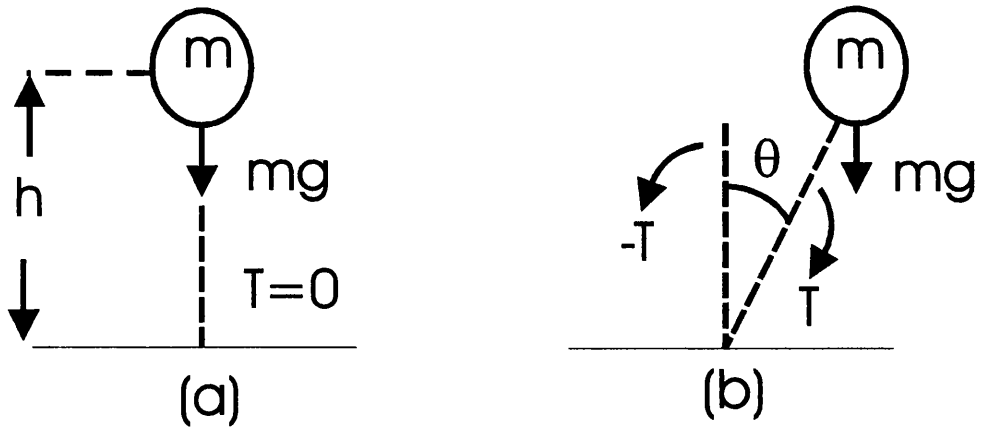



**Figure 1.1.2:** The body as an inverted pendulum, rotating about the ankles. a) T = 0, the COG is exactly above the pivot of the ankles, b) The torque (-T), needed to oppose the forward sway angle of θ degrees is given by T = m g h θ. -T can be measured from the forces acting on the support surface.

The majority of investigations into the postural strategies adopted during quiet stance have been confined to the sagittal plane and intuitively it may seem that the inverted-pendulum model is not applicable to the frontal plane because of the two points of support. Winter, MacKinnon, Ruder & Wieman (1993) used two separate force plates to record COP changes from the left and right feet independently and found that in the sagittal plane, left and right COP changes were closely synchronised indicating synergistic motor patterns of the ankle plantar-flexors and dorsi-flexors. However, in the frontal plane the left and right COP changes were anti-phasic and contributed almost nothing to the net COP changes. They concluded that in the frontal plane there was a loading and unloading of the hip joints during quiet stance and the muscle groups responsible were probably the hip abductors/adductors. Despite this difference in the control of postural sway in the respective planes, the lateral rotations at each ankle accompanied by lateral body shifts may still be likened to a pendulum system (Soames & Atha, 1980). Day, Steiger, Thompson & Marsden (1993) used a motion analysis system to record the three dimensional motion of eight markers placed at various sites on the body during quiet standing. They found that for both the sagittal and frontal plane, body markers situated furthest from the ground, such as those on shoulders, moved faster and further than the markers closer to the ground, such as those on the knees. This finding supports the application of an inverted pendulum model to postural sway in the frontal plane.

Winter, Prince & Patla (1997) suggested that as the body's COG is the weighted average of all the individual segments' COG, one validation of the inverted pendulum model would be for there to be a large positive correlation between the movement records of all the segments. They tested their hypothesis by using a 3-D motion analysis system to record the movement of the head and several other body segments (leg, thigh, pelvis and 4 trunk segments) during quiet stance, eyes open, in 10 young subjects standing with their feet at 3 different stance widths. Their results showed that the segments with the largest movement

such as the pelvis and trunk correlated best ($r$ >0.8) with the head in the A/P direction at all stance widths. In the M/L axis, at narrow stance widths (50% hip distance), leg, thigh, pelvis and trunk segment movements were also strongly correlated with head movement ($r$ > 0.8). The correlation coefficients obtained for the lower limb and pelvis in the M/L axis at stance widths of 100 and 150% hip width were lower and ranged from 0.40 to 0.67. The high positive correlation between the body segments suggests that there was a tendency for all the body segments to move in the same direction at the same time.

It has been suggested that if the body sways about the ankles as an inverted pendulum, the difference between the COP and COM is proportional to the horizontal linear acceleration of the COM (Geursen, 1976; Winter, 1995). Winter *et al.* (1997) were able to provide further support for the inverted pendulum model by demonstrating a high negative correlation between the horizontal acceleration of the COG and the difference between the COP and COG in both the sagittal and frontal planes ($r$ values between -0.82 and -0.92 depending on direction and stance width).

*The use of different postural strategies during standing*

It is generally accepted that when young, normal adults stand on a large, firm surface with their eyes open, they control random movements of their COG by swaying about the ankles like an inverted pendulum, using what is commonly known as the ankle strategy (Do, Breniere & Brenguier, 1982; Horak and Nashner, 1986). An ankle strategy is also used by young adults when recovering from small, slow support surface translations (Horak and Nashner, 1986) and under several conditions incorporating altered proprioceptive and/or visual feedback (Peterka & Black, 1990). When the vertical projection of the body's COG is near the end of the support area, such as when balancing on a narrow beam, a hip strategy will be used. The hip strategy repositions the body's COG by flexing or extending at the hips. If the perturbations of balance are large and the ankle and hip strategies are unable to

control the COG adequately, a stepping strategy will be used. In the stepping strategy, a series of rapid steps are used to realign the feet under an altered position of the COG. Figure 1.1.3 illustrates the three postural strategies commonly used by young adults.

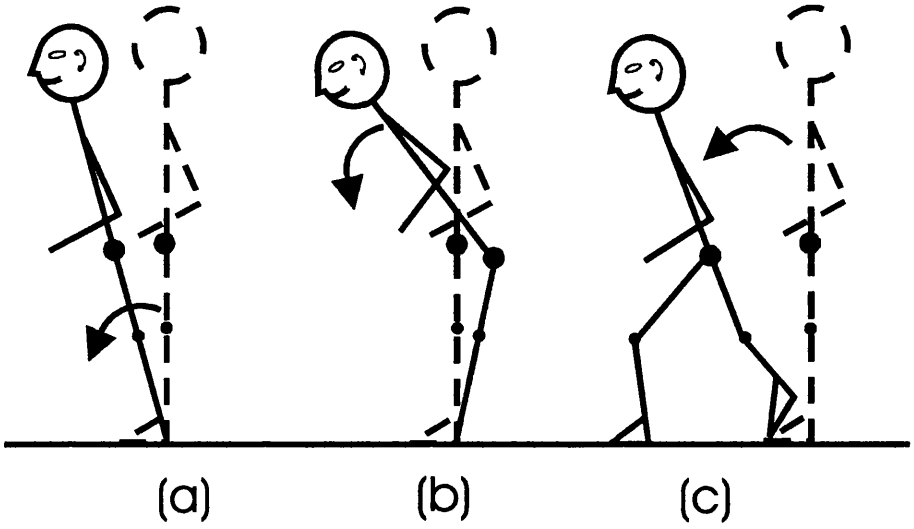



**Figure 1.1.3:** Stick figure diagrams illustrating the three postural strategies adopted by young adults to correct postural sway in the sagittal plane. a) Ankle strategy, b) Hip strategy and c) Stepping strategy.

Differences in the selection of specific postural strategies have been observed in several patient groups and older adults. For example, in older adults, there is a greater use of the hip strategy when recovering from small, slow translations on large support surfaces, a situation which would normally only require an ankle sway response (Horak, Shupert & Mirka, 1989; Manchester, Woollacott, Zederbauer-Hylton & Marin, 1989). Subjects with proprioceptive losses due to diabetic peripheral neuropathy have also been found to favour a hip strategy over an ankle strategy under these conditions (Horak, Nashner & Diener, 1990), as have patients with distorted or mixed types of vestibular disorders (Black, Shupert, Horak & Nashner, 1988). In contrast, patients with a vestibular loss rely exclusively on ankle sway to

control the COG and cannot use a hip strategy, even during balance tasks which require hip movements to maintain equilibrium, such as standing on one foot, standing or walking in a tandem Romberg position, or standing on a narrow beam (Fregly, Smith & Graybiel, 1973; Horak *et al.* 1990). Many elderly subjects also experience difficulty in carrying out balance tasks such as a tandem gait and standing on one foot, (Fregly *et al.* 1973). Although this inability to use a hip strategy may be due to a number of factors, an age-related loss of sensitivity in proprioceptive and/or vestibular function may be responsible.

The above findings suggest that while young normal adults will select a specific postural strategy according to the difficulty of the balancing task, in an elderly population a range of postural strategies may be observed for a single task depending on the neurological status of the individual. Therefore, when investigating postural sway as an indicator of balance dysfunction it is desirable not only to measure the range of postural sway but also the nature of the movements that constitute the sway.

## 1.2 Postural stability during walking

*Walking can be a difficult and dangerous activity for the older adult*

Many falls experienced by older adults occur during some aspect of walking, such as the initiation or termination, turning, or negotiating uneven surfaces (Sheldon, 1960; Overstall *et al.* 1977; Prudham and Evans 1981; Gabell, Simons & Nayak, 1985; Luukinen, Koski, Hiltunen & Kivelä, 1994; Berg, Alessio, Mills & Tong, 1997). Walking itself is an inherently unstable activity as for 80% of the stride period the body is in the single support phase during which time the body's COG falls outside the narrow confines of the stance foot (Shimba, 1984). In addition, the greatest proportion of the body mass is located 2/3 of the body height above the ground and therefore represents a large inertial load for the CNS to control if balance is to be maintained (Winter, Prince, Stergiou & Powell, 1993). Further requirements of walking include a safe forward progression and an accurately controlled foot trajectory during the swing phase of the gait cycle. Thus, the motor control demands of walking are much more complex than those of quiet stance.

There are several other important differences between walking and standing as far as control of stability is concerned. For example, the optic flow provided by focal and peripheral vision, which is critical to the control of many goal-directed walking activities, is absent during standing (Wolfson, Whipple, Amerman & Tobin, 1990). The vestibulospinal reflexes, which are involved in the regulation of body sway during stance, play only a minor role in balance recovery during walking (Dietz, Trippel & Horstmann, 1991). Furthermore, the stereotypical distal-to-proximal muscle activation sequence observed during perturbed stance (Horak & Nashner, 1986) is reversed during swing leg clearance of obstacles during walking (Patla, Prentice, Robinson & Neufeld, 1991).

*Age-related changes in walking patterns*

Studies of walking patterns in healthy elderly have found that the elderly walk more slowly than young adults and exhibit a shorter stride, necessitating a faster cadence for a given speed of walking (Finley, Cody & Finizie 1969; Murray, Kory & Clarkson, 1969; Hageman and Blanke, 1986; Blanke and Hageman, 1989). The reduced stride length and velocity have an influence on other gait parameters. For example, a reduced stride length can lead to an increased time spent in double-limb support, reduced arm swing, reduced rotation of the hips, knees and ankles and a more flat-footed heel-contact and toe-off (Murray & Clarkson, 1966; Murray, Sepic & Barnard, 1967; Kirtley, Whittle & Jefferson 1985). Elderly individuals who fall also tend to walk more slowly, have a shorter stride length, adopt a wider step width and spend a greater proportion of the gait cycle in double-limb support. (Guimares & Isaacs, 1980; Imms & Edholm, 1981; Tinetti, 1986; Gehlsen & Whaley 1990; Wolfson, Whipple, Amerman & Tobin, 1990). Slower walking speed, longer double support phase and increased step width have also been cited as adaptations which would increase stability and therefore reduce falls (Finley *et al.* 1969; Murray *et al.* 1969; Winter, Patla, Frank & Walt, 1990). It is possible that an elderly person may introduce these adaptations as a response to a previous fall or to awareness of reduced balance capabilities. Fear of falling is a common problem for the elderly, not only do 50% of individuals with a history of recent falls express a fear of falling but 33% of the elderly with no history of falls are concerned for their own safety (Tinetti, Speechley & Ginter, 1988; Downton & Andrews, 1990). By adopting a more stable gait pattern these 'fearful' elderly may reduce the likelihood of experiencing a fall. However, a fear of falling can also cause great anxiety and loss of confidence leading to a restriction of activity. A prolonged restriction of activity may lead to loss of muscle strength, joint flexibility and mobility and as a consequence, the risk of falling in these fearful elderly will increase, despite any stabilising gait changes they adopt. Maki (1997) demonstrated in a prospective study of spatial-temporal gait parameters in elderly subjects, that reduced stride length, reduced speed and increased double support

time were associated with a fear of falling ($P < 0.05$) rather than with falling ($p$ values from 0.23 to 0.74). Increased stride-to-stride variability in stride length, speed and double support was associated independently with falling and was not related to a fear of falling. Increased step width (medial-lateral separation between the two feet during double support) showed some evidence of an association with both fear of falling ($P = 0.09$), and falling while walking. A *decrease* in the variability of step width was also predictive of falling during walking. The combination of increased variability in walking speed and decreased variability in step width variation was able to predict fallers with 75% accuracy. This finding that a decrease in the variability of step width was able to predict falling during walking appears counter-intuitive and is in conflict with the suggestion of Gabell and Nayak (1984) that an increase in the variability of step width would indicate a lack of compensation for instability and a possible predisposition to falls. When attempting to explain their results Maki (1997) suggested that the reduced variability in step width was probably a consequence of the increased step width adopted by the elderly fallers. Although an increase in step width may help to increase stability during walking and at the same time reduce the energy costs associated with stride-to-stride adjustment of the lateral foot position (Saunders, Inman, & Eberhart, 1953), a wide step could also produce *instability* by increasing the lateral acceleration of the body's centre of mass (Mackinnon & Winter 1993).

Gehlsen and Whaley (1990) reported an association between increased mean heel width (lateral separation between left and right heels) and falling in a retrospective study comparing the gait patterns of elderly fallers and non-fallers walking on a treadmill. In contrast, Heitman, Gossman, Shaddeau & Jackson (1989) conducted a study of step width and balance in a group of elderly women with and without a history of falls and found no significant difference between fallers and non-fallers in mean or variability of step width (determined by the separation of linked ink footprints on a paper walkway). A low but significant negative correlation ($r$ values ranging from -0.16 to -0.26, $P < 0.05$) was found

between performance on a series of balance tests (one-legged stance and sharpened Romberg test) and the mean and variability of step width for the total subject group. These results suggest that an inability to control lateral movements of the body during static tasks is associated with difficulty controlling the lateral movements of the body while walking.

Although a definite association between increased step width or step width variability and falling is still in question, several retrospective studies have demonstrated that stride-to-stride variability in other spatial-temporal parameters of gait such as cadence, step length and stride time are associated with falling (Guimares & Isaacs, 1980, Gabell & Nayak 1984, Lord, Lloyd & Li, 1996; Hausdorff, Edelberg, Mitchell, Goldberger & Wei 1997). Hausdorff *et al.* (1997) used an ankle-worn foot-switch system to measure stride-to-stride variability in the temporal parameters of the gait cycle during a 6-minute self-paced walk in relatively healthy, community-living elderly men and women and young healthy subjects. When the elderly subjects were divided into two groups based on fall history, all measures of gait variability (stride time, stance time, swing time and % stance time) were significantly larger in the fallers compared to the non-fallers ($P = 0.002$).

Increased stride-to-stride variability in older fallers may indicate an increased risk of falling during walking as a result of errors in control of foot-placement and/or centre of mass displacement. Robbins, Waked, McClaren & Krouglicof (1997) investigated foot position awareness in a group of 13 young men aged 28.13 ± 3.96 years (mean ± S.D.) and a group of 13 older men aged 72.58 ± 4.50 years, in good health. Subjects were asked to walk along a 9 m balance beam resting on the floor, in bare feet and wearing shoes with different midsole thickness and hardness. The number of falls per 100 m of beam walking was recorded and the rear-foot angle was measured using an optical position measurement system. Subjects were also asked to estimate the maximum perceived supination when beam walking. Position error was calculated by subtracting perceived maximum supination from maximum

supination inferred from the rear-foot angle measurements. Foot position awareness (reciprocal of foot position error) was found to be 200% poorer in the older men who underestimated actual foot supination under all conditions. The absolute mean error for the young men was 5.44°, S.E.M. ± 1.03° and for the older men was 15.48 ± 1.56° ($p < 0.001$). For the older men there was a strong correlation between absolute mean estimate error and maximum supination ($r = 0.901$, $p < 0.001$) and a positive relation between absolute mean error and balance failure ($r = 0.367$, $p < 0.001$). Performance (position error and balance failure) was better when shoes with thin, hard soles were worn compared to shoes with thick, soft soles. Loss of foot position awareness in elderly subjects may affect the control of foot placement while walking. In addition, two recent studies have reported reduced abilities in older adults to control movements of their centre of mass. Yack and Berger (1993) studied the frequencies present in records of upper body acceleration during walking in a group of young subjects and in a group of elderly subjects. The elderly subjects were classified as stable or unstable according to their own perception of unsteadiness and their history of falls. Yack and Berger (1993) found that in the A/P and vertical direction elderly subjects with stability problems had more within-stride variation of acceleration measures. They suggested that these within stride variations of acceleration measures reflected inconsistencies in the forces being applied to maintain trunk stability. In the M/L direction, although there was a trend towards increased within-stride variation in unstable elderly, the difference between the groups was not significant. A possible explanation for the lack of significant findings in the M/L direction could be that the accelerometer measures were not sensitive to the frequency of the M/L movements.

*Use of motion analysis systems in gait and balance assessment*

Traditionally, assessments of mobility and balance in the elderly include measures of the ability to walk short distances, turn, rise from a chair or bed, the Romberg test and tandem walking. A limitation of these methods is that the measures obtained are rather qualitative

and dependent upon grading of performance by trained staff. Podsiadlo and Richardson (1991) attempted to make the assessment more quantitative and less time-consuming by simply measuring the time taken to perform the 'Get-up-and-go Test developed by Mathias, Nayak & Isaacs (1986). Patients capable of performing the test in less than 20 seconds were found to be independently mobile and those requiring more than 30 seconds to complete the task were immobile or mobile only with assistance. Gait parameters such as stride length and gait velocity have also been measured and found to correlate with falling and with performance on the Get Up and Go Test (Mathias *et al* 1986). Although these measures may be useful indices of performance, they provide limited insight into the changes in the motor system responsible for impaired balance control.

With the advent of gait laboratories making use of 3-D motion analysis systems and force plates, objective measurements of body segment movement, joint rotation and ground reaction forces has been made possible. In general, only minor changes in the joint angle profiles between young and elderly have been observed (Winter 1991, Oberg *et al.* 1994). Changes in the amplitude of joint angles have been found, including a reduced peak plantar flexion angle with a more toeing-out foot angle (Judge, Davis & Õunpuu, 1996). The heel skid contact velocity is significantly higher in elderly subjects (1.15 m/s) compared to young subjects (0.87 m/s) even though the velocity of walking is lower (Winter, 1991). A possible consequence of higher horizontal heel velocity may be an increased risk of slipping. Older adults also have a reduced push-off power and a more flat-footed landing compared to the young (Winter, 1991; Judge *et al* 1996).

In this thesis the aim is *not* to investigate age-related changes in spatial-temporal gait parameters or joint angles, but to examine variability in the movements of the body during walking. The validity of the inverted pendulum model to movements of the body during walking will also be investigated.

## 1.3 *Is there a relationship between decreased muscle strength and postural instability?*

It is well documented that muscle strength decreases with age (Larsson, Grimby & Karlsson 1979; Aniansson, Grimby & Rundgren 1980; Aniansson, Sperling, Rundgren & Lehnberg 1983; Vandervoort & McComas 1986; Whipple, Wolfson & Amerman 1987; Bruce, Newton & Woledge 1989; Phillips, Bruce, Newton & Woledge 1992) and it is also well recognised that postural stability declines with age (Sheldon, 1963, Bohannon, Larkin, Cook, Gear & Singer 1984; Woollacott, Shumway-Cook & Nashner 1986; Briggs, Gossman, Birch, Drews & Shaddeau 1989). In addition, both reduced muscle strength (Blake *et al.* 1988; Whipple *et al.* 1987, Campbell, *et al.* 1989, Luukinen, Koski, Laippala & Kivelä 1997) and increased postural sway while standing (Overstall *et al.* 1977; Campbell *et al.* 1989; Maki, Holliday & Topper, 1994) have been identified as potential risk factors for falling in the elderly. However, the relationship between muscle strength and postural sway has not been fully investigated. It is not known whether individual measures of muscle strength are related to the ability to maintain balance under a variety of conditions, and how this relationship is affected by age. One of the aims of this thesis was to investigate the relationship between muscle strength and postural sway during standing and walking in independent, community-dwelling older adults.

*Age-related changes in muscle strength*

The decline in muscle strength with age can be partly explained by muscle atrophy (Grimby & Saltin 1983; Essen-Gustavsson & Borges, 1986; Vandervoort & McComas, 1986; Kallman, Plato & Tobin, 1990) but there is also evidence of a reduction in maximum voluntary isometric force per cross-sectional area of muscle (MVF/CSA) with ageing (Young, Stokes & Crowe, 1985; Bruce *et al.* 1989; Klitgaard *et al.* 1990b; Phillips *et al.* 1992). The muscle atrophy is probably due to both a loss of fibre number and a reduction

in fibre size, with type II fibres undergoing the greatest atrophy and lesser changes taking place in the type I fibres (Grimby & Saltin, 1983, Young *et al.* 1985, Lexell, Taylor & Sjostrom, 1988; Klitgaard *et al.* 1990b). The cause of reduced MVF/CSA in the elderly is still under debate. A change in the fibre-type composition of muscle with ageing could account for some of the decrease in MVF/CSA (Maughan, Watson & Weir, 1983; Young *et al.* 1985) but definitive studies have not been conducted. Two quantitative investigations have reported an unusually large incidence of type I fibres in muscle cross-sections of vastus lateralis and tibialis anterior in elderly subjects (Larsson 1983; Jakobsson, Borg & Edström, 1988). However the majority of studies have not shown significant alteration in fibre-type proportions with age (Grimby, Danneskjold-Samsøe, Hviid & Saltin, 1982; Lexell *et al.* 1988). Using monoclonal antibodies to myosin heavy chains (MHCs), Klitgaard, Zhou, Schiaffino, Betto, Salviati & Saltin (1990c) were able to demonstrate in vastus lateralis muscles from elderly men an unusually high incidence of fibres containing more than one MHC isoform. For example coexistence of type IIA and IIB isoforms and increased coexistence of type I isoforms with those of type IIA. The coexistence of myosin isoforms within a fibre may be related to a transformation process where fibres change from one histochemical type to another. The explanation for this phenomenon is at present unknown but an increased coexistence of MHC isoforms is also observed in human skeletal muscle after endurance training (Klitgaard *et al.* 1990a) and thus may reflect a changed activity pattern with ageing. Denervation of large fibres and a reinnervation by smaller motor neurones (Grimby & Saltin 1983) or changes at the neuromuscular junction (Gutmann & Hanzlikova 1972) could also be responsible for inducing changes within the ageing muscle fibres. Another possibility is disuse, as regular usage seems to maintain morphology, fibre size and MHC expression in ageing human muscle (Klitgaard *et al.* 1990b).

The relative amount of connective tissue in skeletal muscle increases with age (Alnaqeeb, Zaid, & Goldspink, 1984), but the magnitude of the increase is not large enough to

significantly affect the quantity of non-contractile tissue in cross-section. Consequently, connective tissue content has little impact on the estimation of MVF/CSA (Brooks & Faulkner, 1993). The fact that the weakness in old muscle is not observed when the active muscle is stretched (i.e. during eccentric contractions) in mice (Phillips, Bruce & Woledge, 1991) and humans (Vandervoort, Kramer & Wharram, 1990; Poulin, Vandervoort, Paterson, Kramer & Cunningham, 1992; Phillips, Rowbury, Bruce & Woledge, 1993c; Hortobagyi, Zheng, Weidner, Lambert, Westbrook & Houmard, 1995) suggests that all the cross-bridges are present and active. This eliminates the possibility that the weakness is due to a reduction in the amount of functioning contractile material per CSA, because in such a case stretch force would also be proportionally lower. Phillips *et al.* (1993c) have suggested that the reduced isometric force but normal stretch force may result from a shift in the equilibrium between high force and low-force producing cross-bridge states. This idea was first suggested by Pate and Cooke (1989) to explain the effects of inorganic phosphate on force. Lombardi and Piazzesi (1990) suggested that when the muscle is being rapidly stretched all the cross-bridges are constrained into a common state eliminating the effect of differences in the force producing state of the crossbridges.

Obtaining an estimate of the MVC/CSA is essential for comparing the force generating capabilities of the muscle between individuals of different ages and sizes. If only MVF is measured than it is impossible to determine whether any change in force is due to a change in the number of force producing units (e.g. atrophy) or to a change in the amount of force that each unit exerts. The force developing capacity of skeletal muscles is proportional to the total muscle fibre CSA measured at right angles to the individual fibres contributing to the force. Computerised Tomography has been used to estimate the CSA of the quadriceps by measuring the whole muscle CSA normal to the muscle's axis (Maughan, Watson & Weir, 1983). However, the complex multipennate structure of this muscle group means that this measure of CSA is not the same as the summated CSA of all the fibres and therefore

may not be a good estimate of its force producing capabilities. The architectural complexity of the quadriceps muscle group may partly explain why Maughan *et al.* (1983) found only a weak correlation between quadriceps MVF and CSA in young women ($r = 0.51$, $p < 0.01$) and men ($r = 0.59$, $p < 0.01$) and Young, Stokes & Crowe (1985) found no significant correlation ($r = 0.015$) between quadriceps MVF and CSA in 12 young men. Changes in muscle bulk due to hypertrophy leads to a change in the angle of pennation of the fibres (Binkhorst & van'T Hof, 1973) and this may also contribute to non-linearity in the relation between force and CSA for the quadriceps (Huxley, 1980). In comparison to the quadriceps, the adductor pollicis muscle shows a strong correlation between MVF and CSA ($r = 0.926$) (Bruce, Phillips & Woledge, 1985). Bruce *et al.* (1985) obtained a profile of the hand in the plane that bisects the adductor pollicis using an apparatus consisting of 2 potentiometers mounted in a light frame which was moved over the hand while the position was monitored by a third potentiometer. Standard texts on the anatomy of the hand describe the adductor pollicis as a fan-shaped muscle arising by oblique and transverse heads. However the muscle is approximately parallel fibred at the point where the measurements are made so the problems associated with measuring CSA in the multipennate quadriceps are not encountered. The CSA measured by this method is also well correlated ($r = 0.937$) with measurements of muscle CSA obtained from CT and NMR images through the same plane (Bruce, Newton & Woledge, 1989). The technique used for obtaining force measurements minimises the differences in lever ratio between individuals because the point of opposition of the force at the base of the proximal phalanx of the thumb is close to the attachment of the muscle. Force measurements of the quadriceps are usually taken at the ankle, which is some distance from the muscle insertion, hence the absolute force generated by the muscles is greater than the measured force. In addition, as the adductor pollicis muscle consists almost entirely of type I fibres (Round, Jones, Chapman, Edwards, Ward & Fodder, 1984), the force measurements were unlikely to have been affected by the type II fibre atrophy associated with ageing (Grimby & Saltin 1983; Young *et al.* 1985; Lexell *et al.* 1988;

Klitgaard *et al.* 1990b). These factors contribute to the better correlation between force and CSA obtained for the adductor pollicis (Bruce *et al.* 1985, 1989) compared to those published for quadriceps (Maughan *et al.,* 1983; Young *et al.* 1985).

Bruce *et al.* (1989) and Phillips *et al.* (1992) have demonstrated a decrease with age in the ability of the adductor pollicis to produce force. Bruce *et al.* (1989) measured MVF and CSA in a group of 23 healthy elderly subjects aged 76 to 94 years (mean age 81.3 years) and compared their results with those of a group of 55 young subjects aged 18 to 54 years (mean age 29 years). The ratio of MVF/CSA for the elderly subjects was found to be 27 ± 4% (mean ± S.E.M.) lower than that observed for the young subjects. Phillips *et al.* (1992) demonstrated a similar reduction (26 ± 3%, mean ± S.E.M) in the MVF/CSA of the adductor pollicis in an active, independent group of 39 elderly subjects aged 74 - 90 years (mean = 80 years). This reduction in MVF/CSA observed in the elderly was not due to an inability to fully activate the muscle as the results from a twitch interpolation experiment showed that even those elderly with reduced MVF/CSA measurements were fully activated during a maximal voluntary contraction (Phillips *et al.* 1992).

*Influences of reproductive hormones on muscle force*

The menopause is defined as a loss of ovarian function, characterised by very low concentrations of oestrogen and progesterone (Whitehead & Godfreee, 1994). This hormonal change has been associated with a significant reduction in MVF/CSA in post-menopausal women for the adductor pollicis muscle (Phillips, Rook, Siddle, Bruce & Woledge, 1993b; Phillips *et al.* 1993c) and the quadriceps (Rutherford & Jones, 1992). Administration of hormone replacement therapy (HRT) can prevent the loss of MVF/CSA associated with the menopause (Phillips *et al.* 1993b). In a cross-sectional study comparing MVF/CSA in the adductor pollicis in post-menopausal users and non-users of HRT, Phillips *et al.* (1993b) showed that in non-users, MVF/CSA declined dramatically in the five years or

so after the menopause compared with a gradual decline from about 60 years in men. In women using HRT from the time of their menopause no decline in MVF/CSA was observed. In non-users, the force in eccentric contractions of adductor pollicis was spared relative to isometric contractions. This demonstrates that the decline in isometric force observed following the menopause is not due to a decreased number of crossbridges but is probably due to a change at the level of the crossbridge itself as postulated by Phillips *et al.* (1993c). How oestrogen is able to influence the crossbridge state has yet to be determined.

Studies on cyclic changes in MVF/CSA of adductor pollicis during the human menstrual cycle (Phillips, Gopinathan, Meehan, Bruce & Woledge, 1993a) demonstrate that although the changes in MVF follow the same pattern as that of urinary oestrogen excretion (Moghissi, Syner & Evans, 1972) there is no significant correlation between plasma oestrogen levels and force measured on the same day. This suggests that the action of oestrogen on the muscle is not immediate and a phase lag exists between the changes in oestrogen level and force. McGoldrick *et al.* (1998) studied changes in force during the oestrus cycle in muscles excised from young female mice. They found that normalised isometric muscle force was dependent upon the phase of the oestrus cycle but the rise in force did not follow the rise in blood oestrogen as it does in humans. The force was greatest during dioestrus when hormone levels were lowest. In muscle that had been kept in Ringer solution for approximately 2 hours before testing, the effects of the oestrus cycle on force production were diminished and in all phases apart from dioestrus the muscle was as strong or stronger than fresh muscle. From these results McGoldrick *et al.* (1998) concluded that an inhibitory substance which was dependent on the phase of the oestrus cycle was washed out of the muscle while it was kept in Ringer solution. The increase in force seen during the human menstrual cycle is therefore probably due to oestrogen antagonising an inhibitory effect on the muscle.

*Influences of reproductive hormones on postural sway*

Post-menopausal oestrogen therapy appears to reduce the risk of hip fracture in older women (Cummings, Kelsey, Nevitt & O'Dowd, 1985; Kiel, Felson, Anderson, Wilson & Moskowitz, 1987) and has been shown to retard bone loss (Lindsay, Hart, Aitken, MacDonald, Anderson & Clarke, 1976; Horsman, Gallagher, Simpson & Nordin, 1977; Jensen, Christiansen & Transbϕl, 1982; Horsman, Jones, Francis & Nordin, 1983). However there has been a suggestion that the fracture-protective effect of oestrogen is mediated by systems or functions that are more rapidly affected by oestrogen than bone mass. Cauley, Seeley, Ensrud, Ettinger, Black & Cummings (1995) showed that oestrogen had an effect on fracture risk even after adjustment for bone mineral density. It is possible that maintenance of muscle strength may help prevent a hip fracture. For example Phillips *et al.* (1998) demonstrated that hip fracture patients produce substantially lower MVF/CSA in the adductor pollicis muscle, 4 and 18 days post-operatively than healthy elderly women. It has also been suggested that oestrogen therapy may influence balance function and therefore play a role in preventing a hip fracture (Naessen, Persson, Adami, Bergstrom & Bergkvist, 1990). In a recent study, Naessen, Lindmark & Larsen (1997) compared the postural sway velocities of 16 long-term users of 17β-estradiol implants (mean age 67.9 years) with those of 16 age-matched nonusers. Sway velocities were significantly lower in oestrogen users than in non-users ($p = 0.0067$) and similar to those in young pre-menopausal women. The difference between HRT users and non-users was accentuated by blindfolding and by increasing frequencies of vibration to the calf muscle. Serum levels of estradiol and estradiol/sex hormone binding globulin were negatively correlated with sway velocity ($r = -0.5$, $P = 0.0036$). Contrasting results were reported by Armstrong, Obourne, Coupland, Macpherson, Bassey & Wallace (1996) after conducting a randomised controlled trial on the effect of oral HRT. They found there was no significant change in the body sway (measured in degrees) of HRT users following 24 weeks of HRT treatment compared to the control group. After 48 weeks of treatment there was also no significant difference in grip strength

between the two groups. Part of this conflict between the results obtained by Armstrong *et al.* (1996) and those obtained by Naessen *et al.* (1997) for postural sway and Phillips *et al.* (1993c) for muscle force, may be due to the fact that many of the participants in the Armstrong study, both test and control subjects, had pre-menopausal oestradiol levels (> 150 pmol/l) at the start of the trial and hence even women receiving HRT showed a decline in oestradiol level after 48 weeks. A scatter plot of individual percentage changes in oestradiol levels and handgrip strength (Figure 1., page 688, Armstrong *et al.* 1996) showed that approximately 40% of the test group had less than a 20% increase in oestradiol levels. The majority of subjects whose oestradiol levels increased by more than 20% did show an increase in grip strength. Whether the test subjects with increased oestradiol levels also had improved sway measures could not be ascertained from the data presented. It has been suggested that cross-sectional studies on the effects of HRT may be biased, as HRT users are known to be thinner and to undertake more physical activity than nonusers (Barrett-Connor, 1991). However, there was no significant difference with regard to body weight or walking activity between the HRT users and non-users in the Naessen *et al.* (1997) study.

*The effects of reproductive hormones on the central nervous system*

How oestrogen therapy influences balance function is uncertain. There is some evidence that oestrogen acts directly on the central nervous system by facilitation of neuronal transmission (Smith, 1989). Oestrogen has also been shown to alter the auditory brain response (ABR) in young women (Elkind-Hirsch, Stoner, Stach & Jerger, 1992a; Elkind-Hirsch, Wallace, Stach & Jerger, 1992b) and thus may alter vestibular reflex functions. When the ABR latencies from 9 young, normally cycling women were compared with 9 young women on oral contraceptives (Elkind-Hirsch *et al.* 1992a) there was a significant lengthening of wave V peak latencies during the mid-cycle oestrogen peak in the normally cycling women. The origin of the latency change was attributed to the central auditory neural pathways as wave V reflects central conduction time. The authors suggested that oestrogen enhanced secretion

of the inhibitory neurotransmitter gamma-amino-butyric acid (GABA) at auditory nerve synapses and this lead to the delayed synaptic conduction time. Oestrogen treatment of adult ovariectomized rats has also been shown to induce GABA receptors in motor areas such as cortex, striatum and cerebellum (Perez, Zucchi & Maggi, 1986).

Administration of 17$\beta$-oestradiol has also been found to enhance glutamate-induced excitatory responses in rat cerebellar Purkinje cells (Smith, 1989). The cerebellum is thought to be a CNS locus for motor learning (McCormick and Thompson, 1984) and co-ordination (Brooks and Thach, 1981), behavioural parameters which are improved in the presence of high circulating oestradiol (Beatty, 1979; Becker, Synder, Westgate & Jenuwine, 1987; Hampson and Kimura, 1988). For example, Hampson and Kimura (1988) described improved performances on tests of speeded motor co-ordination in women during the mid luteal phase (high levels of oestradiol and progesterone) relative to the performance during menses. Enhanced performances on tests of articulatory and fine motor skills during the late follicular phase (high oestradiol levels) have also been reported (Hampson, 1990).

*The relationship between muscle strength and postural sway*

There have been no other studies investigating the relationship between MVF/CSA and postural sway in active independent older adults. There have been a few reports on the relationship between muscle force and postural sway but the subjects in these studies have tended to be frail, institutionalised elderly. For example, Lord, Clark & Webster (1991) examined the relationships between isometric quadriceps and ankle dorsiflexor strength and body sway in 95 elderly persons (mean (SD) age = 82.7 (6.6) years) living in a hostel for the aged. No significant correlation was found between quadriceps strength/height or ankle strength/height and body sway when the subjects stood on a firm surface (values for $r$ ranged from 0.09 - 0.18). However when subjects stood with their eyes open on a compliant surface which reduced peripheral sensation, there was a low but significant negative

correlation between body sway and ankle dorsiflexion strength ($r$ = -0.24, $P < 0.05$). A low but significant negative correlation was also observed between body sway with eyes closed on the compliant surface and quadriceps and ankle dorsiflexion strength ($r$ = -0.23 and -0.25 respectively, $P < 0.05$). Era and Heikkinen (1985) measured mean sway amplitude and frequency in men aged between 31 and 75 years, along with muscle forces for the trunk (flexion and extension), arm flexion, knee extension and grip. Grip strength was the only force measure that was significantly correlated with sway, eyes open ($r$ values ranging from -0.222 (31-35 years) to -0.55 (71 - 75 years), $P < 0.01$). Sway with the eyes closed was also significantly correlated with grip strength but only in the oldest age group (71 - 75 years, $r$ = -0.211, $P$ = -0.05). In contrast to the previous studies, Hughes, Duncan, Rose, Chandler & Studentski (1996) found a low positive correlation between sway (measured as sway area) and measures of both isokinetic and isometric strength ($r$ values ranging from 0.24 to 0.35). The subjects in this study were 100 community dwelling elderly (mean age 77.2, range 66 - 96 years) with moderate functional impairment (unable to climb stairs step over step).

For the three studies described above the correlation between muscle force and sway is very low suggesting that if there is an association between strength and sway, it is only weak. What level of association exists between the MVF/CSA in the adductor pollicis and postural sway during standing in the elderly is also worth investigating. Postural sway measures will be made under conditions of varying sensory input (visual and somatosensory) as there is some evidence that older adults are less stable when sensory inputs are absent or conflicting (Woollacott, Shumway-Cook & Nashner, 1986; Lord *et al* 1991). The degree of association between MVF/CSA and postural stability during walking will also be investigated.

## 1.4 Recovery responses to a trip

Tripping is one of the most prevalent causes of falls in the elderly (Overstall *et al.* 1977; Blake *et al.* 1988; Tinetti & Speechley, 1989; Campbell, Borrie, Spears, Jackson, Brown & Fitzgerald, 1990). Trips that occur during walking are usually triggered when the swing foot strikes an obstacle in its path and will generally lead to a forward-directed fall. To prevent a trip becoming a fall, inputs from sensory receptors must be rapidly integrated to generate an appropriate response to return the body to a more stable position and allow the safe continuation of the locomotor pattern.

Hazardous obstacles that can induce a trip are found both indoors and outdoors and vary in height from a few millimetres, in the case of a crack in a pavement, to more than 15 cm for a curb. Defects in flooring, electrical cords, door thresholds, and exposed tree roots may also be responsible for initiating a fall. Depending on a number of factors including illumination, distractions and attention, older people may or may not be aware of potentially hazardous obstacles (Archea, 1985; Owen, 1985). Changes in the visual system with age could also be instrumental in causing trips among older adults. When full visual capacity is present, individuals can spot obstacles easily and walk around them but an age-related constriction of the visual field may obscure hazards from view resulting in a trip (Berg, Alessio, Mills & Tong, 1997). A reduction in the height of the foot during the swing phase of gait has been observed in older adults (Kaneko, Morimoto, Kimura, Fuchimoto & Fuchimoto, 1991) and this factor could potentially increase susceptibility to tripping, particularly on uneven surfaces. However, Winter, Patla Frank & Walt (1990) found no statistical difference in toe clearance between young adults and healthy, active elderly subjects. In addition, Gehlsen and Whaley (1990) found no significant difference in toe or heel height between elderly with a history of falling (experienced a fall ten months or less before testing) and non-fallers during treadmill walking.

The number of investigations into the biomechanical and/or neuromuscular responses subsequent to a simulated trip is limited. Cutaneous electrical stimulation has been used extensively in the past to produce a disturbance of the locomotor pattern in both animals and man. In cats, electrical stimulation of receptors in the lower limbs during locomotion produce an excitation of the extensors during the stance phase and an excitation of the flexors during the swing phase (Forssberg 1979; Forssberg, Grillner & Rossignol, 1977; Duysens, Loeb & Weston, 1980; Gauthier and Rossignol 1981; Drew and Rossignol 1987). The flexor excitation elicited by cutaneous electrical stimulation of the swing limb results in a removal of the limb from the stimuli. Forssberg (1979) called this reflex pattern the 'stumbling corrective reaction' because the action assists the animal maintaining its balance despite the perturbation and is integrated within the step cycle. Recording of electromyographic (EMG) responses after electrical stimulation in humans has shown that these reflexes are task dependent e.g. an increased amplitude of cutaneous reflexes is observed during running as compared to standing (Duysens, Tax, Trippel & Dietz, 1993), intensity dependent (Belanger & Patla, 1984; Crenna & Frigo, 1984), and phase dependent, e.g. tibialis anterior has an excitatory response during early swing but an inhibitory response during late swing (Duysens, Trippel, Horstmann & Dietz, 1990; Yang & Stein 1990).

The fact that the EMG responses to the stimulation arise bilaterally with a latency of 90 ms (Berger, Dietz & Quintern, 1984), which is more than 50 ms longer than the H-reflex latency, makes it unlikely that they represent monosynaptic stretch reflexes. In addition, it has been widely reported that the monosynaptic reflexes are suppressed during gait (Morin, Katz, Mazieres & Pierrot-Deseilligny, 1982; Berger *et al.* 1984; Dietz, Quintern & Berger, 1985). Berger *et al.* (1984) reported that ischaemic block of group I afferents did not affect the corrective responses to a perturbation and suggested that responses were mediated primarily by group II and III afferents in a polysynaptic spinal pathway. The observation that the polysynaptic spinal reactions are reduced or absent in patients with spastic paresis

due to a supraspinal lesion of the motor system suggests that the responses are dependent on supraspinal control (Berger, Horstmann & Dietz, 1988; Dietz and Berger, 1984).

Although Forssberg (1979) demonstrated that cutaneous electrical stimulation and mechanical stimulation of the paw of the cat resulted in similar EMG responses, others have reported that the responses from a mechanical stimulus are more powerful and complex than those evoked from electrical stimulation (Drew and Rossignol, 1987). The difference in response may be due to convergence from different types of receptors. With a mechanical perturbation there is a physical obstruction to overcome and a genuine threat of falling if the task is not completed successfully. Lundberg (1979) has proposed a flexor reflex afferent model (FRA) with different receptor function (e.g. cutaneous, muscle) converging on common interneurons in reflex pathways to explain the observed difference in magnitude of response between cutaneous and mechanical stimulation.

One of the reasons that the responses to a mechanical perturbation have seldom been examined during human locomotion is that it is difficult to control the exact timing and magnitude of the perturbation. Nashner (1980) used longitudinal, vertical and rotational movements of a platform incorporated into a 4-m walkway to perturb subjects during the stance phase of gait. Berger *et al.* (1984) and Dietz Quintern & Berger (1984) also produced perturbations of the stance phase of gait by using randomly timed, short acceleration or decelerating impulses applied to a treadmill. However the majority of naturally occurring stumbles occur during the swing phase when the foot strikes an unexpected obstacle. Grabiner, Koh, Lundin & Jahnigen (1993) produced a stumble in young male subjects (mean age 27.1 years) by manually raising a mechanical obstacle as the subject walked through the test environment. Modified spectacle frames were used to restrict the subject's visual field so that they were unaware of the raised obstacle. A motion analysis system was used to record kinematic data of the response to the perturbation. The results demonstrated that

there were substantial increases in trunk flexion, hip flexion and knee flexion following the perturbation. Disadvantages of this experimental protocol include the possibility that the subjects were able to anticipate the obstacle and the restricted visual field may have led to an abnormal response. In addition the protocol was deemed unsuitable to apply to healthy or frail elderly subjects, the populations of interest.

Eng, Winter & Patla (1994) also used the raising of obstacles set into a walkway to perturb the gait of young healthy men (19 - 28 years). The raising of the obstacles was triggered by weight acceptance on a force plate. This automation enabled the gait to be perturbed at two distinct phases in the gait cycle (approximately 20% and 60% into the swing phase). EMG activity was recorded from 16 muscles in the leg and trunk and video cameras were used to record kinematic data. Their results demonstrated that in general there was an elevating strategy adopted in response to the early-swing perturbation and a lowering strategy in response to the late-swing perturbation. Each of these two strategies was associated with a distinct muscular activation profile.

Chen, Ashton-Miller, Alexander & Schultz (1991) investigated age differences in the strategy used for stepping over fixed obstacles of different heights. Healthy volunteers (N = 48) were divided into 4 groups of 12 subjects: young adult females (age 21.7 ± 2.1 years, mean ± S.D), young adult males (21.5 ± 2.0 years), old females (71.2 ± 5.5 years) and old males (71.3 ± 4.5 years). A motion analysis system was used to record approach and obstacle crossing speeds as well as bilateral lower extremity kinematic parameters. No age differences were found in obstacle-free gait but older adults exhibited a significantly more conservative strategy when crossing obstacles, with slower crossing speed, shorter step length and a shorter distance between the obstacle and the heel strike of the crossing foot. Four of the 24 elderly subjects stepped on the obstacles due to this shortened stride length and obstacle-heel strike distance. More recently Chen, Ashton-Miller, Alexander & Schultz

(1994) made an attempt to improve the safety aspect of the test by using a band of light to produce a 'virtual' obstacle on the walkway at the predicted location of the next footfall. The risk of contact was compared for young and elderly adults. The available response time before heel strike was varied randomly in 50 ms increments between 200 and 450 ms. In general, older subjects were less successful than young adults at avoiding the virtual objects although the differences were not significant for any particular response time. Chen, Schultz, Ashton-Miller, Giordani, Alexander & Guire (1996) also studied the effects that attention-dividing tasks had on risk of contact. Both young and old adults had a significantly increased risk of obstacle contact when their attention was divided but the obstacle avoidance score for the elderly was significantly decreased compared to the young adults under these conditions.

An alternative protocol for perturbing the swing leg has been adopted by other investigators and involves applying a momentary resistance to a cord attached to the swing leg while subjects walk on a treadmill (Garrett & Luckwill 1983; Dietz, Quintern, Boos & Berger, 1986; Ghori & Luckwill, 1989). Reflex responses in the lower limbs were investigated using both EMG and kinematic techniques. A number of different responses were observed depending on where in the gait cycle the resistances were applied (Dietz *et al.*, 1986; Ghori & Luckwill, 1989). Dietz *et al* (1986) used a torque motor to apply perturbation impulses of between 20 and 160 ms duration to the swing leg. They observed that when the resistance was applied at the beginning of the swing phase, a weak rectus femoris response appeared in the swinging leg and responses were also recorded from gastrocnemius and biceps femoris of the contralateral, standing leg. When the resistance was applied at the end of the swing phase, strong tibialis anterior and rectus femoris responses appeared in the swing leg and gastrocnemius and biceps femoris responses in the standing leg. The latency between onset of the resistance and the response in the leg muscles was 65-70 ms in both legs. The apparatus used by Ghori and Luckwill (1989) consisted of a loop of copper wire attached

between a strap around the subject's ankle and a nylon string passing around the pulley of an inertial clutch mounted on a motor. The resistance occurred in response to brief activation of the motor that caused clamping of the nylon string to the pulley and operation of the inertial clutch, resulting in a sharp backward pull to the leg. The result of this was that the copper wire loop broke quickly after applying a momentary resistance to the moving leg. In response to the perturbation it was found that the ipsilateral leg muscles (gluteus medius, vastus lateralis, rectus femoris and tibialis anterior) produced a response throughout the step cycle regardless of whether the muscle was silent or active when the perturbation was applied. In contrast, contralateral leg muscles showed a different pattern of response to perturbations applied during early swing compared to late swing. The latency of the responses was approximately 80 ms, suggesting that the supra-spinal pathways thought to be responsible for the responses to cutaneous stimulation may also be involved in the responses to mechanical perturbation of gait

Although the experimental protocol used by Dietz *et al.* (1986) and Ghori & Luckwill (1989) to perturb the swing phase of gait may be more artificial than using raised obstacles on a walkway, there are several advantages to the above protocol. For example, the timing and magnitude of the perturbations can be controlled fairly accurately and are very reproducible and this allows the responses from several trials to be averaged. The magnitude and duration of the force the subject experiences at the ankle due to the perturbation can also be recorded and can be used to determine whether the responses are graded to the size of the perturbation. The use of a treadmill allows the subject to establish a regular walking pattern and the perturbations can be applied in a random manner when the subject is not expecting it. Differences in stride length and velocity for a given cadence have been reported between treadmill walking and overground walking (Brandell & Williams, 1974). However, no significant differences were found between the two conditions for 'limb motion' (Brandell & Williams, 1974) or in EMG profiles recorded from soleus, rectus

femoris, biceps femoris, vastus medialis and tibialis anterior muscles (Arsenault, 1986). To some extent the recovery responses will be influenced by the physical constraints of the treadmill. For example, the subjects must continue walking after the perturbation so that they are not carried backward off the treadmill belt. In spite of these constraints the protocol is a reasonable approximation of a naturally occurring trip and is suitable for restricted laboratory space.

In this thesis, the latter protocol will be adapted to investigate the recovery responses to a perturbation of gait in young subjects. The difference between the experimental protocol developed in this thesis and the studies conducted previously, is the use of the Coda *mpx30* motion analysis system which enables both the biomechanical effects and the muscle EMG responses to the perturbation to be recorded synchronously. The Coda can also be used to trigger the perturbation by using the real-time position of a marker placed on the subject's foot. The aim of this study is to develop a protocol of perturbing gait that is not only repeatable and quantifiable but is safe enough to apply to both young and elderly populations.

## 1.5 General aims of the thesis

The experimental work of this thesis was designed to achieve the following aims:

1. To determine the movements of the body that are responsible for the larger movements of the centre of pressure observed in older adults compared to young adults.
2. To investigate whether greater postural sway can also be observed in the elderly during walking.
3. To investigate the relationship between postural sway during standing and postural sway during walking.
4. To determine whether there is a relationship between increased postural sway while standing and decreased muscle force in a group of elderly subjects.
5. To design a safe, repeatable method of perturbing gait that can be used for both young and elderly subjects.
6. To investigate the recovery responses elicited as a result of a gait perturbation in a group of young adults.

## 1.6 The hypotheses to be examined

1. The increased postural sway observed in elderly subjects is due to larger rotations of the body about the ankles.
2. The elderly will exhibit greater postural sway during walking than young subjects.
3. Elderly subjects that make large sway movements while standing will also have large postural sway during walking.
4. Elderly subjects with decreased muscle force (compared to young subjects) will exhibit greater postural sway while standing than elderly subjects with force measurements equivalent to those measured in young subjects.
5. A pattern of recovery responses will be observed following a perturbation of gait that ensures the rapid re-establishment of balance and the continuation of the normal gait pattern.

# *Chapter 2: Materials and Methods*

## 2.1 Subjects

The elderly subjects were recruited via an article in the local newspaper requesting for volunteers to participate in a study investigating muscle strength, balance and falls in the older adult. All the elderly participants were living independently in the community. The young subjects included university students and members of the UCL Institute of Human Performance and their families. Prior to participating in the study the subjects were requested to fill in a medical questionnaire adapted from that used by Greig, Young, Skelton, Pippet, Butler, & Mahmud, (1994). A copy of the medical questionnaire is presented in Appendix C. Subjects were also questioned about their current health status when they attended the Institute for testing. Elderly subjects were excluded from the study of movements of the body during standing and walking if they were unable to walk without an assistive device. Subjects with pain or stiffness of movements of the thumb were excluded from the study relating movements of the COP during standing to measurements of MVF/CSA in the adductor pollicis. Only young, healthy subjects without serious musculoskeletal problems affecting their gait were recruited for the study of corrective responses to tripping. Details of the subjects are given with the results of the individual experiments. There is some overlap, particularly of elderly subjects, between the experiments. The ethical committee at the Royal National Orthopaedic Hospital gave approval for the studies and informed consent was obtained from all subjects.

## 2.2 Apparatus

*Motion Analysis System*

To investigate the movements of the body during standing, a Coda *mpx30* motion analysis system (Charnwood Dynamics) was used to simultaneously acquire 3-D body segment position data and force data from a force plate. Body segment position data was also collected while subjects walked across the laboratory so that movements of the body during

walking could be analysed. For the study of gait perturbations, the Coda was used to collect data on body segment position; to trigger and record the perturbation and to record the force experienced at the ankle during a trip. The hardware consists of a Coda *mpx30* scanner unit containing 3 special cameras that detect infrared pulses of light emitted by the Coda markers. The cameras are rigidly mounted in the scanner unit and the system is pre-calibrated by Charnwood Dynamics. The system measures the positions of the markers within a three dimensional co-ordinate system that is fixed in relation to the scanner unit. The angular resolution of each camera is approximately 0.03 mrad (0.002 degrees); this results in a lateral position resolution of about 0.1 mm at 3 metres distance (horizontally and vertically), and a distance resolution of around 0.6 mm. The markers that are tracked by the Coda Scanner are small infrared light emitting diodes (LED). The LED markers are powered from small power packs that contain rechargeable button cell batteries and control circuitry. The circuitry in the power pack responds to infrared optical control signals sent out from the Scanner Unit when it is acquiring data. In response to the control signals, the marker LEDs are pulsed with current pulses of 40 microseconds duration at up to 200 Hz repetition rate. Three cables transmit the acquired data from the scanner unit to 3 digital signal processor boards (DSP) housed in a computer. Each board has a powerful AT & T DSP32C processor and associated circuitry to enable the vast amounts of data generated during an acquisition to be processed very quickly.

*Optically telemetered EMG*

The Coda *mpx30* also has provision for acquiring surface electromyogram (EMG) data. The muscle activation patterns in the lower limb during normal walking and during a trip were recorded. The EMG equipment includes 8 amplifier modules and connecting leads, prepared electrodes (Skintact®), EMG ground lead, telemetry transmitter and receiver units, 9-volt PP3 battery and DSP data acquisition module. High performance amplifiers are contained in lightweight modules with press-stud fittings for the electrodes. The amplifiers also have

visible red LED's that emit light with brightness proportional to the EMG signal. The amplifiers connect to the transmitting telemetry unit via light flexible wires. The transmitting telemetry unit contains circuitry that further amplifies and rectifies the EMG signals from each of the amplifier modules. The transmitter unit encodes the data from each of the eight EMG channels and transmits it via infrared optical signals to the receiver unit situated near the host computer. The receiver unit detects the optical signals and conveys them to a processing module that is situated on one of the DSP boards in the computer. The processing module decodes the telemetry signals and stores them in the computer memory. This is done synchronously with movement data obtained from the Coda markers.

*The force plate*

The force plate used in all the balance tests described in this thesis was the 'Postural Sway Meter' (Patent application 9318472.9) devised by Mills and McIntyre (1994). A single, four-sided pillar stain gauge is mounted centrally between two aluminium plates. Semiconductor strain gauges are mounted, in pairs, on opposing faces of the pillar. The movements of the centre of pressure applied by a subject standing on the footplate are measured by the semiconductor strain gauges as lateral and sagittal bending forces on the central pillar. The voltage produced by each pair of strain gauges can be used to locate the centre of pressure on the footplate. The strain gauge signals are amplified and then processed and stored on one of the Coda DSP interface boards in the PC. The analog inputs accept a voltage range of ± 10 V and use 12-bit Analog-to-Digital (A/D) converters that give a resolution of 4.88 mV. The signals are sampled at the same rate as the Coda markers. A calibration, using known weights placed at a measured distance enables the output to be converted from arbitrary strain gauge units into torque. Mills and McIntyre (1994) determined the reliability of the force plate by taking three measurements of sway in ten subjects standing with eyes open. The measurements were repeated three days later and the difference from repeat measurements gave a coefficient of variation of 4%.

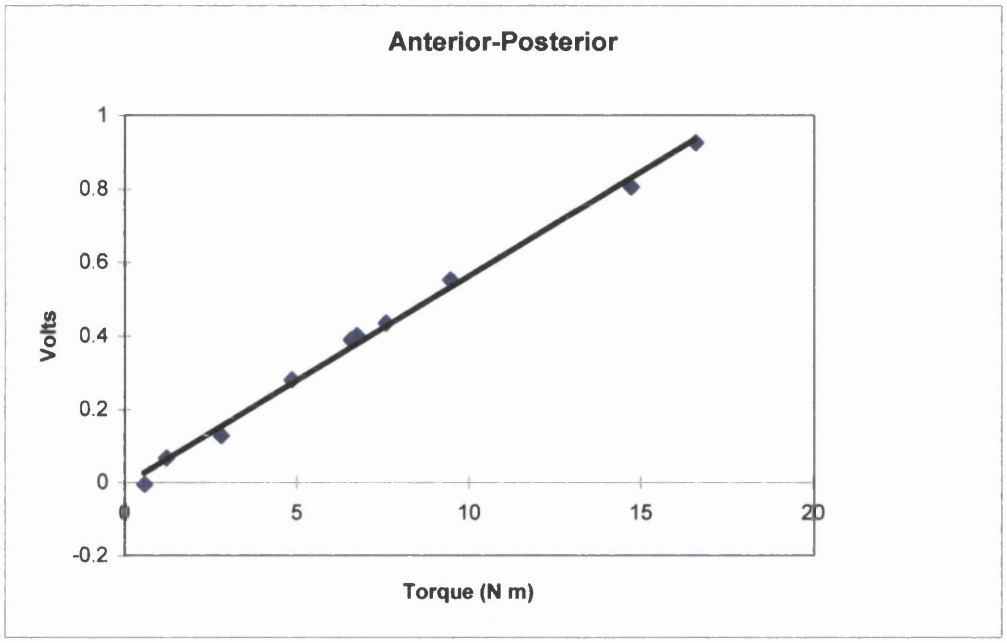
*Calibration of the force plate using the Coda mpx30 Motion Analysis System*

The force plate was calibrated using the *Coda mpx30* Motion Analysis System. Markers were stuck with double-sided tape to known weights that were then placed in various positions on the plate. A record of the position of the markers and the output of the force plate (anterior-posterior and medial-lateral) was acquired for each position of the weight. To convert the arbitrary Coda units to volts, a power function generator (Feedback PFG605) was connected to the force plate inputs and voltages of varying magnitude were introduced while records were made of the Coda output. The power function generator itself was calibrated using a Nicolet 420 digital oscilloscope. The calibration lines (Volts against Torque) for the two force plate channels (anterior-posterior and medial-lateral) are presented in Figures 2.2.1a and b.

*Measurement of Muscle Force*

The apparatus used to measure maximum voluntary force (MVF) in the adductor pollicis muscle consists of 4 strain gauges mounted on an angled metal bar in a Whetstone bridge circuit. The subjects hold their thumb flat in the plane of the palm of the hand and the metal bar is wedged between the bases of the proximal carpal bone of the thumb and the metacarpal bone of the index finger. The subjects are requested to squeeze the bar as hard as possible against the metacarpal of the index finger. An instrumental amplifier powered by a 9-volt battery amplifies the signal before it is fed via an interface to an A/D board in the computer. Customised software was written to prompt the subject to squeeze the metal bar, to record the force exerted, to display the results and to average the records selected by the investigator as being representative of the subject. The apparatus was calibrated each day by suspending known weights from the metal bar and recording the output using a Nicolet 420 digital oscilloscope.

A)




B)

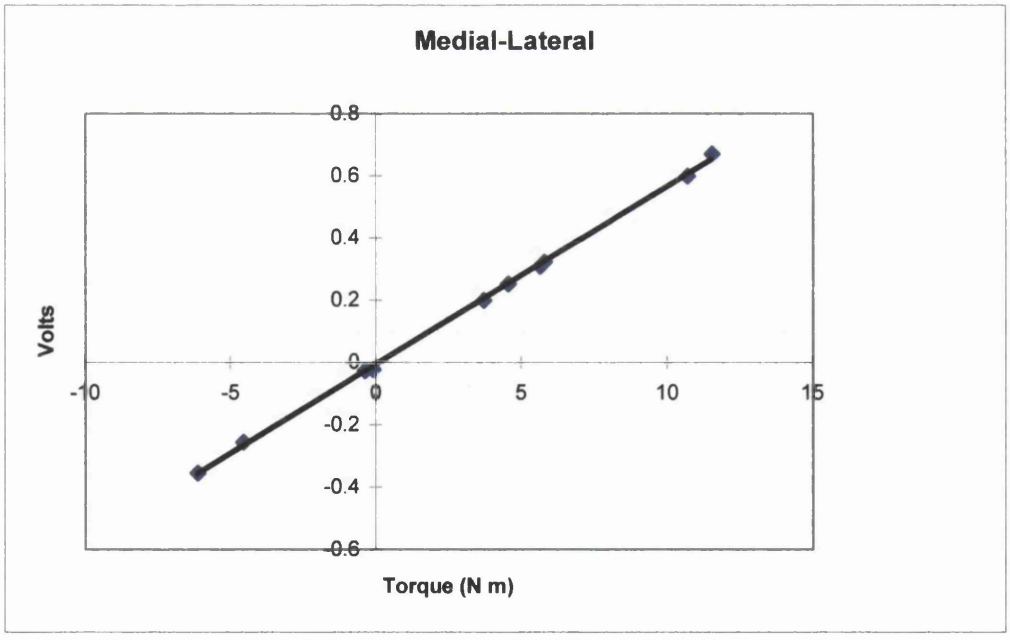



**Figure 2.2.1:** Force plate calibration using the *Coda mpx30*. Coda output (volts) versus torque exerted on the force plate by a 19.95kg weight. A) Anterior-Posterior axis, equation of the line, y = 0.057x – 0.006, $R^2$ = 0.99. B) Medial-Lateral axis, equation of the line, y = 0.057x – 0.007, $R^2$ = 0.99.

*Determining the Cross Sectional Area (CSA) of the adductor pollicis muscle*

The apparatus used for measuring the CSA is that described by Bruce et al. (1986). Briefly, the thickness of the hand is measured in the plane that bisects the adductor pollicis muscle by the difference in the outputs of two linear potentiometers, the shafts of which are held by springs against the two surfaces of the hand. The potentiometers are held in a light frame that can be moved over the hand while its position is monitored by a third potentiometer. An X-Y plot of thickness against distance moved can represent the profile of the hand. This profile is integrated (after allowing for skin thickness) to give an estimate of the CSA. A 500 $mm^2$ triangular perspex block was used to calibrate the apparatus. The area measured by this method is well correlated ($r = 0.937$) with measurements of muscle CSA obtained from computerised tomography (CT) and nuclear magnetic resonance (n.m.r.) images through the same plane (Bruce et al. 1989). The estimated CSA obtained from the hand profiles underestimates the actual muscle CSA by approximately 40% (Bruce et al. 1989). This is partly because some of the muscle is proximal to the bases of the metacarpal bones and is therefore not included in the CSA measurements. In addition, there is a small compressing effect of the springs holding the potentiometers against the two surfaces of the hand. This underestimate does not affect the results which are based on the relationship between force and CSA and not on the absolute value of the ratio.

*Apparatus for perturbing gait*

The Coda *mpx30* motion analysis system and a treadmill (Powerjog GX 100 by Sport Engineering Limited) are used in the study measuring corrective responses to a trip. To obstruct the swing leg during walking an adjustable foot- strap is fitted to the subject's right shoe (See Figure 2.2.2. inset). One end of a lightweight plastic tape is attached to the ankle strap and the other end to a series of small springs (Figure 2.2.2). The system of spring allows the leg to move forwards and backwards during normal walking with little resistance to movement experienced by the subject. The braking mechanism consists of a solenoid with

a rubber bicycle brake attached to the core. The plastic tape feeds between the brake and a rubber mat attached to the underside of the solenoid. When the solenoid is on, the tape is firmly gripped as the two rubber surfaces come together resulting in a braking of the forward progression of the foot (Figure 2.2.2 inset).

*Triggering the gait perturbation*

Prior to recording the perturbation trials, several records of normal walking were obtained for each subject. The maximum height of the LED marker placed on the heel of the subject's shoe was used to estimate the point at which 'toe off' occurred in the gait cycle. This height is used to define the 'trigger' for applying the brake. The solenoid/brake is controlled via a relay unit connected to a digitimer that in turn is connected to the Coda. An external switch operated by the experimenter opens the circuit. Once the circuit is open, the next time the heel marker exceeds the defined trigger height, a 5 volt pulse is sent from the Coda to the digitimer and from there via the relay to the solenoid. The duration of the perturbation is set on the digitimer and the Coda records the timing and duration of the pulse on one of its analog channels. This trigger mechanism enables the timing of the perturbation within the gait cycle to be consistent.

The amount of force the braking produces at the heel is measured as the voltage produced by 2 pairs of strain gauges attached to a metal ring fitted between the foot strap and the plastic tape (Figure 2.2.2 inset). The signal is amplified and recorded on one of the Coda analog channels. The strain gauge circuit is calibrated by hanging known weights from the metal ring and recording the voltage change with the Coda.

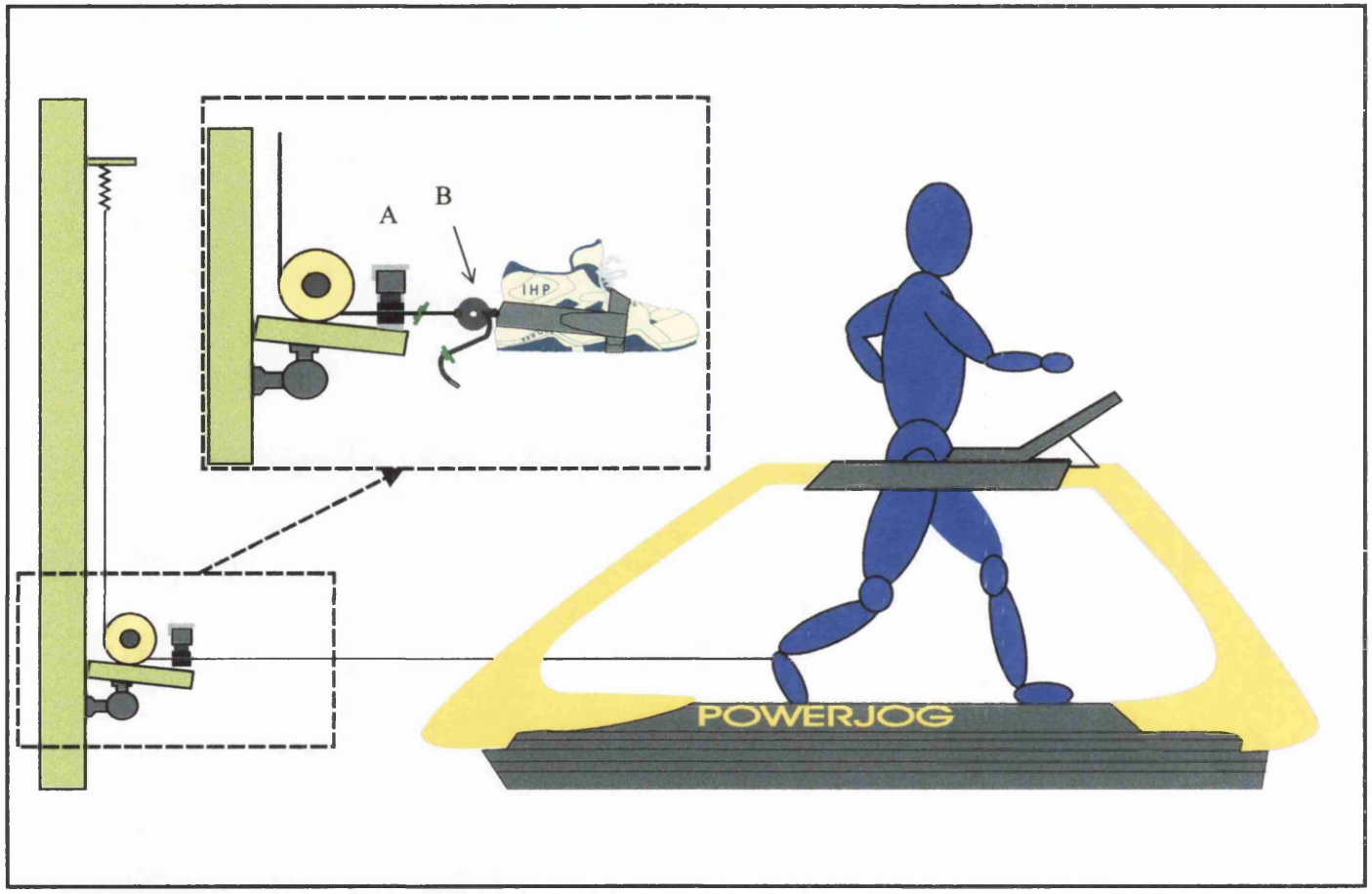



**Figure 2.2.2:** Schematic diagram of the experimental set-up for perturbing gait. An adjustable foot strap is fitted to the right shoe (see inset). One end of a lightweight plastic tape is attached to the foot strap and the other end to a series of small springs. The braking mechanism consists of a solenoid with a rubber bicycle brake attached to the core (A). The force produced at the heel during the perturbation is measured by two pairs of strain gauges attached to a metal ring fitted between the foot strap and the plastic tape (B).

## 2.3 Procedures

### Postural Stability during Standing

The *Coda mpx30* motion analysis system was used to record the positions of fourteen infrared LED markers placed on the body at a sample rate of 50 Hz. The LED markers were attached with double sided tape to the skin overlying the subject's left and right ankle joint (medial malleolus), the knee (patella), pelvis (anterior superior iliac spine), shoulder (coracoid process of scapula), and wrist (ulnar styloid). Four markers were also placed on the head: two markers were attached to small plastic blocks which were then attached to the side of the head at the level of the temporomandibular joint and the remaining two markers were placed at the lateral aspect of the eye. All markers were positioned to ensure they were in view when the subject was facing the *Coda mpx30* scanner unit. The Coda system also recorded the ground reaction forces in the A/P and M/L axes from the force plate. The analogue force signals were sampled at a rate of 50 Hz and were stored in synchrony with the recorded marker movements by the *Coda mpx30* motion analysis system.

The subject was positioned on the force plate facing the Coda Scanner. All subjects stood in bare feet on the force plate, which was covered with a thin nylon carpet. The subjects were instructed to adopt a comfortable self-selected stance. The mean stance width of the young adults was 103.0 ± 6.4 mm (distance between ankle markers, mean ± S.E.M.) or 6.0 % ± 0.4 % of their height. The mean stance width for the elderly group was 98.4 ± 7.6 mm or 6.0 % ± 0.8% of their height. There was no significant difference in the mean stance width adopted by the two groups ($t = 0.45$, $p = 0.651$, two-tailed Student's $t$-test). Three trials of 60 seconds duration were recorded with the subject standing 'as still as possible' with the eyes closed. Between each trial the subjects opened their eyes and took a short walk around the room. A few of the older, more frail subjects sat in a chair between trials. Three trials

were conducted with the eyes open prior to the eyes closed trials but this data is not reported.

## *Postural stability during walking*

Subjects walked barefoot, at their chosen pace along a 9-m marked path on a carpeted floor in the laboratory. The walking trials were conducted immediately following the quiet stance trials described above and therefore the position of the LED markers are identical to those listed previously. The positions of the fourteen markers were recorded by the Coda at a sample rate of 100 Hz. The subjects stood at a point 9 m from the *Coda mpx30* scanner unit (the limit of the measurement field) and initiated their walk across the room after an instruction to 'go' by the experimenter. The subjects walked directly towards the *Coda mpx30* unit and data collection was terminated once the ankle markers disappeared from the measurement field (approximately 2.5-m from the unit). Five trials were acquired for each subject. This procedure was chosen as the most suitable for several reasons: 1) many of the elderly subjects would not have felt comfortable walking on a treadmill without several familiarisation sessions prior to data collection; 2) the sensory inputs during treadmill walking, particularly visual inputs, are different from those experienced during normal walking over ground and 3) some of the variability in walking patterns may have been masked by the imposition of a constant walking speed.

## *Relationship between movement of COP and MVF/CSA*

### *Clinical measures of balance*

All subjects were asked to perform several clinical balance tests so that an estimate of balance capability could be obtained. In the Romberg test (Romberg, 1853), the subjects were asked to stand on the floor with their feet together, first with their eyes open and then with their eyes closed, for 30 seconds each time. The Romberg test was defined as positive

if the subjects took a side step within 10 seconds of eye closure. The subjects were then asked to walk a short distance in a tandem gait (heel to toe). The tandem gait was graded as normal (able to walk 10 steps in tandem), fair (able to walk between 2 and 10 steps without a side step) or poor (unable to walk > 2 steps without a side step). Lastly, the subjects were asked to stand on one leg with their eyes open for up to 30 seconds, or until they were forced to regain their balance by returning the suspended foot to the floor. The time spent standing on one leg was recorded.

*Muscle Force Measurements*

The thumb was positioned in the plane of the palm of the hand and the fingers and interphalangeal joint of the thumb were kept maximally extended. The metal bar was wedged between the thumb and index finger and the subjects were asked to squeeze the metal bar as hard as possible in response to a tone and verbal encouragement from the experimenter. Nine maximal contractions of 4 seconds duration were recorded and the average of the best three to five contractions were used as the measurement of MVF.

*CSA Measurements*

For each subject 3-4 measurements of the CSA were made with the subject removing their hand from the apparatus between measurements.

The mean MVF determined for each subject was divided by their mean CSA measurement to obtain an estimate of maximum voluntary force per unit cross sectional area (MVF/CSA) for each subject. A previous study by Phillips *et al.* (1993) determined mean coefficients of variations for each subject as 3.5% for MVF and 7.5% for CSA.

*COP Measurements*

Subjects stood in bare feet on the force plate with their feet at a 10° angle and their heels separated by either 12 or 16 cm (whichever they felt the most comfortable). Four different test conditions were imposed, each with differing amounts of sensory input available. Subjects stood either (1) on the bare force plate with their eyes open (EO) or (2) with their eyes closed (EC), (3) on a 45 mm X 40 mm X 10.3 mm piece of compliant foam with their eyes open (FEO) or (4) with their eyes closed (FEC). The subjects were instructed to remain as still as possible for 90 seconds. Rest periods of 2-5 minutes were allowed between tests. The tests were conducted in order of increasing level of difficulty, with the EO test considered to be the easiest and the FEC test the most difficult.

## *Recovery responses to a trip*

Subjects underwent a period of habituation on the treadmill (approx. 10 minutes). This ensured that the subject established a regular gait pattern on the treadmill and also helped to stabilise muscle temperature. During the experiment the subjects wore shorts and trainers. Each walked at their own comfortable speed (4.0 - 4.7 km/hr) which was carefully determined by repeatedly increasing and decreasing the speed with the subject reporting each time the speed became to fast or too slow. The selected speed was then used throughout the experiment.

Once the subject had become familiarised with the treadmill, eight Coda markers were attached to the right side of the subject's body and shoe. On the body, markers were placed on the skin surrounding the right lateral malleolus, the lateral epicondyle, the greater trochanter, the highest point of the iliac crest, the upper arm (just below the greater tubercle), the lateral aspect of the right eye and the temporomandibular joint. Markers were placed on the right shoe at the position of the fifth metatarsal head and on the lateral aspect of the heel. EMG activity was recorded from 8 muscles of the leg using surface electrodes

(Skintact® 25 ECG, pre-gelled electrodes, Ag/AgCl ) and the Coda *mpx30* telemetry system. The following muscles were recorded bilaterally: medial gastrocnemius (MGa), Tibialis Anterior (TA), hamstrings and the quadriceps. The subject was positioned on the treadmill and the foot strap was fitted over the subject's shoe. Several normal walking trials were recorded and the mean maximum height of the heel marker during the gait cycle was calculated. This height was used to set the trigger for the perturbation and was an estimate of the point at which toe-off occurred during the gait cycle

During the perturbation trials subjects walked on the treadmill at their self-selected speed for 10 minutes during which time 5 holding perturbations of different duration (120, 180, 240 ms) were applied in a random sequence and at random time intervals within the 10 minutes. There was a minimum of 20 seconds and a maximum of 60 seconds between the perturbations. Three, 10 minute trials were conducted, with the subject stepping off the treadmill and resting for approximately 5 minutes between trials. In total 15 perturbations were applied, 5 at each of the holding impulses (120, 180, 240 ms).

## 2.4 Data Analysis and Statistics

All statistical analyses were carried out using the SigmaStat® software package. An alpha level of 0.05 was employed to determine significance in all statistical analyses.

### *Postural stability during standing*

#### *Marker data*

The mean position of each marker over the 60-second trial was calculated and then subtracted from the raw marker position record to obtain a record of marker movement from mean position. These records were used for determining the correlation between movements of the body segments, in the development of the inverted pendulum model of the body and for calculating the variance of the marker movement.

#### *Transformation of correlation coefficient data*

The association between different variables has been measured by determining the coefficient of product-moment correlation (correlation coefficient) '*r*'. Due to the nature of the data being investigated and the properties of the correlation coefficient calculation the data set is not normally distributed. In order to compare the results of the young group with those obtained for the elderly it was necessary to carry out a transformation of the data. The same transformation was used for all data sets of correlation coefficients. To determine a suitable transformation to use, the correlation coefficients calculated for individual trials of pelvis movement correlated with shoulder movement (122 values in total) underwent a number of different transformations until the distribution satisfied the criteria for normality. The transformation chosen was:

$$Y = LOG(1 - X)$$

Where X equals the calculated *'r'* value.

A two-way analysis of variance (ANOVA) model was used to test for differences in the mean values of the transformed data between the young and elderly groups, between the mean values obtained for the three trials and interactive effects between the group and trials. When a statistically significant difference was found a multiple comparison procedure (Tukey Test) was used to isolate which group or trial differed from the others. When no significant difference was found between the trials and no group x trial interaction effects were observed the values obtained for the three trials were averaged to give a resultant value for each subject.

*The inverted pendulum model*

The development of the inverted pendulum model is described fully in the Results (Section 3.1).

*The variance of the marker and model movements*

For each marker, at each instant, and on each axis, there is an actual movement from position ($q$) and a movement predicted by the model ($m$). The variance of '$q$' and '$m$' was calculated for each marker in both the A/P and M/L axes and the results combined to give the marker movement and model variance for the set of 14 markers. The variance values obtained for each subject trial were then averaged for each set of 3 trials to give a resultant variance for each subject in the A/P and M/L axes. A Mann-Whitney Rank Sum Test was used to compare the median marker and model variance values obtained for the young and elderly groups. The elderly were classified as having either good balance ability 'GBA' or being balance impaired, 'BI' according to their performance on the clinical balance tests and the FEC test (See Results 3.1 and Appendix 1 for further details). A one-way ANOVA was used to compare the variance results obtained for young subjects 'Y', 'GBA' and 'BI' elderly.

*COP data*

The data from the force-plate was converted to A/P and M/L records of movement of the COP (mm). The mean position of the COP over the 60-second trial was subtracted from the time series record to give an average COP position of (0,0). The root mean square (RMS) amplitude of the movement in each axis was calculated using the equation:

$$RMS = \sqrt{\frac{1}{R}\cdot\sum_{r}\left(COP_r\right)^2}$$

R equals the number of data points in the record and r equals the range from 0 to R-1.

The RMS data was tested for trial repeatability and group x trial interactions using a two-way ANOVA. The variance of the COP movement in the A/P and M/L axes for each subject trial was calculated by squaring the RMS value. Resultant variance values for each subject were obtained by averaging the values for the three trials. A Mann-Whitney Rank Sum Test was used to compare the variance results for the young and elderly groups as the data failed the Kolmogorov-Smirnov normality test.

A fast Fourier Transform (FFT) was used to estimate the frequency composition of the COP record and calculate the power spectra (bandwidth 0.0167 - 4.99 Hz, resolution = 0.0167 Hz) in the A/P and M/L axes. Three power spectra were averaged for each subject resulting in a spectral signature for that individual in each axis. Averaging the individual spectral signatures generated spectral envelopes for the young and elderly groups. The mean spectral frequency (centroidal frequency) was determined for each subject from the calculated spectral signature.

The mean spectral frequency (MSF) is given as:

$$MSF = \frac{\sum_{k} f_k \cdot P_k}{\sum_{k} P_k}$$

Where k = 0 - R/2 (R = no of data points), $f_k$ is the frequency and $P_k$ is the power spectrum at that frequency.

The power contained within two bandwidths, a low frequency bandwidth (0.0167 - 0.251 Hz) and a higher frequency bandwidth (1.01 - 4.99 Hz) was determined by calculating the sum of the areas under the power spectra for each bandwidth. The power determined for a discrete bandwidth is equal to the variance of the COP record in this bandwidth. Hence the square root of the total power calculated for a specific bandwidth will be equivalent to the standard deviation (S.D.) of the signal, which is the amplitude of the COP movement in the specified bandwidth. The amplitude of the COP movement in the high and low frequency bandwidths for the young and elderly groups were compared using a Mann-Whitney Rank Sum Test as the data had a non-normal distribution. For the subgroup analysis a one way ANOVA was used to compare the mean amplitude of the BI, GBA and young subgroups. If a significant difference was found, an all pair-wise multiple comparison procedure (Tukey Test) was used to isolate the group(s) that differed.

*Stabilogram-Diffusion Analysis*

The COP trajectories recorded from the force plate during quiet stance were analysed using a technique based on *stabilogram-diffusion* analysis (Collins and De Luca, 1993, 1995). *Stabilogram-diffusion* analysis is based on the assumption that maintaining an upright posture is partly a stochastic process. Collins and De Luca (1993) were able to demonstrate

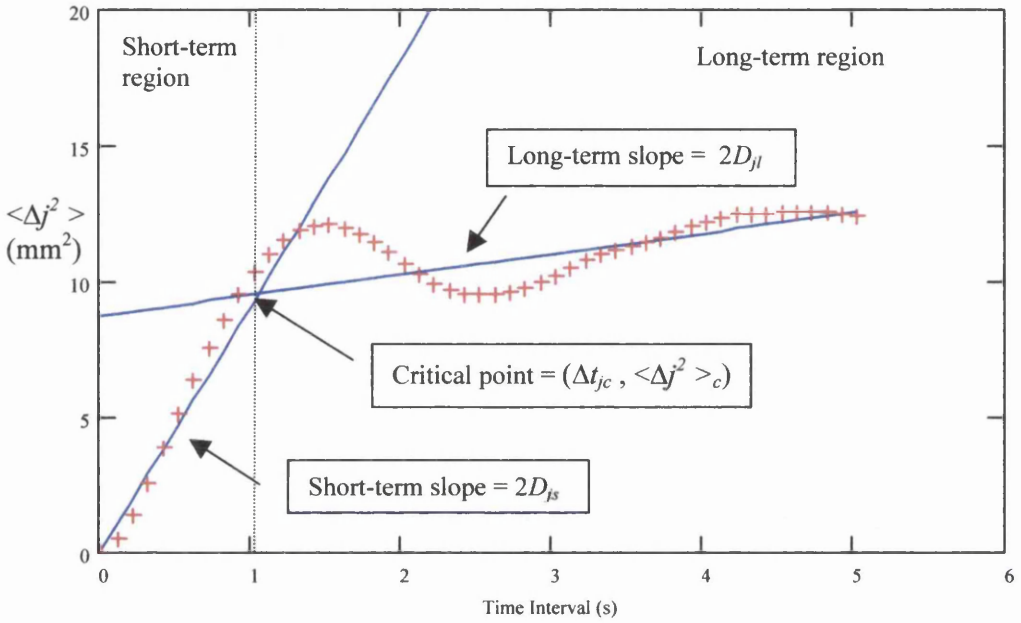
using concepts and principles from statistical mechanics that the movements of the COP during quiet stance could be modelled as one dimensional and two-dimensional random walks. In this thesis, movements of the COP were analysed only as a one-dimensional random walk. In a one-dimensional random walk (Brownian motion), past movements are uncorrelated with future movements and the mean square movement $< \Delta x^2 >$ of a one-dimensional random walk is related to the time interval $\Delta t$ by the expression:

$$< \Delta j^2 > = 2D_j \Delta t$$

Where $< \Delta j^2 >$ is mean square movement, $D_j$ is the diffusion coefficient and reflects the level of stochastic activity of the COP along the anterior-posterior ($j = y$) or medial-lateral ($j = x$) axis. Diffusion coefficients can be used to quantify postural instability, i.e. larger $D_j$ corresponds to a less tightly regulated or 'more random' control system and vice versa (Collins and DeLuca, 1993). In order to calculate the diffusion coefficients, the square of the movements between all pairs of points separated in time by a specified time interval $\Delta t$ are calculated. The square movements are then averaged over the number of $\Delta t$ making up the COP time series. This process was repeated for increasing values of $\Delta t$. The number of calculated square movements is inversely proportional to the size of the time interval. A plot of mean square COP displacement versus time interval $\Delta t$ is referred to as a *stabilogram-diffusion* plot. Diffusion coefficients, $D$ are calculated from the slopes of the resultant linear-linear plots of mean square COP movement versus time interval curves. The slopes are determined by utilising the method of least squares to fit straight lines through defined portions of the plots. Figure 2.4.1 illustrates a *stabilogram diffusion* plot calculated for an elderly subject. Two distinct regions can be identified on a *stabilogram-diffusion* plot: a short-term region and a long-term region. These regions are separated by a transition period where the slope of the *stabilogram-diffusion* plot changes considerably. The critical

point ($\Delta t_{jc}$, $< Dj^2 >$) is defined by the intersection of the lines fitted to the two regions of the plot and quantifies the spatial and temporal characteristics of the transition.

**Figure 2.4.1:** Schematic representation of *stabilogram-diffusion* plot




A typical stabilogram plot ( $<\Delta j^2>$ *vs* $\Delta t$) generated from a medial lateral COP record. The diffusion coefficients $D_{js}$ and $D_{jl}$ are computed from the slopes of the lines fitted to the short-term and long-term regions respectively. The critical point ($\Delta t_{jc}$ , $<\Delta j^2>_c$) is defined by the intersection of the lines fitted to the two regions of the plot.

According to Collins and De Luca (1993) the observed temporal structure of the *stabilogram-diffusion* plot suggests that at least two different postural control systems are operating during the regulation of quiet stance. Over short time periods, sway operates in an open-loop manner such that motion is uncontrolled (not influenced by afferent signals from the visual, vestibular and somatosensory systems). Open-loop sway continues until it exceeds 'some systematic threshold' (Collins & DeLuca, 1993) after which a closed-loop (feedback) mechanism is activated in order to modify the activity of the postural musculature. Collin and Deluca (1993) suggest that this open-loop/closed-loop control

strategy may have evolved to take account of the inherent time delays of feedback loops and to simplify the task of integrating vast amounts of sensory information when the system is not in any danger of instability.

*Stabilogram-diffusion* plots were computed for each subject trial in the A/P and M/L direction. The parameter extracted from the plot was the short-term diffusion coefficient, $D_{js}$ calculated from the initial slope of the linear-linear plot of mean square movement versus $\Delta t$. This measure was used as an estimate of an individual's postural stability during standing. *Stabilogram-diffusion* plots were also computed for the inverted pendulum model record (derived from movements of the markers) and the residual record (COP - model). The values obtained for $D_{js}$ of the COP, model and residual records were normalised using a logarithmic transformation. A two-tailed *t*-test was then used to compare the mean values calculated for the young and elderly groups.

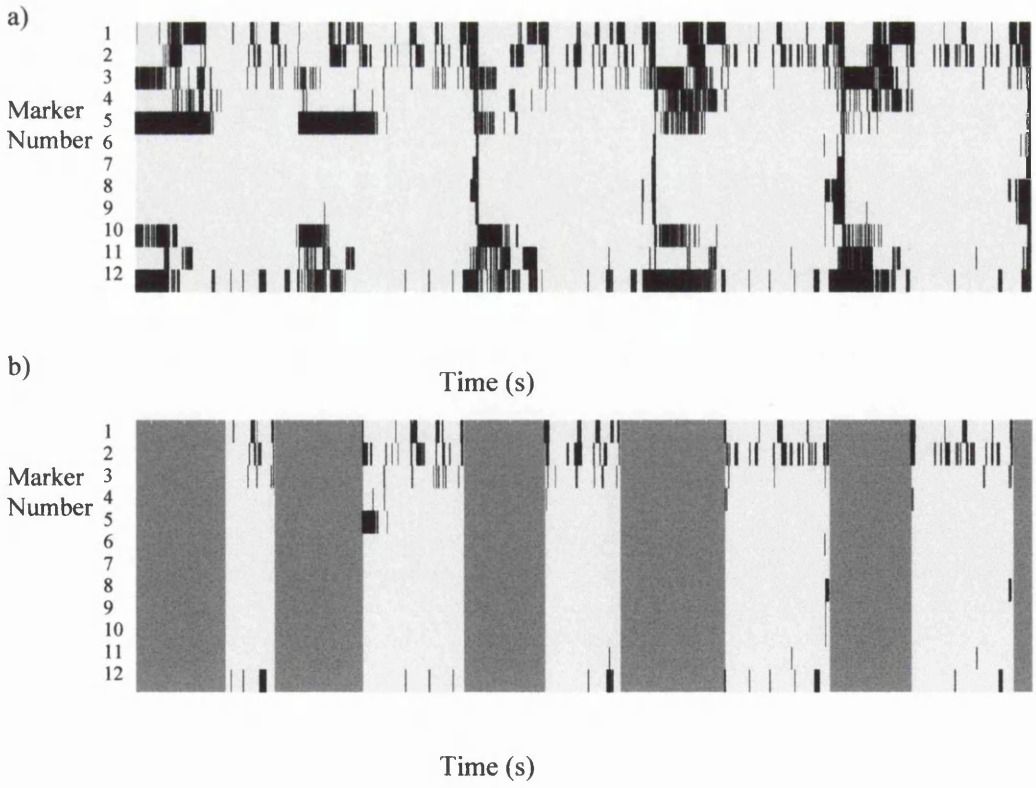
## *Postural Stability during Walking*

### *Selection of strides for analysis*

The data from approximately five strides were extracted from each of the five walking trials. The first 1-2 strides of each trial were often atypical due to the initiation of the walk and so were excluded from the analysis. To obtain sufficient 'normal' strides for averaging the five individual subject trials were joined end-on-end to produce a single record for each subject. The time points when the body weight was being transferred to the left foot i.e. when the right ankle was above the left were determined and these time points were used to calculate the stride times. From the combined record, strides were selected for averaging if: 1) the majority of the markers were in view of the *Coda mpx30* for the duration of the stride and 2) the stride was not affected by the initiation of gait as judged by the stride time.

To determine whether individual markers were in view of the scanner unit, a second data record was produced which contained information about the 'in-view' status of the markers for each trial. For each marker, at each time point, the second data record contained either a '1' when the marker was in view or a '0' when it was out of view. The data was plotted so that pale grey areas represented 'in-view' markers and black areas represented time-points where marker information was missing (Figure 2.4.2a). From the list of stride times, strides were removed from the combined record if there was a large proportion of missing marker data during that stride. Strides affected by initiation of gait were also removed at this point. To view the strides that were to be included in the averaging the diagram was re-plotted with the removed strides represented in dark grey. (Figure 2.4.2b).

**Figure 2.4.2**: a) Plot showing time intervals for which the markers were 'in-view' of the Coda scanner unit (pale gray areas) and time intervals for which no position data was recorded (black areas). b) Plot showing the strides removed from the analysis due to lack of position data (dark gray bands).



*Marker and Model Variance*

The values obtained for the variance of marker and model deviations were normalised using a logarithmic transformation. Differences in the mean values of the transformed data between the young and elderly groups were determined using a two-tailed Student's *t*-test. A Mann-Whitney Rank Sum Test was also performed on the raw data. A subgroup analysis of the data was conducted using the same individuals selected in the experiment investigating postural stability during standing. The subjects were divided into three groups: young adults (Y), 'balance impaired' elderly (BI) and elderly with good balance ability (GBA), The values obtained for the marker and model variance for the three groups were compared using a one way ANOVA. When a significant difference was found a Tukey test was used to make pair-wise multiple comparisons between the groups.

*Correlation between walking variance and static variance*

The association between the variance of the marker and model movement during walking and quiet standing for the young and elderly groups was measured using a Spearman Rank Order Correlation due to the non-normal distribution of the data.

*Correlation coefficient data*

The association between the marker and the inverted-pendulum model movement data for each subject was measured by calculating the correlation coefficient '*r*' for each subject. The data was normalised using the same transformation described for correlation of the markers and model during standing i.e. Y = LOG(1-X). A two-tailed Student's t-test was used to compare the mean values of the transformed data for the young and elderly groups.

*Step width determination and correlation with M/L marker variance*

To determine the mean step width for each subject, the mean separation between the left and right ankle markers for the period of double stance prior to right toe off was calculated for

all strides selected for the averaging process (10 strides minimum). Stride-to-stride variability in step width was determined by calculating the standard deviation of the ankle separation from mean step width. A two-tailed *t*-test was used to detect significant differences between the young and elderly groups. The association between M/L marker variance and mean step width and stride-to-stride variability was measured by the correlation coefficient '*r*'. A linear regression analysis was also performed on the data and the significance of the slope of the regression line was evaluated using a *t*-test.

## *Relationship between movement of the COP and MVF/CSA*

A Pearson Product Moment Correlation and linear regression were used to determine the relationship between MVF and CSA in the young adults. A Spearman Rank Order Correlation was used to determine the association between MVF and CSA in the elderly because the MVF data was not normally distributed. The COP data sets were also not normally distributed so a logarithmic transformation of the data was carried out. Unpaired Students two-tailed t tests were used to test for significant differences in the mean values of the young and elderly groups (MVF/CSA, COP variance). The M/L EC and A/P FEO data sets for the elderly subjects failed the normality tests even after the transformation so Mann-Whitney Rank Sum tests were used for these data sets instead. A Pearsons Product Moment Correlation was used to determine if there was an association between an elderly individual's MVF/CSA and movements of their COP under any of the 4 test conditions (EO, EC, FEO, FEC). A Spearman Rank Order Correlation was used for the correlation between MVF/CSA and A/P COP variance (EO). A Pearson Product Moment Correlation (and Spearman Rank Order Correlation where appropriate) was also used to determine the relationship between MVF/CSA and walking variance and COP variance and walking variance in a small group of elderly.

## *Recovery responses to a trip*

To compare the responses of perturbed strides with control, non-perturbed strides, data for individual strides were extracted from the records containing perturbations of the same duration and placed in a bin of 'control' strides or 'perturbed' strides. The perturbed strides were selected by the timing of the trigger pulse. The individual heel strike times were determined for the entire record and this information was used to calculate the mean stride time for the 5 trials. The control strides were averaged to obtain a mean stride path for each of the 8 markers and the mean stride pattern of muscle activity for each of the 8 muscles studied. The standard error of the mean for the 'control' strides was also calculated. Approximately 80 strides were included in the mean for the 'control' strides. The marker and EMG data for the five perturbed strides (one from each trial) were also averaged and the SEM associated with the mean was calculated. This procedure was repeated for each of the three holding impulse times.

Profiles of the force experienced at the foot as a result of the perturbation were also averaged. The time point at which the force increased to a maximum was arbitrarily chosen as the point from which to calculate the onset latency of the reflex responses in the muscles.

The mean marker data for the control and perturbed strides was used to create a series of stick figure diagrams that illustrate the position of the body segments at various time points in the stride. As markers were only placed on the right side of the body only the kinematics of the right leg could be determined. The mean marker data was also used to plot trajectories of the knee, ankle, heel and toe in control and perturbed strides

# *Chapter 3: Results*

## 3.1 Postural stability during standing

*Subjects*

Twenty young men and women aged between 19 and 43 years (mean ± S.E.M., 28.7 ± 1.5 years) and 22 elderly men and women aged between 64 and 85 years (mean ± S.E.M., 72.4 ± 1.2 years) participated in this experiment. See Table 3.1.1 for a description of the general characteristics of the subject groups. The young group were significantly taller than the elderly group, Student's *t*-test ($t = 2.93$, $P < 0.01$) but there was no significant difference in the mean weight of the two groups or in the proportion of women subjects included in each group. Information on health status, fall history and physical activity of the older adults was gathered via a questionnaire (See Appendix C) and face-to-face interview. A summary of the results is presented in Tables 3.1.2 and 3.1.3.

**Table 3.1.1:** General characteristics of subject groups

| | YOUNG<br>N = 20 | ELDERLY<br>N = 22 | |
|---|---|---|---|
| Mean (± S.E.M.) or Proportion (%) | | | *P* |
| No. of women | 11/20 (55%) | 9/22 (41%) | 0.35 |
| Height (m) | 1.72 (0.02) | 1.66 (0.02) | < 0.01* |
| Weight (kg) | 66.5 (3.0) | 68.0 (2.4) | 0.43 |

# Tests for differences associated with age: Student's *t*-Test (Height, Weight) and chi-squared test (No. of women).

*, Statistically significant difference between the groups at $p < 0.01$.

**Table 3.1.2:** Clinical characteristics of elderly participants (N = 22).

| | N (% of Group) | | N (% of Group) |
|---|---|---|---|
| **Diagnoses** | | **Medications** | |
| Cardiac disease | 4 (18.2) | No medication | 6 (27.3) |
| Hypertension | 4 (18.2) | Cardio-vascular | |
| Raynaud's disease | 1 (4.5) | *Diuretic* | 4(18.2) |
| Respiratory disease | 4 (18.2) | *Amiodarone* | 1( 4.5) |
| Diabetes | 1 (4.5) | *ACE inhibitor* | 2( 9.1) |
| Hypothyroidism | 2 (9.1) | *Calcium channel blocker* | 1 (4.5) |
| Stroke / TIA | 1 (4.5) | *Anticoagulent* | 1 (4.5) |
| Visual problems | 6 (27.3) | *Antiplatelet* | 3 (13.6) |
| (cataract, glaucoma) | | | |
| Musculo-skeletal | | Respiratory | |
| Joint pain | | *Inhaled Bronchodilators* | 2 (9.1) |
| neck | 2 (9.1) | *Inhaled Corticosteroids* | 4 (18.2) |
| hand (excluding thumb) | 1 (9.1) | NSAID | 2 (9.1) |
| knee | 2 (9.1) | Lithium | 1 (4.5) |
| Low back pain | 4 (18.2) | Endocrine | |
| Old knee injury | 1 (4.5) | *Sulphonylurea* | 1 (4.5) |
| Diagnosed osteoporosis | 3 (13.6) | *Thyroxine* | 2 (9.1) |
| History of neoplasia | 2 (9.1) | Gastro-intestinal | |
| (prostrate, meningioma) | | *Acid supressing* | 2 (9.1) |
| Psychotic illness | 1 (4.5) | Genito-urinary disorders | |
| | | *Oxybutynin* | 1(4.5) |

**Table 3.1.3:** Characteristics of elderly participants: mobility, experience of falling and activity level (N = 22).

| | N (% of Group) |
|---|---|
| **Use of mobility aid** | 0 (0) |
| **Falls in the past 2 years** | |
| At least once | 5 (22.7) |
| More than one fall | 4 (18.2) |
| **Physical Activity** | |
| (1-2 times/week) | |
| Light | 2 (9.1) |
| *(walking < ½ hour)* | |
| Moderate | 11 (50.0) |
| *(walking > ½ hour, bowls, gardening)* | |
| Vigorous | 9 (40.9) |
| *(jogging, tennis, keep-fit, cycling, swimming)* | |

*Classification of elderly into 'balance impaired' (BI) and 'good balance ability' (GBA) subgroups*

Three measures of balance capacity were taken on each elderly subject during a previous visit to the laboratory: ability to stand on one leg (OLS), to walk heel-to-toe (tandem gait) and to stand on a thick foam pad with the eyes closed (FEC). The elderly subjects were ranked according to their performance on each test. For each subject the ranks for the three tests were averaged to give a 'mean balance score'. Individuals with a mean balance score of 1.0 were classified as having 'good balance ability' (GBA) while those with a mean balance score greater than 1.0 were classified as 'balance impaired' (BI). Two individuals were considered to have 'severe balance impairment' (SBI) and are considered separately as case studies. A summary of individual performances on the balance tasks, rank for each task and the mean balance score of the GBA, BI, and SBI elderly are presented in Appendix A1. Two elderly participants were not included in the subgroup analysis due as they had not participated in the previous experiment and therefore the data for the balance tests was

incomplete. Two other elderly subjects that performed well on the balance tests were excluded from the 'good balance ability' subgroup because they were taking medications that can cause postural hypotension (bendrofluazide, lithium carbonate).

*Repeatability of COP measures*

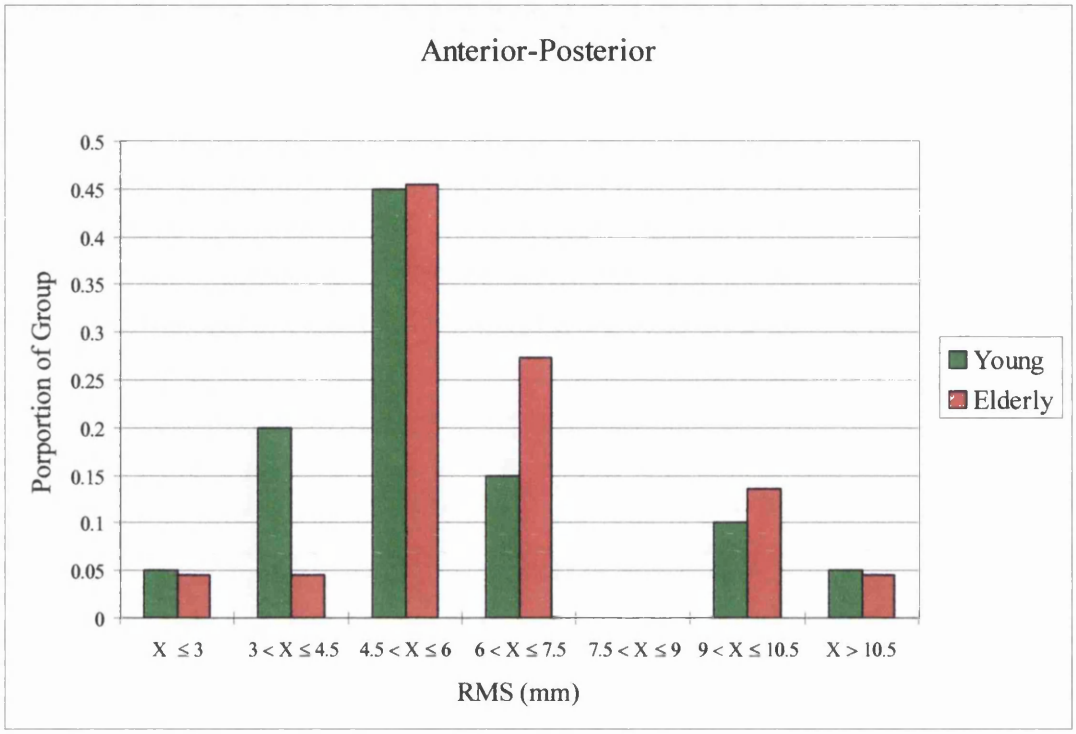
The root mean square (RMS) amplitude of the COP movement was calculated in both the A/P and M/L axes for each subject trial. The values obtained for the three individual trials were averaged together to obtain a resultant value for each subject in the two axes. Figures 3.1.1a and 3.1.1b illustrate the distribution of the resultant RMS values for the two groups. In the A/P axis, there were a small number of young (3) and elderly (4) subjects with RMS values much greater (above 9 mm) than the rest of the group. In the M/L axis, 3 elderly subjects had RMS values much greater (above 6.5 mm) than the other subjects. The bimodal shape of the M/L histogram suggested that these individuals might belong to a separate population. To determine whether it was valid to average the individual RMS values for each subject, a two-way (Group x Trial) analysis of variance (ANOVA) was carried out. As suggested by the frequency distribution histograms the RMS COP movement values obtained for the young and elderly subject groups were not normally distributed in either the A/P or M/L axes. The data failed to pass the normality test even after several transformations of the data set were tried. In order to examine the repeatability of the data, the 7 subjects (3 young, 4 elderly) with an average A/P RMS value above 9 mm were excluded from the group and tested separately. The results of a two-way ANOVA on the remaining 35 subjects showed that there was no significant difference between the trials in the A/P axis ($p = 0.576$, d.f. = 2) and no interactive effects between group and trial were found ($p = 0.474$, d.f. = 2). The results of a two-way ANOVA on the 7 excluded subjects also showed no difference between trials ($p = 0.265$, d.f. = 2) or interactive effects ($p = 0.790$, d.f. = 2). In the M/L axis, the results for the 3 elderly subjects with RMS values above 6.5 mm were excluded from the data set prior to testing for repeatability and

interactive effects. The results of the two-way ANOVA showed that for the remaining 39 subjects there was no significant difference between the 3 trials after allowing for the effects of differences in-group ($p$ = 0.726, d.f. = 2) and no Group x Trial interactive effects were observed ($p$ = 0.770, d.f. = 2). As there was no significant difference between the trials, it seemed valid to average the results to obtain a single RMS value for each subject.

*Comparing the variance of the COP movement for the young and elderly*

The RMS COP movement values calculated for each subject trial were squared to obtain an estimate of the variance of the COP movement in the A/P and M/L axes. The variance values for the three trials were averaged to give a resultant value for each subject. As this data set also failed the normality test, a Mann-Whitney Rank Sum Test was used to compare the variance results of all young and elderly subjects. In the A/P axis, the difference in median values between the young (30.75 $mm^2$, N = 20) and elderly (33.28 $mm^2$, N = 22) was not statistically significant ($p$ = 0.399). In the M/L axis the COP movement variance calculated for the elderly group (8.99 $mm^2$) was significantly greater ($p$ = 0.043) than the variance calculated for the young group (6.66 $mm^2$).

a)




b)

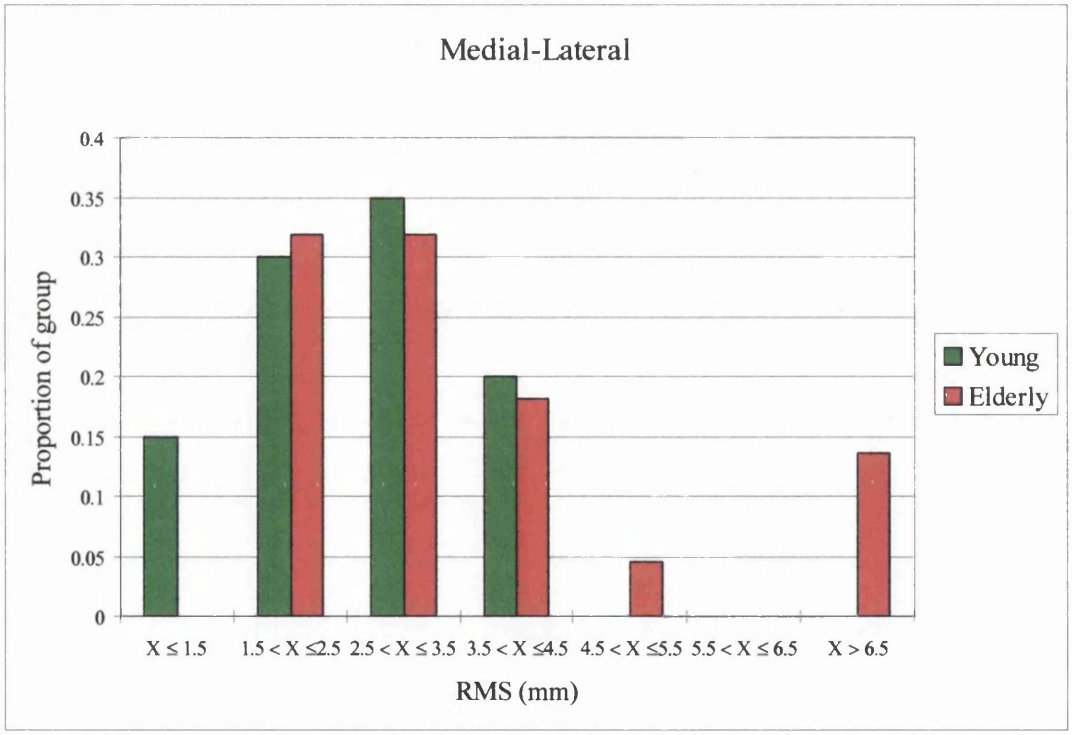
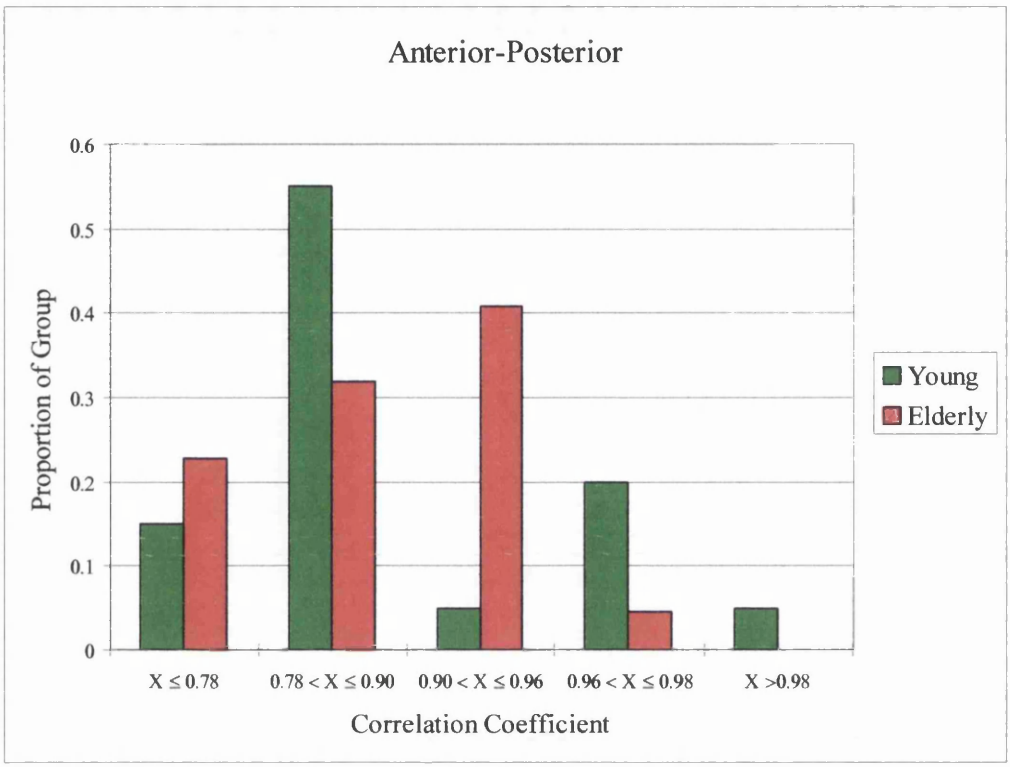



**Figure 3.1.1:** Histogram of RMS COP movement in the a) Anterior-Posterior, b) Medial-Lateral axes. Area of bar is equal to the proportion of the group whose RMS values fall within the limits of the bin. N(Young) = 20, N(Elderly) = 22.

*Correlation between pelvis and shoulder motion*

If the human body behaves as an inverted pendulum during quiet stance, all the body segments should move in the same direction at the same time and there should be a high positive correlation between movement records of the body segments. The records of marker co-ordinates were examined for evidence of a positive correlation between the movements of the pelvis and shoulders in the A/P and M/L axes. The mean position of each marker was calculated and then subtracted from the raw marker position record to obtain a record of *marker movements from mean position.* The records obtained for the left and right pelvis markers were averaged to give a single pelvis movement record in each axis. Similarly, *marker movements from mean position* records were obtained for the shoulders and other body segments. Time-series correlation coefficients for the pelvis and shoulder movement were calculated for each subject trial. The values were transformed to obtain a normal distribution of the data (See Methods) and then the transformed values for each set of 3 trials were averaged to obtain a resultant value for each subject. The validity of averaging the results of the three trials was confirmed by the results of a two way ANOVA (Group x Trial) on the transformed data. The results showed that there was no significant difference between the means of the three trials ($p$ = 0.296 (A/P) and 0.876 (M/L)) after allowing for the effects of differences in-group. There was also no significant interaction between Group and Trial ($p$ = 0.590 (A/P) and 0.995 (M/L). Figure 3.1.2 illustrates the frequency distribution of the resultant values for the two groups. The geometric mean ± 1 S.E.M. obtained for the two groups are presented in Table 3.1.4. The data in Table 3.1.4 shows that a strong positive correlation exists between the movements of the pelvis and shoulders in the A/P and M/L axes in both subject groups. The group means were compared using a two-tailed Students $t$-test. The results of the $t$-test indicated that in the A/P axis there was no difference between the young and elderly group means ($t$ = -0.189, $p$ = 0.851, d.f = 40). In the M/L axis there was a significantly larger correlation between pelvis and shoulder movement in the elderly group compared to the young ($t$ = 3.751, $p$ < 0.001, d.f = 40).

a)




b)

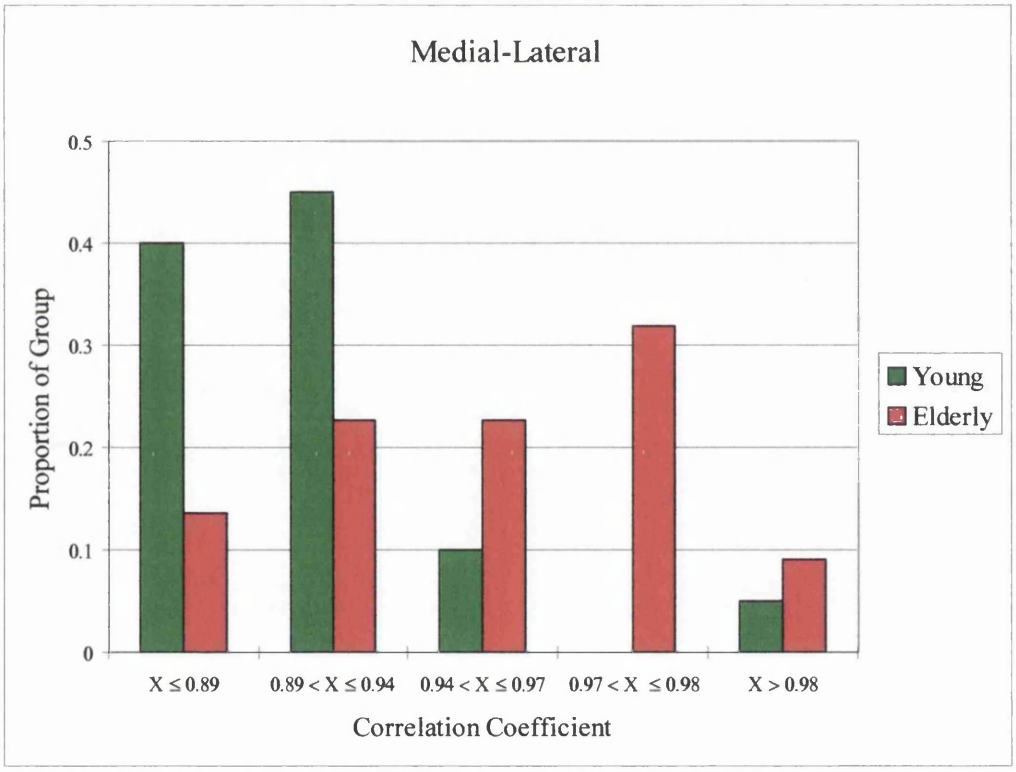



**Figure 3.1.2:** Histogram of correlation coefficients calculated for shoulder movement correlated with pelvis movement. Areas are equal to the proportion of the group whose '*r*' values fall within the limits of the bin. N(Young) = 20, N(Elderly) = 22. Histogram calculated using transformed resultant values for each subject but results shown in original units for clarity.

**Table 3.1.4:** Summary of the correlation coefficients calculated for pelvis movement correlated with shoulder movement during quiet stance, eyes closed trials, for the young (N = 20), and elderly (N = 22). Mean ± 1 S.E.M. calculated from transformed data. Also shown are the results of a two-tailed *t*-test (d.f. = 40).

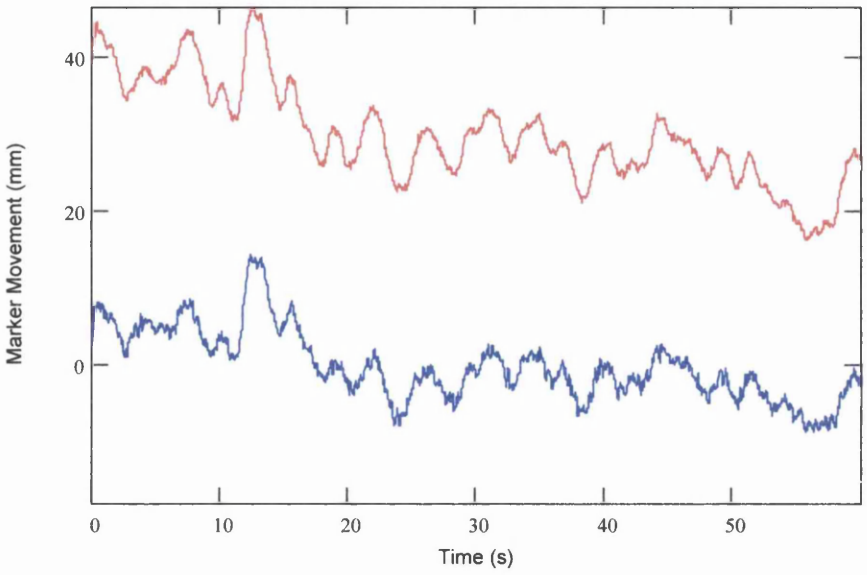
| AXIS | GEOMETRIC MEAN (± 1 S.E.M.) | | *P* |
|---|---|---|---|
| | Young | Elderly | Young = Elderly |
| Anterior-Posterior | 0.91 (0.88, 0.92) | 0.89 (0.87, 0.91) | 0.851 |
| Medial-Lateral | 0.91 (0.90, 0.92) | 0.96 (0.95, 0.97) | <0.001* |

* Denotes significant difference, $p < 0.001$

High positive correlation between the movement of other body segments was also observed, including pelvis-knee, pelvis-head, shoulder-head, shoulder-knee, and head-knee. A summary of these results is presented in Appendix A2. The high positive correlation between the movement of the pelvis and shoulders as well as other body segments suggests that all the body segments move in the same direction, at the same time during quiet stance.

The correlation coefficient obtained for each subject trial is an average over the entire trial, and therefore changes in postural strategy during the trial are not characterised by this single measure. Consideration of individual subject trials showed that for a few of the young and elderly subjects there were short episodes of shoulder movement independent of the pelvis within the 60 second duration of the trial. These movements were usually only observed in the A/P axis. When such episodes occurred, correlation between the pelvis and shoulder movement averaged over the whole trial was less positive. Figures 3.1.3 and 3.1.4 present graphs of trials with a high and low correlation respectively. In Figure 3.1.4 there are short time intervals where the pelvis and shoulder movements are negatively correlated, suggesting a hip-strategy was used during these time periods.

a)




b)

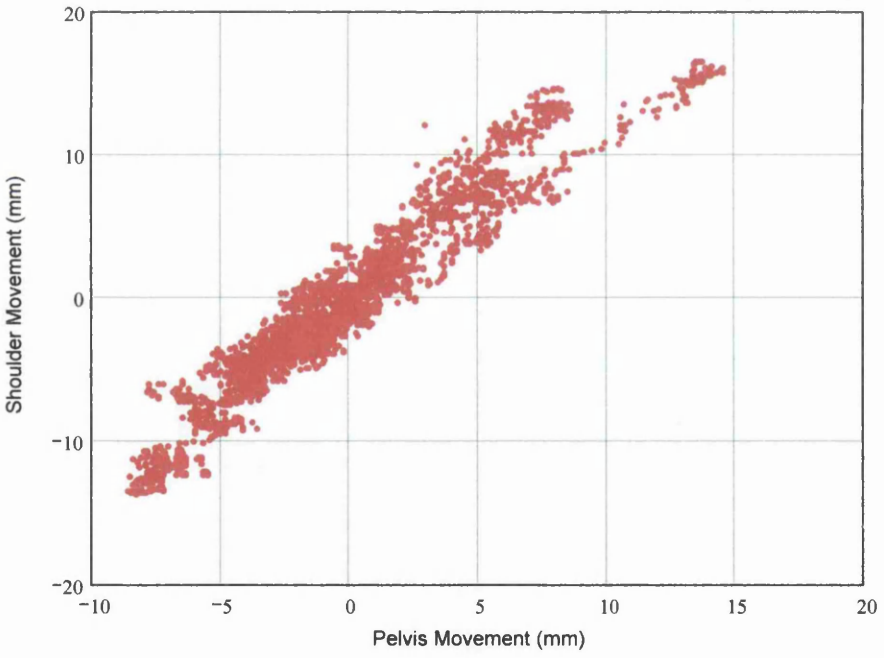
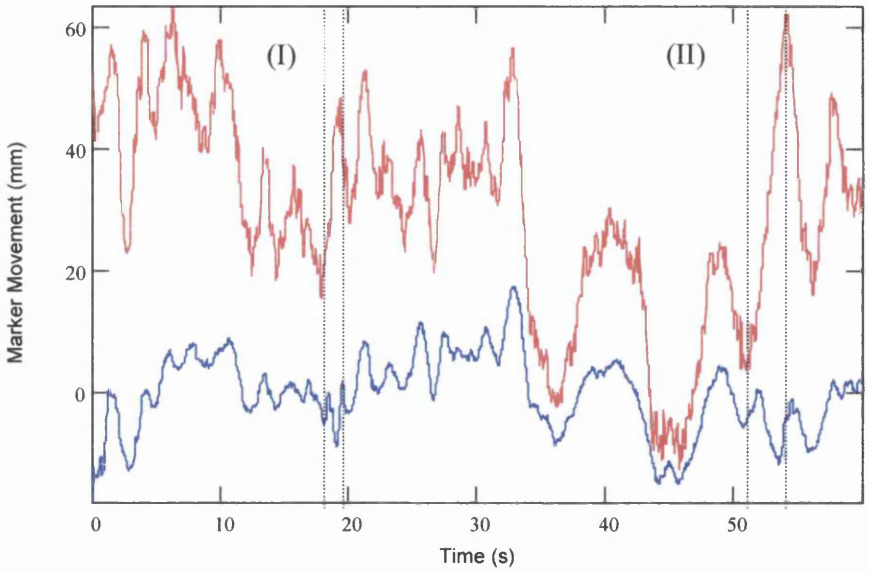



**Figure 3.1.3:** Shoulder and pelvis marker motion for an individual subject trial in the A/P axis. A) Time-series graphs of shoulder and pelvis movement from mean position (shoulder movement offset by 30 mm for clarity). B) Time-series correlation between shoulder marker movement and pelvis marker movement, $r = 0.96$.

a)




b)

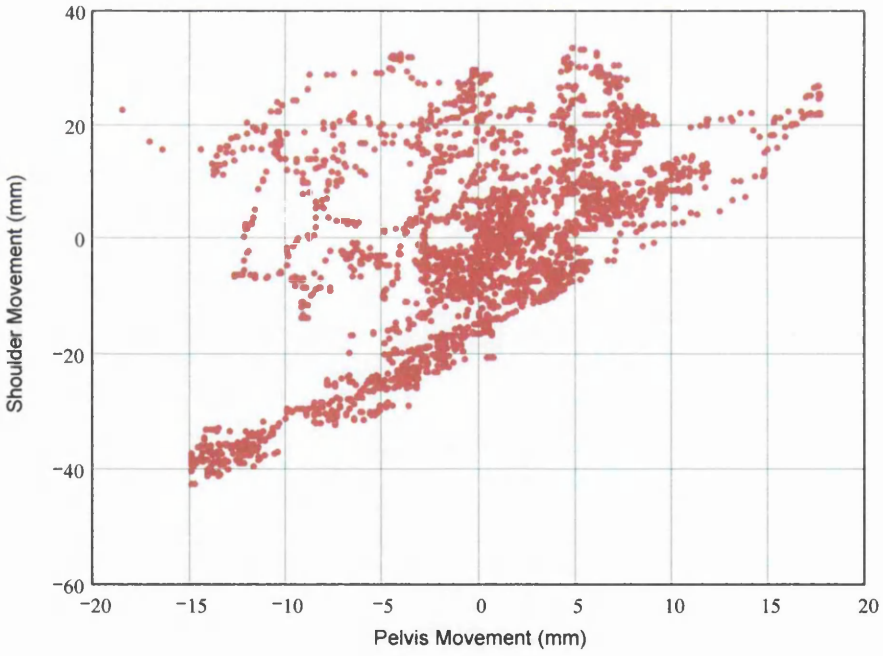


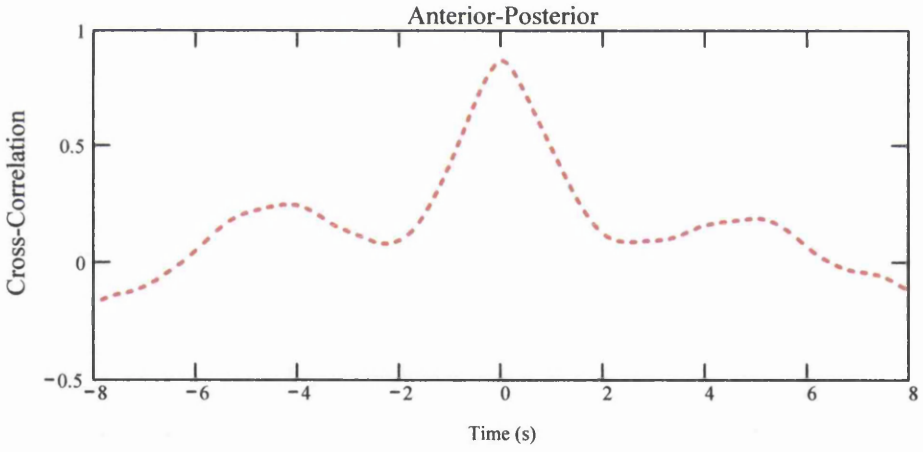

**Figure 3.1.4:** Shoulder and pelvis motion recorded for an individual subject trial in the A/P axis. a) Time-series graph of shoulder and pelvis movement from mean position, (I) and (II) represent time intervals where shoulder and pelvis motion are negatively correlated, $r$ = -0.94 and –0.67 respectively. b) Time-series correlation between shoulder and pelvis marker movement for the 60 s trial, $r$ = 0.55.

*Cross-correlation between pelvis and shoulder movement*

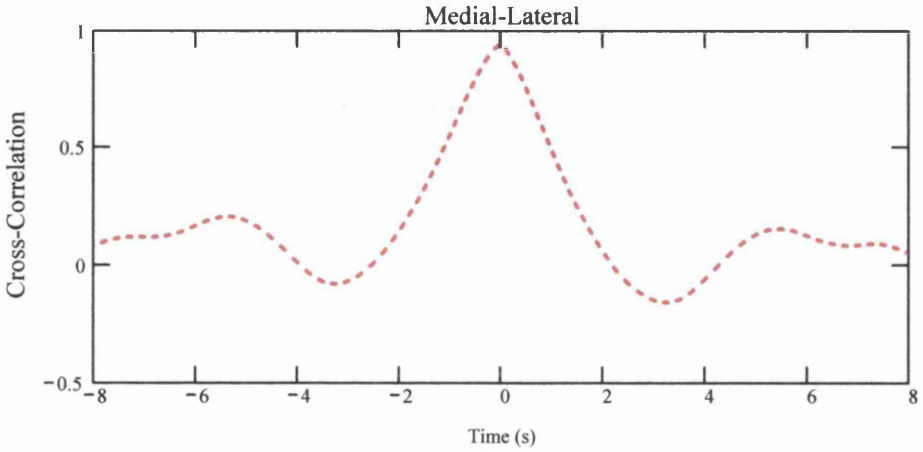
It is possible for the movements of two body segments to be correlated but not synchronous. Movements may show a time lag or phase difference between successively moving segments because of physical or other physiological factors. To determine whether a time lag existed between the movement of the pelvis and shoulders, a cross-correlation analysis was performed on 25 subject trials. Figures 3.1.5a and b are the cross-correlation functions determined for one subject trial in the A/P and M/L axes respectively.

**Figure 3.1.5:** Cross-correlation functions between pelvis and shoulder marker movement for one subject trial in the A/P (a) and M/L (b) axes. The abscissa gives the time lag between the pelvis and shoulder movement. Ordinate: Cross-correlation coefficient. For the significant peak, $r = 0.87$ and 0.94 in the A/P and M/L axes respectively.

a)




b)



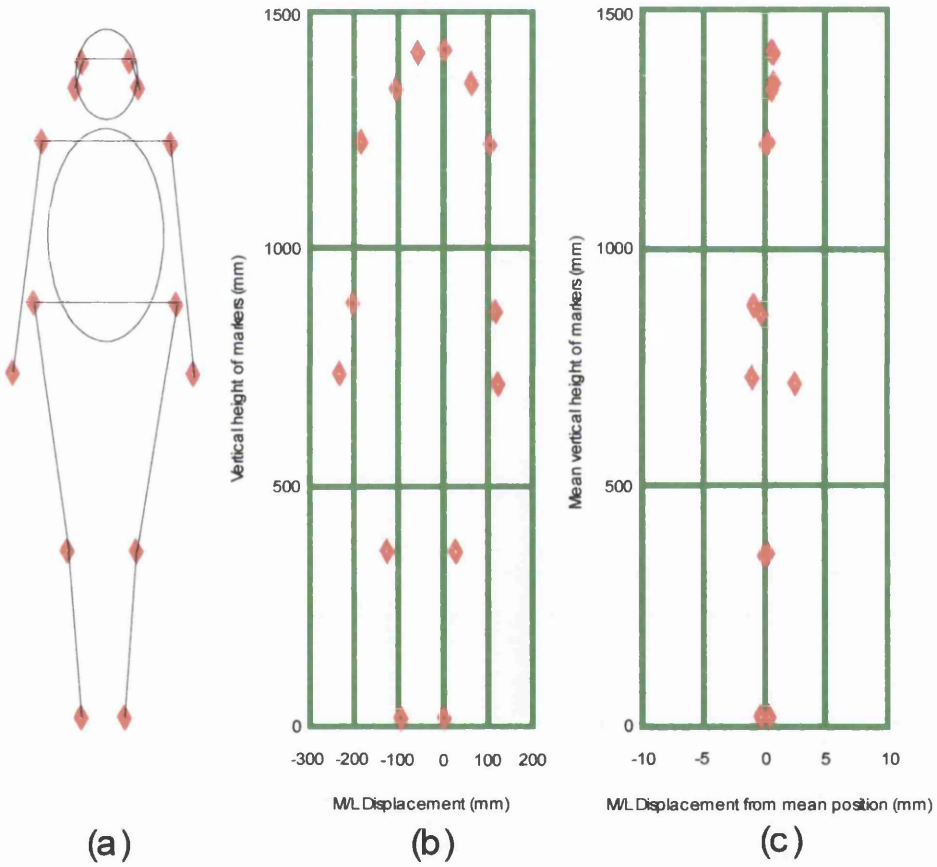
For the majority of cases in the A/P axis (17 out of 25) there was a well defined maximum occurring at t = 0 seconds indicating no time lag between the pelvis and shoulder movement (Fig. 3.1.5.a). However, in 8 records, the maximum peak occurred at either -0.1 seconds or 0.1 seconds. This apparent time shift in the peak appeared random in nature and was probably caused by the existence of noise in the records rather than being an indication of a true time lag between segments. Results obtained in the M/L axis were similar, with 21 out of the 25 records showing the maximum peak of the cross-correlation function at t = 0 seconds. In general, it appears the movements of the segments are synchronous and the correlation coefficient adequately describes the movement relationship between the segments.

*Defining the inverted pendulum model from the marker data*

To translate the marker co-ordinates recorded by the *Coda-mpx30* to an inverted pendulum model of the body, the mean position of each of the fourteen markers over the duration of the trial was calculated in three axes, anterior-posterior (A/P), medial-lateral (M/L) and vertical. Records of movement of the individual markers from their mean position in the A/P and M/L axes were determined as before. The movement from mean position for each marker in the A/P and M/L axes was plotted against the marker mean vertical position for a selection of the 3000 time points making up the record. In these plots, the 3-D co-ordinates of the markers in space becomes reduced to movement of the markers from a mean position (zero). Figures 3.1.6b and 3.1.6c overleaf highlight the difference between the raw movement records acquired from the *Coda-mpx30* and the movements from mean position records respectively. In this example, the plot of raw marker movement against vertical position in the M/L axis (Figure 3.1.6b) shows the separation of the markers relative to each other as well as their movement relative to the origin (defined by the right ankle marker at t = 0 seconds). However, on the plot of movement from mean position versus vertical position

(Figure 3.1.6c) data points representing markers at the same vertical position are separated by only a few millimetres and even overlap in several cases.

**Figure 3.1.6** **a)** Diagram showing the placement of markers on the body. **b)** Plot of raw marker position in the M/L axis at t = 54.5 seconds. Note that the origin of two co-ordinate system is the right ankle. **c)** Plot of movement from mean position along the M/L axis versus vertical mean position at t = 54.5 seconds.



This 'reduction of body width' enables a linear regression line to be fitted to the data points corresponding to the *A/P-shift* and *M/L-shift* of the markers as a function of height from the ankle. To model an inverted pendulum motion i.e. rotation of the body about the ankles the regression line is constrained through zero, the slope of the line $b$ is given by the equation:

$$b = \frac{\sum_{i=1}^{n} x_i \cdot y_i}{\sum_{i=1}^{n} (x_i)^2}$$

Where $x_i$ equals the mean vertical height of a marker and $y_i$ equals the movement of the marker from its mean position along the A/P or M/L axes, at the instant considered.

Changes in the slope of the regression line and in movement of the markers from their mean position were observed over a selection of the 3000 time points of the trial and matched to the corresponding time points on the records obtained from the force plate (Figures 3.1.7a and 3.1.7b). The results show that when there is a sway movement registered by the force plate in either the A/P or M/L direction there is a corresponding change in the slope of the regression line due to the tendency of the markers to move together. The markers furthest from the ankles (i.e. those on the head) tended to be displaced furthest from the mean position. In this respect, the regression line, and therefore the body moves in a way predicted by the single-link inverted pendulum model.

If we assume that the angle that the inverted-pendulum model of the body rotates about the ankle is small (i.e. $\tan(\theta) \sim \theta$), then $\theta$ is equal to the slope of the regression line. In other words, the slope of the regression line approximates to the angle of sway of the inverted pendulum model of the body.

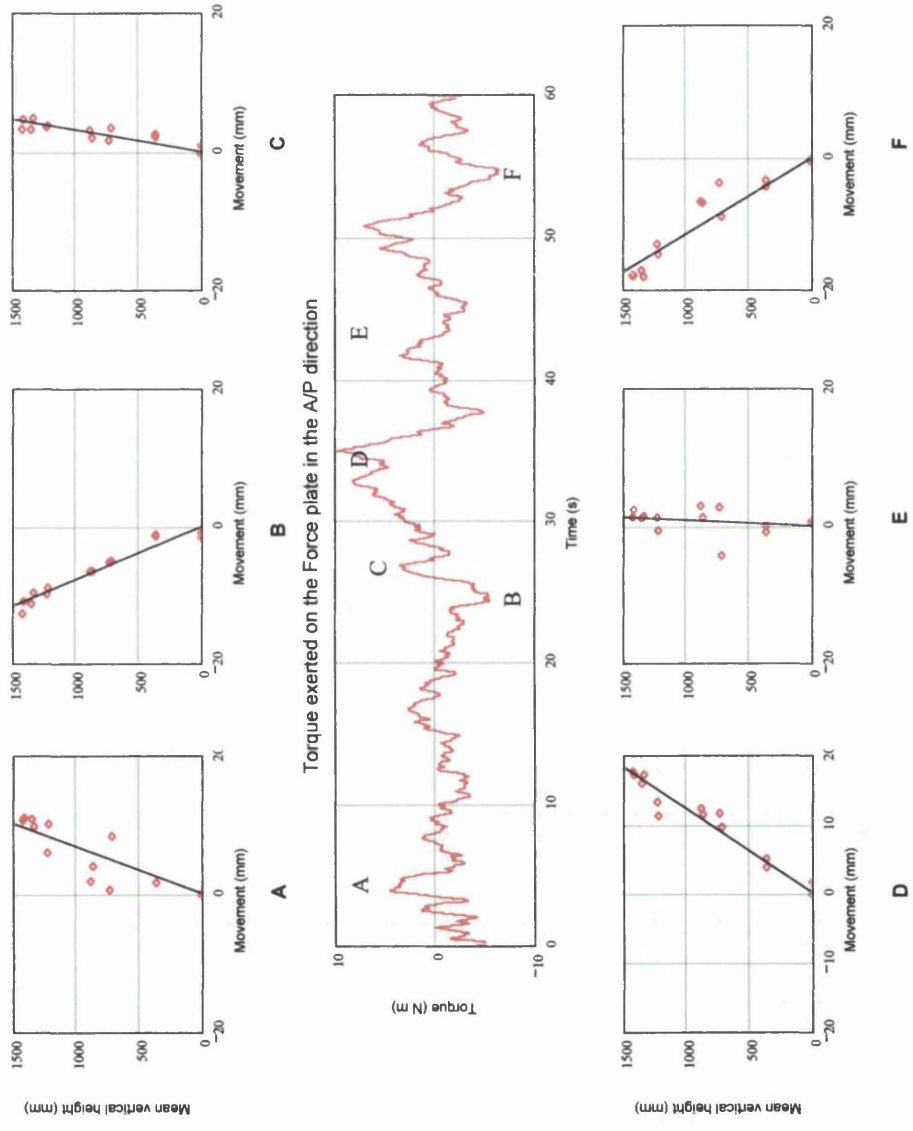



**Figure 3.1.7a:** Comparison of the position of the markers and the slope of the regression line with the force plate record at 6 selected time points A-F in the A/P axis. The results show that when there is a sway movement registered by the force plate e.g. at the time point designated A, there is a corresponding change in the slope of the regression line due to the tendency of the markers to move together. When a sway movement occurs in the opposite direction e.g. at time point B there is also a reversal in the slope of the regression line.

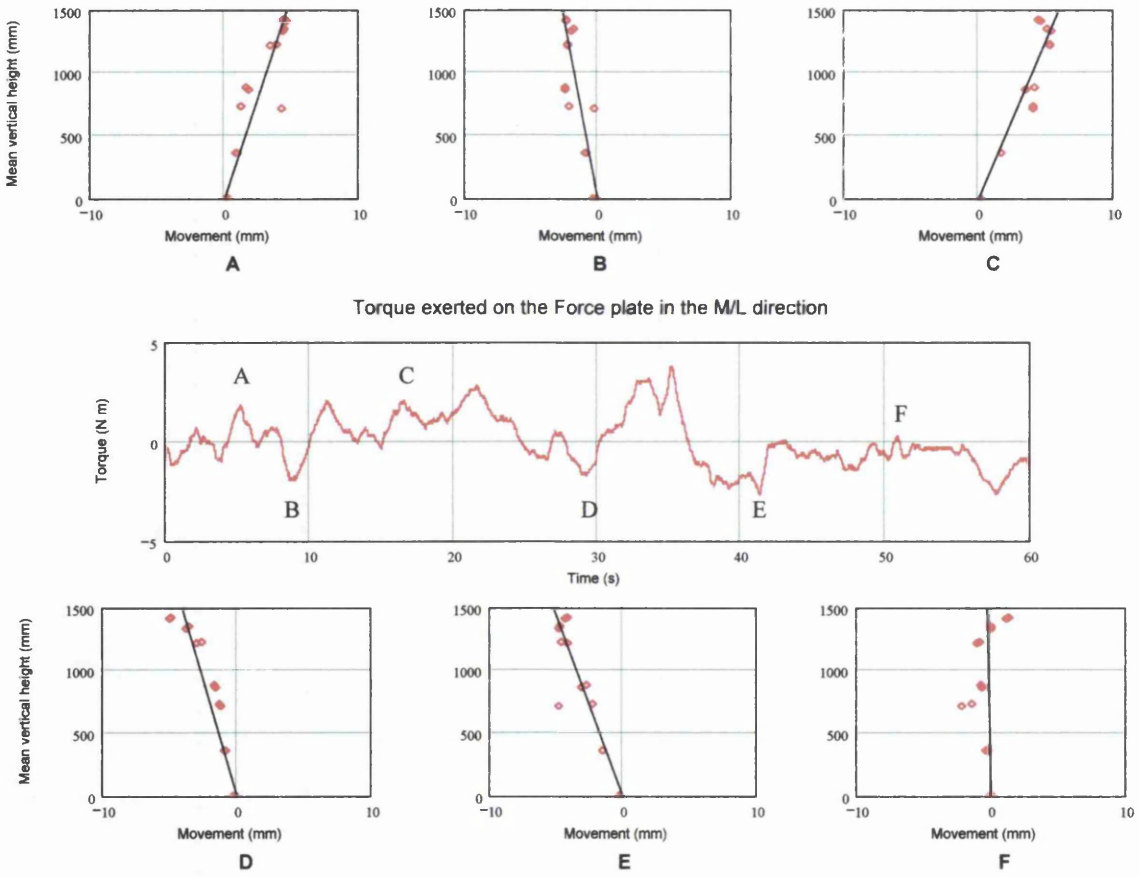



**Figure 3.1.7b:** Comparison of the position of the markers and the slope of the regression line with the force plate record at 6 selected time points A-F in the M/L axis. The results show that when there is a sway movement registered by the force plate e.g. at the time point designated A, there is a corresponding change in the slope of the regression line due to the tendency of the markers to move together. When a sway movement occurs in the opposite direction e.g. at time point B there is also a reversal in the slope of the regression line.

*The relationship between output from the force-plate and the inverted pendulum model*

The relationship between the angular position of the inverted pendulum model and the output from the force plate was investigated further by determining the time-series correlation between the two records. The movement of the COP (mm) from its mean position, measured from the force plate, was correlated with the angular motion of the inverted pendulum (radians) for each subject trial. Each correlation coefficient calculated underwent a logarithmic transformation and a resultant value for each subject was obtained by averaging the values for the three trials. The geometric mean and range values obtained for the young and elderly groups are presented in Table 3.1.5. The results show a high positive correlation between the movement of the COP and the angular motion of the inverted pendulum model, in both A/P and M/L axes and in both the young and elderly subject groups. The values for $r$ are high in both groups indicating that a strong association exists between the movement of COP and the angular motion of the inverted-pendulum model in both the young and elderly. A students $t$-test was used to compare the geometric means of the two groups and a summary of the results is shown in Table 3.1.5. In the A/P axis the young adults had a significantly greater correlation between the two records than the elderly group ($t = 1.68$, $p = 0.021$, d.f = 40). In the M/L axis, the reverse was found, with the elderly group showing a greater positive correlation between the COP movement and the inverted pendulum model ($t = 3.02$, $p = 0.004$, d.f. = 40).

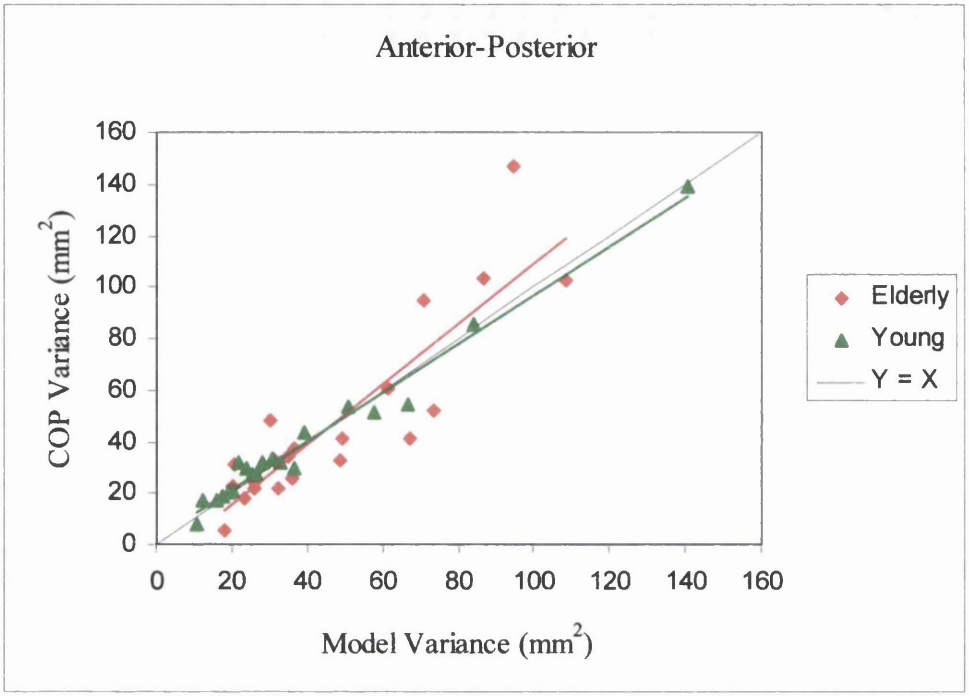
**Table 3.1.5:** Summary of the correlation coefficients calculated for movement of the COP (mm) correlated with the angular motion of the inverted-pendulum model (radians) during quiet stance. Mean ± 1 S.E.M. calculated from transformed data. Also shown are the results of a two-tailed *t*-test comparing the mean transformed values obtained for the two groups, young (N = 20) and elderly (N = 22).

| AXIS | GEOMETRIC MEAN (± 1 S.E.M) | | *P* |
|---|---|---|---|
| | Young | Elderly | Young = Elderly |
| Anterior-Posterior | 0.95<br>(0.94, 0.96) | 0.92<br>(0.91, 0.93) | 0.021* |
| Medial-Lateral | 0.91<br>(0.90, 0.92) | 0.95<br>(0.94, 0.95) | 0.004** |

*, ** Denotes significant difference $p<0.05$ and 0.005 respectively

The close association between COP movement and the movements of the model are also illustrated by the linear regression of COP movement variance upon the model movement variance (Figures 3.1.8a and b). To obtain the variance of the model movement, the angular motion of the model (radians) was converted to distance moved in mm and then multiplied by an estimate of the height of the individual's COG (55.72% of the height above the ankle, following Dempster 1955). The variance of the model movement calculated for each subject (mean of the three trials) was plotted against COP movement variance values. The results show that for both young and elderly subject groups, the COP and model movement variances are approximately equal.

A)




B)

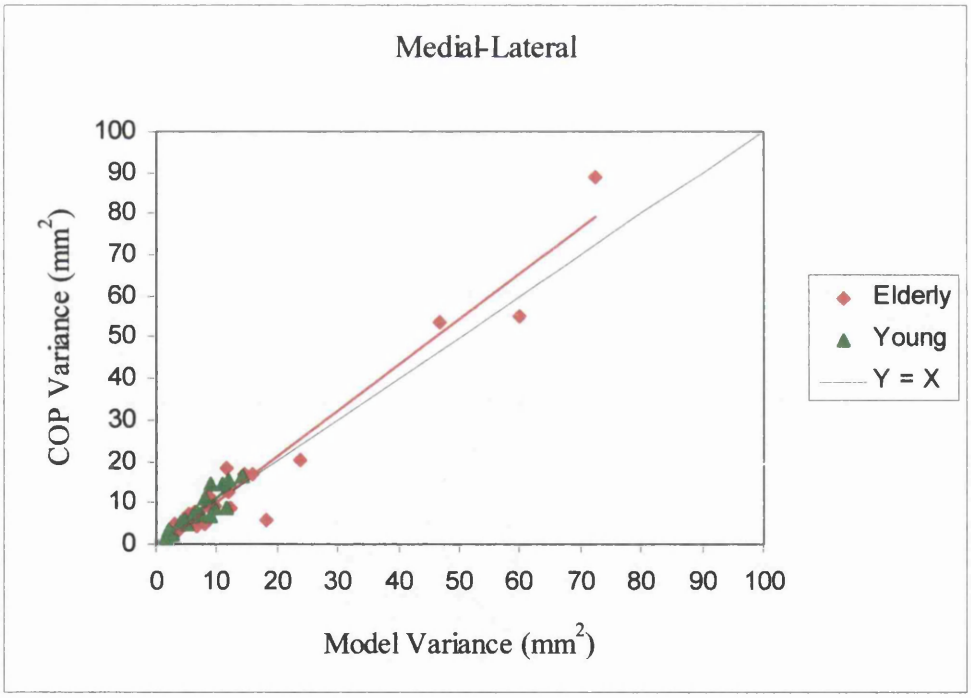


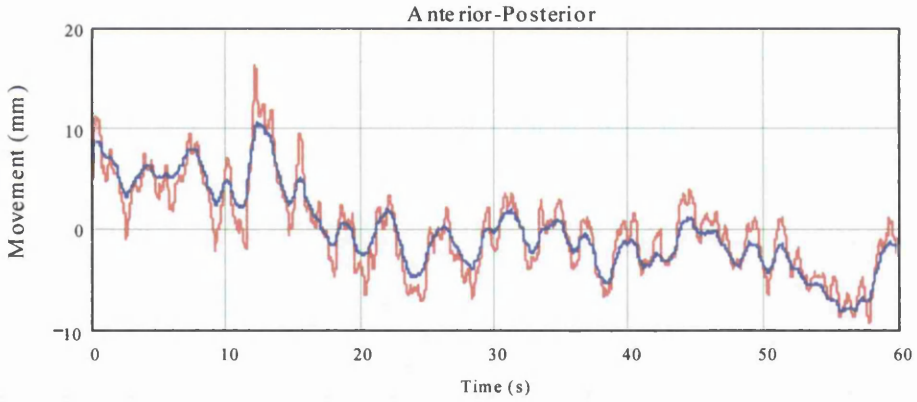

**Figure 3.1.8:** Linear regression of COP movement variance upon movement variance of the model. A) Anterior-Posterior, equation of the regression line for elderly, y = 1.17x – 7.68, $R^2$ = 0.78, young, y = 0.94x + 2.81, $R^2$ = 0.98. B) Medial-Lateral, elderly, y = 1.11x – 1.17, $R^2$ = 0.95, young, y = 1.14x + 0.06, $R^2$ = 0.84. Individual points represent resultant variance values for a subject (average of three trials).

*Angular motion of the inverted pendulum model represents movement of the COG*

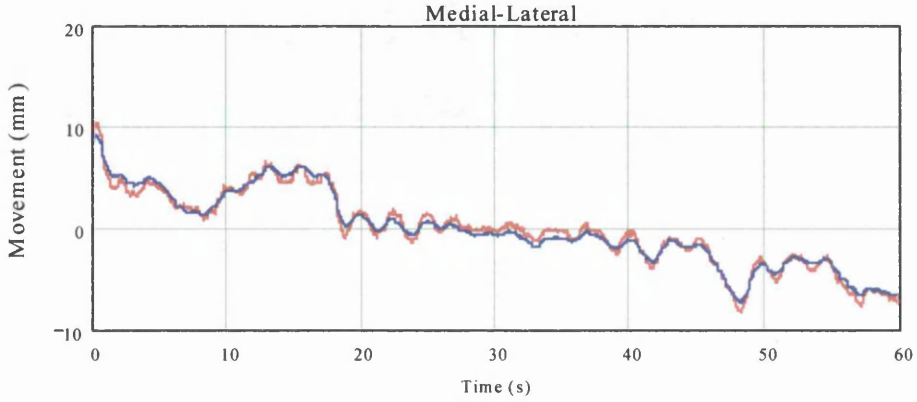
For each subject trial, a linear regression function was performed on the plots of COP movement against angular position of the inverted-pendulum model. The slope of this regression line (regression coefficient) was used to optimally scale the model record to the same units of measure as the movement of COP record (mm). Figure 3.1.9a and b are examples of the movement of COP and the scaled inverted-pendulum model records in both the A/P and M/L axes for a single subject (one of three trials).

**Figure 3.1.9:** Time series graph of COP movement and the product of angular position of the model and the regression coefficient, *b* (see text). The data plotted represents the results for one trial of an elderly subject, a) A/P and b) M/L axis.

a)



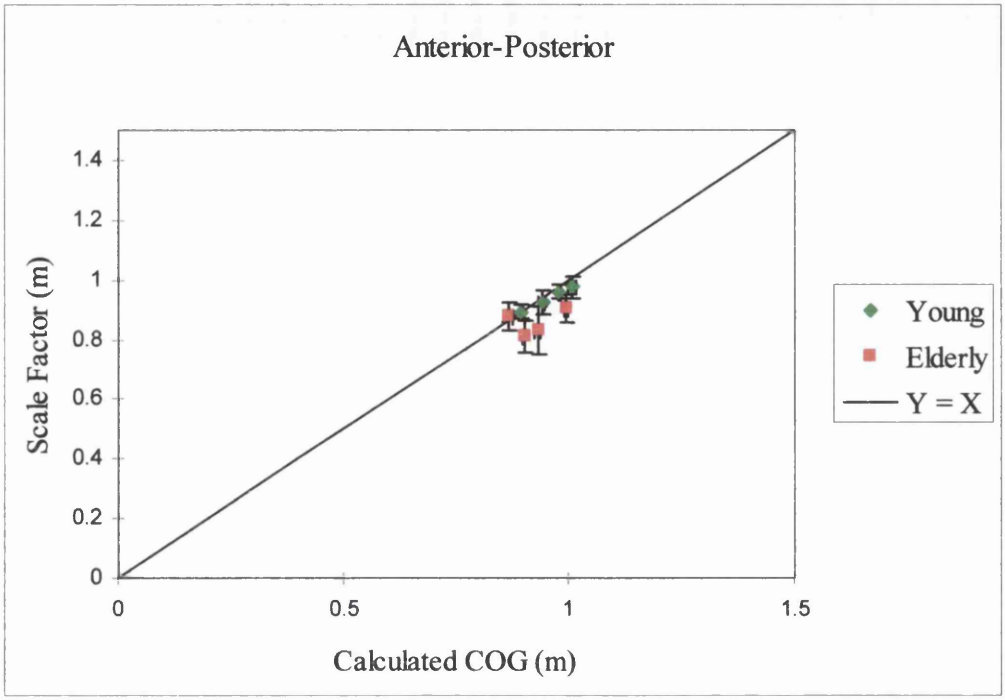

b)



What is the justification for using the regression coefficient to scale the model record? The inverted-pendulum model was derived from horizontal movements of the body segments and therefore reflects the angular position of the body's Centre of Mass (COM). In the analysis of a human body subject to gravitational force, the COM may also be referred to as the Centre of Gravity (COG), a point about which the sum of the torque produced by the weights of the body segments is equal to zero. The record of angular motion of the inverted-pendulum model can be converted to movement of the COG from its mean position by multiplying the angular position of the model by a factor that equates to the vertical height of the body's COG. If the scale factor is a reasonable estimate of the height of the COG, Figures 3.1.9a and b represent plots of movement of the COP and movement of the COG in the A/P and M/L axes respectively. The relation between the scale factor and the body's 'real' COG was examined by comparing the scale factor to a value of the height of the COG calculated using parameters based on the work of Dempster (1955).

For each subject, scale factors for the A/P and M/L axes were obtained by averaging the values obtained for the regression coefficient across the three trials. The height of the COG above the ankle for each subject was estimated following Dempster (1955) to be 55.72 % of body stature. For each subject group (young and elderly), the scale factor and estimated COG for an individual was sorted by the COG data. The results for each group were then divided into 4 sub-groups and the mean and S.E.M. for each sub-group calculated. Figures 3.1.10a and b depict plots of these results in the A/P and M/L axis respectively. Considering the approximations used for calculating the body's COG and the assumptions of the inverted-pendulum model, there is good agreement between the values obtained for the scale factor and the estimated height of the COG. Therefore it seems reasonable to use the regression coefficient as an optimal scale factor to convert the records of angular motion of the inverted-pendulum model to movement of the body's COG.

a)




b)

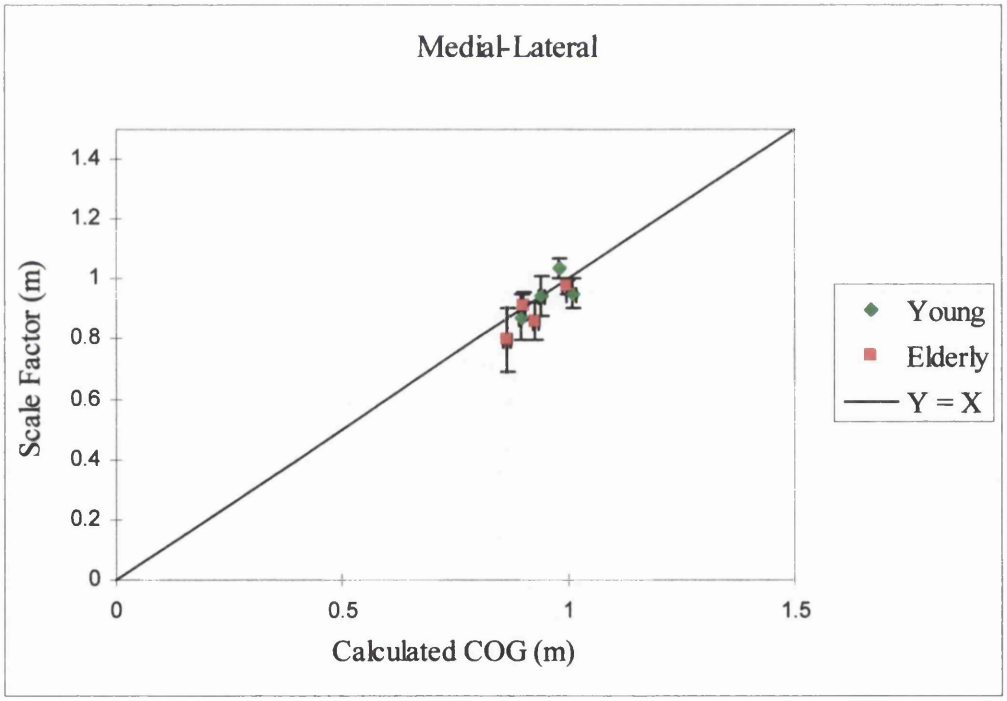



**Figure 3.1.10:** Comparison of the scale factors (regression coefficient of movement of COP vs model) with the expected value for the height of the COG calculated from anthropometric parameters. Data points represent group mean ± SEM. For 6 out 0f 8 groups N = 5, for the remaining two elderly groups N = 6.

*The involvement of reaction forces during quiet standing*

It is obvious from figures 3.1.9a and b that the movement of the COP is not identical to the movement of the COG. Although there is a high correlation between the two records the excursions of the COP are somewhat larger and more rapid than those of the COG. When the body is modelled as an inverted pendulum the product of body weight and the difference between the COP and the COG is the controlling torque at the ankle (Ruder *et al.*, 1989).

In angular terms Newton's second law may be stated as:

$$T = I\alpha$$

Where a net torque (T) produces angular acceleration ($\alpha$) of a body that is directly proportional to the magnitude of the torque and in the same direction as the torque. By rearranging the equation to:

$$\alpha = \frac{T}{I}$$

It becomes obvious that $\alpha$ is also inversely proportional to the body's moment of inertia (I).

*Mean spectral frequency of the COP movement and residual movement (COP - COG)*

Gurfinkel (1973) found that the difference between the COP and COG was less than 10% at sway frequencies below 0.2 Hz. However, the higher the frequency of body sway, the greater the acceleration, and the larger the contribution of inertial forces. For frequencies of 0.5 Hz, inertial forces may determine approximately 50% of the COP movement and at a frequencies larger than 1 Hz, the movement of the COP reflects mainly inertial forces (Gurfinkel, 1973).

A Fast Fourier Transform (FFT) was used to estimate the frequency composition of the COP movement and the residual movement (COP - COG) records. From the calculated power spectra the mean spectral frequency (centroidal frequency) of the record was determined (See Methods). A summary of the mean spectral frequencies of the COP movement and residual movement records for the two subject groups are displayed in Table 3.1.6. A *t*-test was used to compare the mean spectral frequencies of the two groups. A summary of these results is also presented in Table 3.1.6.

**Table 3.1.6:** Summary of the mean spectral frequency (Hz) of the COP movement and the residual movement (COP - COG) during quiet stance, eyes closed trials, for the young (n = 20), and elderly (n = 20). Also shown are the results of a *t*-test comparing the means of the two groups.

| AXIS | GROUP MEAN ± S.E.M. (HZ) | | *P* |
|---|---|---|---|
| | Young | Elderly | Young = Elderly |
| A/P | 0.20 ± 0.018 | 0.23 ± 0.019 | 0.261 |
| Residual A/P | 0.64 ± 0.044 | 0.68 ± 0.046 | 0.482 |
| M/L | 0.25 ± 0.013 | 0.20 ± 0.013 | 0.014* |
| Residual M/L | 0.63 ± 0.030 | 0.61 ± 0.047 | 0.709 |

* Denotes significant difference, $P < 0.05$.

The mean spectral frequency of the COP movement records for both the young and elderly is close to the value of 0.2 Hz, the frequency at which Gurfinkel (1973) estimated the inertial forces to make a minimal contribution to the COP movement. In the M/L axis, the elderly group had a significantly lower mean spectral frequency for the COP record than the young group suggesting that there was a greater concentration of power at low frequencies in the elderly group. There was no difference between the two groups for the mean spectral

frequency in the A/P axis. The mean spectral frequencies calculated for the residual record (COP - COG), were more than double that of the COP record for both subject groups and axes. However, there was no significant difference between the two groups for the residual mean spectral frequency in either axis.

*The relationship between residual torque and angular acceleration of the model*

How much of the observed residual COP movement is determined by angular acceleration of the inverted pendulum model? To investigate this, the association between residual torque and angular acceleration of the model was determined. The residual torque (N.m) was calculated by multiplying the record of residual movement (m) by the subject's body weight (N). The angular acceleration of the inverted-pendulum model (radians.sec$^{-2}$) was derived from differentiating the angular velocity (radians.sec$^{-1}$) of the model that in turn came from differentiating with respect to time, the record of angular position (radians). Time series correlation coefficients were calculated for the residual torque and angular acceleration records. Examples of this correlation for one elderly subject's trial in the A/P and M/L axes are depicted in Figures 3.1.11a and b respectively. The correlation coefficient data was transformed using the procedure described in the methods and a resultant value was obtained for each subject. The data set failed the normality test even after transformation so a Mann-Whitney Rank Sum Test was used to compare the medians of the two groups. A summary of the results is presented in Table 3.1.7.

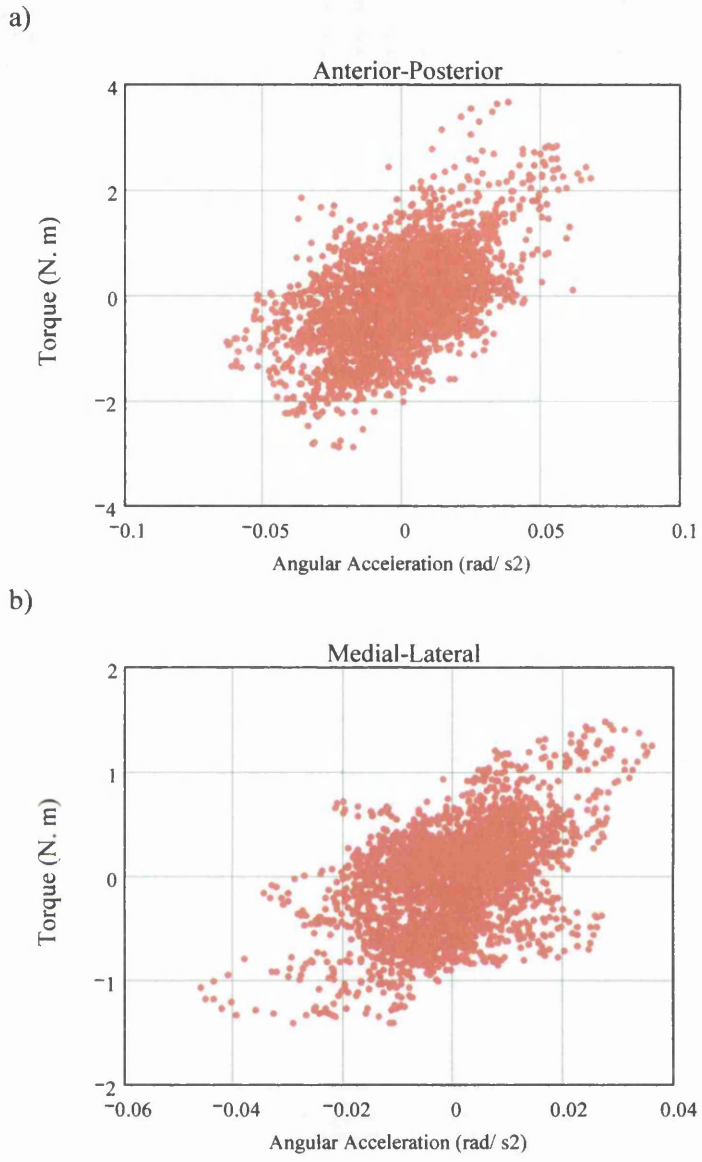



**Figure 3.1.11:** Correlation between residual torque (N.m) and angular acceleration (rad/s$^2$) of the inverted pendulum model for an individual subject trial. a) Anterior-Posterior, $r$ = 0.55. b) Medial-Lateral, $r$ = 0.49.

**Table 3.1.7:** Summary of the median correlation coefficients calculated for the residual torque (N.m) correlated with angular acceleration of the inverted-pendulum model (radians.sec$^{-2}$), for the young (n = 20), and elderly (n = 22). Also shown are the results of a Mann-Whitney Rank Sum test comparing the medians of the two groups.

| AXIS | MEDIAN | | *P* |
|---|---|---|---|
| | Young | Elderly | Young = Elderly |
| A/P | 0.31 | 0.44 | 0.089 |
| M/L | 0.34 | 0.43 | 0.121 |

Generally, the results show a that a positive correlation exists between the residual torque and the angular acceleration of the inverted-pendulum model for both the young and elderly. The correlation is significantly different from zero, based on the knowledge that there are 2998 degrees of freedom (d.f.) and the value of *r* that is required for significance at the 5% level with 100 d.f is 0.195. The results of the Mann-Whitney test showed there was no significant difference between the medians of the two groups in either the A/P or M/L axis.

*Estimating 'I' the body's moment of inertia from the residual torque and angular acceleration records.*

It was noted in the previous section that the angular acceleration was inversely proportional to the body's moment of inertia. An estimate for 'I', the body's moment of inertia, was obtained by calculating the slope of the linear regression line for residual torque (T) upon angular acceleration ($\alpha$).

Linear regression gives the equation of the straight line that describes how the *y* variable increases (or decreases) with an increase in the *x* variable. Conventionally, there is a 'one way' relationship between the two variables, such that changes in one (the independent variable) are thought of as causing changes in the other (the dependent variable) and the

reverse relationship has no real meaning. The independent variable is plotted on the *x*-axis and the dependent variable on the *y*-axis. The equation of the regression line is:

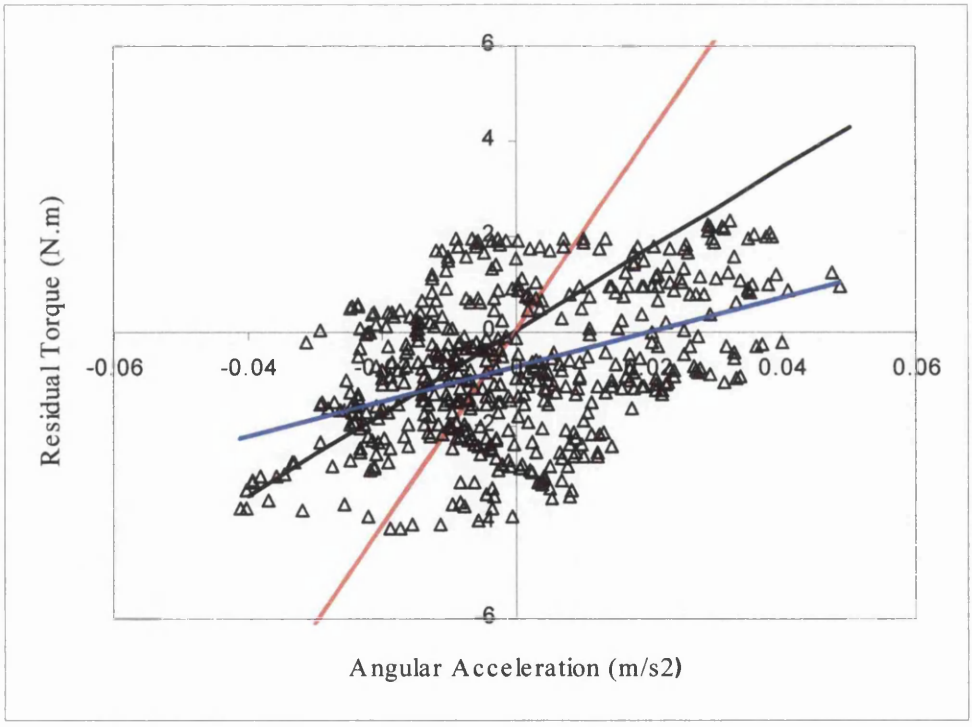
$$y = a + b\,x$$

Where **a** is the intercept and **b** is the slope of the line. The values for **a** and **b** are calculated so as to minimise the sum of the squared vertical distances of the points from the line. This is called the least squares fit. The relationship between residual torque and acceleration of the COG is not strictly a 'one way' relationship, neither variable can be classified as being 'dependent' upon the other. In fact both the torque and acceleration of the COG are reliant on muscle contraction. Despite this unconventional relationship between the variables it is still possible to use linear regression analysis to obtain a reasonable estimate for 'I'. A major assumption of regression analysis is that errors only occur in the 'y' variable. However, with the regression of residual torque upon acceleration of the COG there may be appreciable error associated with both '*x*' and '*y*'. The errors in '*x*' (the acceleration) may be considerable due to the differentiation process used to obtain the record. Differentiation tends to amplify the high-frequency components of the angular position data i.e. the noise associated with the body segment position data. Therefore it is necessary to obtain an estimate of 'I' which will take into account errors in both 'T' and 'α'. If the true errors in 'T' and 'α' were known then it would be possible to weight the estimate of 'I' (the slope of the regression line) accordingly. In this situation, where the errors are unknown it is necessary to obtain two estimates for 'I':

1. ' $I_1$' equals the slope of the regression line fitted to the plot of residual torque against α, where the error is assumed to occur in 'T'.
2. ' $I_2$' equals the inverse of the slope of the regression line fitted to a plot of 'α' against 'T', where the errors are assumed to occur in α.

The ratio of $I_1$ / $I_2$ is equal to the correlation coefficient calculated for the association between the 'T' and 'α'. Two estimates of 'I' are obtained for each subject trial in both the M/L and A/P axes. To obtain a single 'I' value for each axis, the values obtained for $I_1$ and $I_2$ undergo a logarithmic transformation. The transformed values are then averaged and the antilog of the mean, the geometric mean, is used as the estimate of 'I' in the M/L or A/P axis. Figure 3.1.12 illustrates the linear regression of 'T' on 'α' giving ' $I_1$' (blue line) and a line whose slope ($I_2$ ) is equal to the inverse of the slope of the regression line fitted to a plot of 'α' against 'T' (red line). The slope of the black line is equal to the geometric mean of $I_1$ and $I_2$ and is the estimate of 'I' obtained for the trial in the specified axis.

**Figure 3.1.12:** Linear Regression of residual torque upon angular acceleration calculated for a 10 second time interval extracted from an individual subject trial (blue line). Equation of the blue line: y = 36.227x - 0.7412. The equation of the red line: y = 208.0x. Therefore $I_1$ = 36.2 kg.m$^2$ and $I_2$ = 208.0 kg.m$^2$ . Equation of black line equals y = 86.0x.



The product of the angular acceleration '$\alpha$ ' and the estimate of 'I' was plotted against time along with the record of residual torque. Figures 3.1.13a and b represent examples of these plots for one elderly subject in the A/P and M/L axes respectively. It is evident from the figures that many of the high-frequency components present in the residual torque record are due to angular acceleration of the inverted pendulum model of the body.

Figures 3.1.14a and b represent plots of the torque remaining once the contribution of inertial forces has also been removed. Components such as respiration and cardiac events may be responsible for some of this remaining torque measured by the force plate.

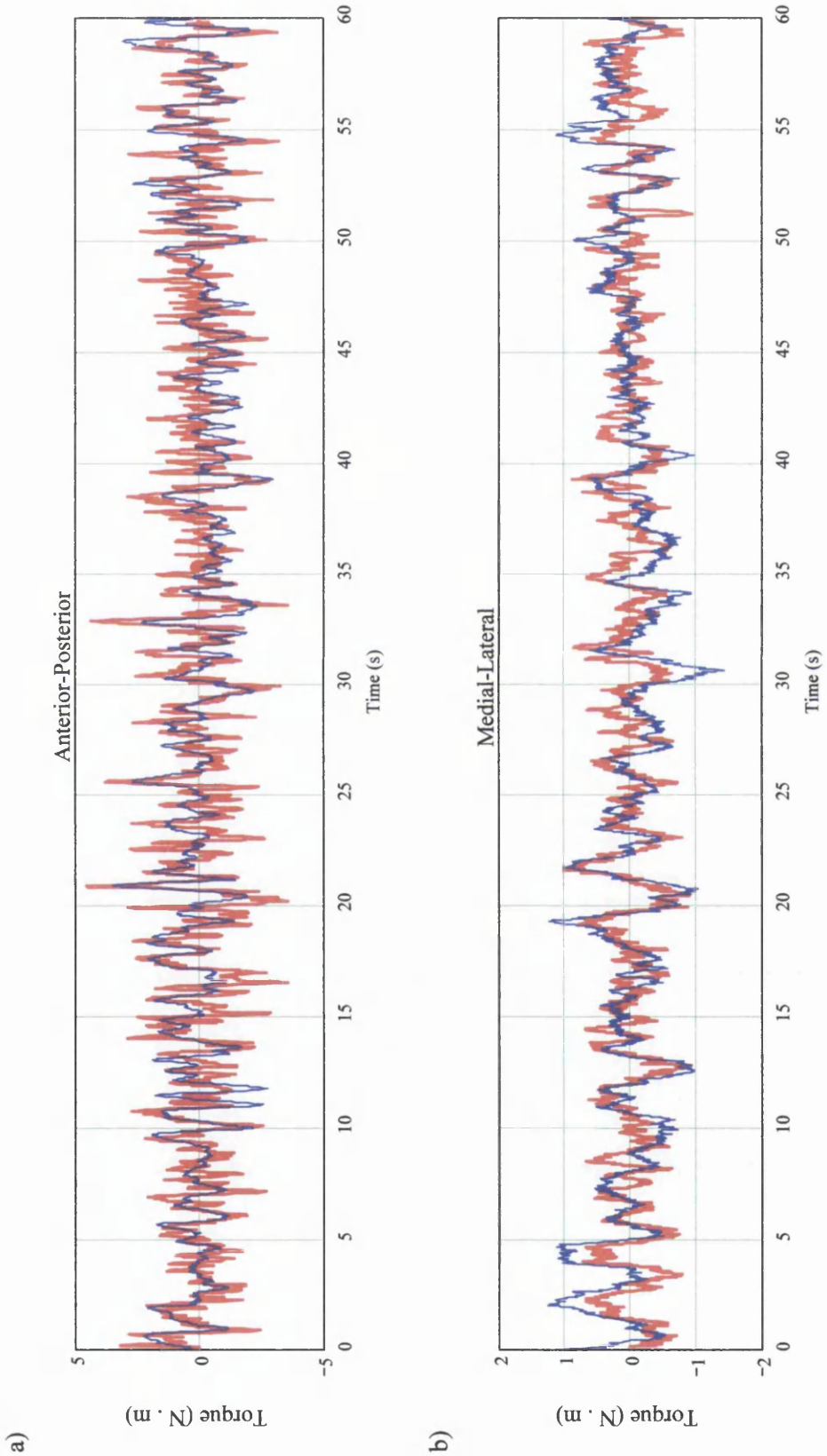



**Figure 3.1.13:** Plots of residual torque (blue) and the product of the angular acceleration 'α' and the estimate of 'I' (red) for an individual subject trial in the anterior-posterior and medial-lateral axes.

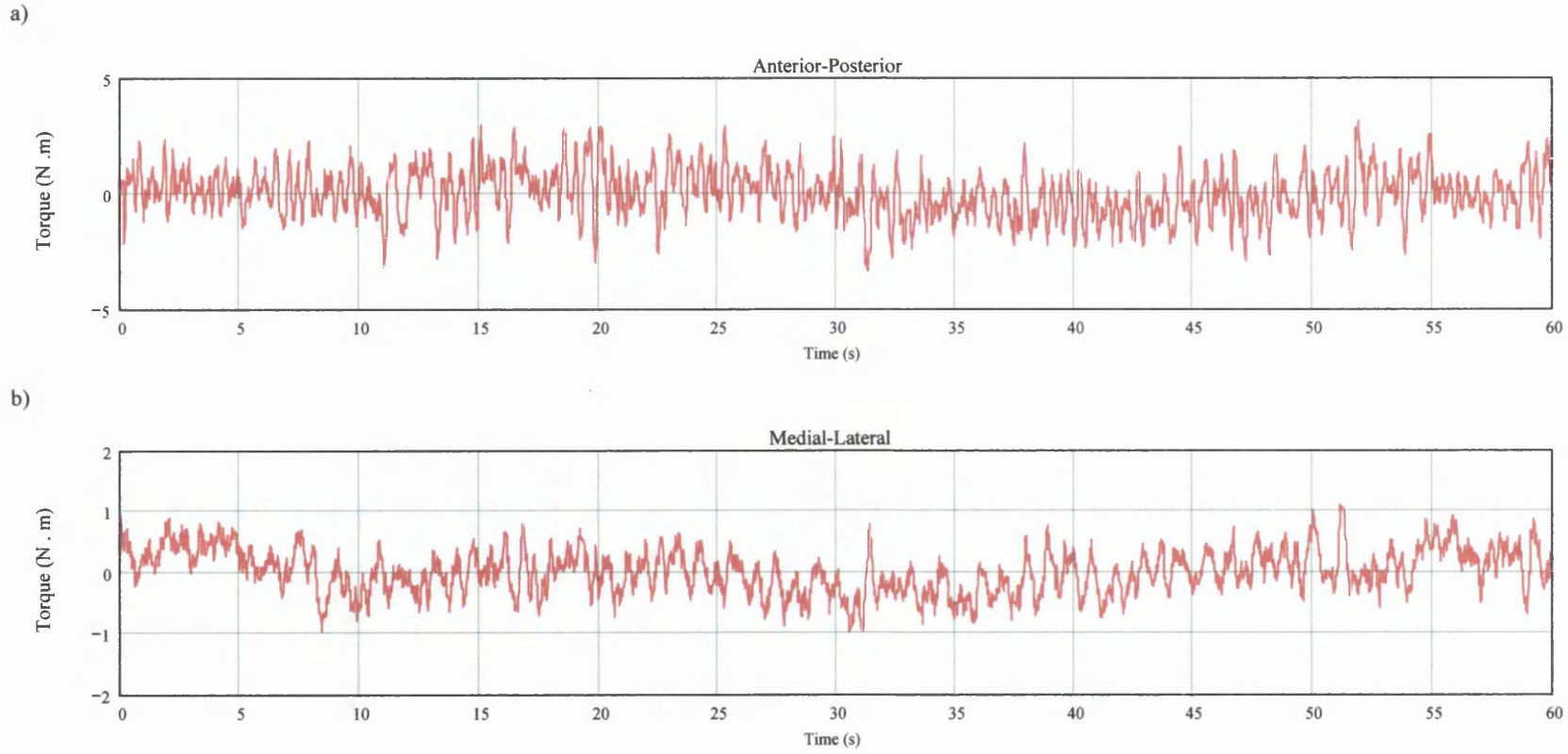



**Figure 3.1.14:** Plots of torque remaining after removal of contribution of inertial factors to the residual torque. Results for an individual subject trial in the anterior-posterior (a) and medial-lateral axes (b).

To verify that the estimates of 'I' calculated from the linear regression analysis were a close approximation for the body's true moment of inertia these values were compared to an estimate of the body's moment of inertia calculated using anthropometric parameters (Dempster, 1955).

*Calculating the body's moment of inertia: Background*

The relationship between the residual torque about the ankle and the angular acceleration is dependent upon the moments of inertia of the body. Inertia is the body's tendency to resist acceleration and is directly proportional to the body mass. The greater the body mass, the greater the resistance to angular acceleration. The moment of inertia of a single body segment is the sum of the moments of inertia of all the mass particles the segment contains.

$$I = m_1 r_1^2 + m_2 r_2^2 + \ldots + m_n r_n^2$$

$$I = \sum_{i=1}^{n} m_i (r_i)^2$$

Where '$m$' is the particle's mass and '$r$' is the particle's radius of rotation.

It is obvious from the above equation that the distribution of mass with respect to the axis of rotation is more significant than the total amount of body mass in determining resistance to angular acceleration because '$r$' is squared. Because it is impractical to measure the distance of each particle of body mass from an axis of rotation, mathematical models based on cadaver studies (Dempster, 1955) are often used to calculate approximate values for the mass of a segment and the distribution of mass within the segment.

The value of 'I' also depends upon the point about which the rotation is taking place, and is at a minimum when the rotation takes place about the segment's centre of mass. However,

most body segments do not rotate about their mass centres but about a joint at either the proximal or distal end of the segment. The relationship between this moment of inertia and that about the centre of mass is given by the parallel-axis theorem. The equation for calculating the moment of inertia of the body segment becomes:

$$I = I_{01} + m_1 r_1^2$$

Where $I_0$ = moment of inertia of the segment about its' centre of mass, $r$ = distance between the centre of mass and the centre of rotation and $m$ = the mass of the segment.

This equation can be used to estimate the moment of inertia of the whole body by calculating the sum of the moments of inertia for all the body segments with respect to the point of rotation, in this case the ankle:

$$I = \Sigma (I_{01} + m_1 r_1^2) + (I_{02} + m_2 r_2^2) + \ldots + (I_{0n} + m_n r_n^2)$$

$$\mathrm{I} = \left[ \sum_{i=1}^{n} I_{oi} + m_i \cdot (r_i)^2 \right]$$

Where $I_0$ = moment of inertia of a segment about its' centre of mass, $r$ = distance between the centre of mass of the segment and the ankle, $m$ = mass of segment

*Calculation of the body's moment of inertia: Results*

For the purposes of this study the body was divided into 14 segments including head-neck, upper arm, forearm, hand, upper torso, and middle torso, lower torso, thigh and shank. The mass of the body segments, the centre of gravity of the segments and the principle moment of inertia of each segment about the frontal (M/L) and transverse (A/P) plane were estimated for each subject using the multiple regression equations of Zatsiorsky and Seluyanor (1983).

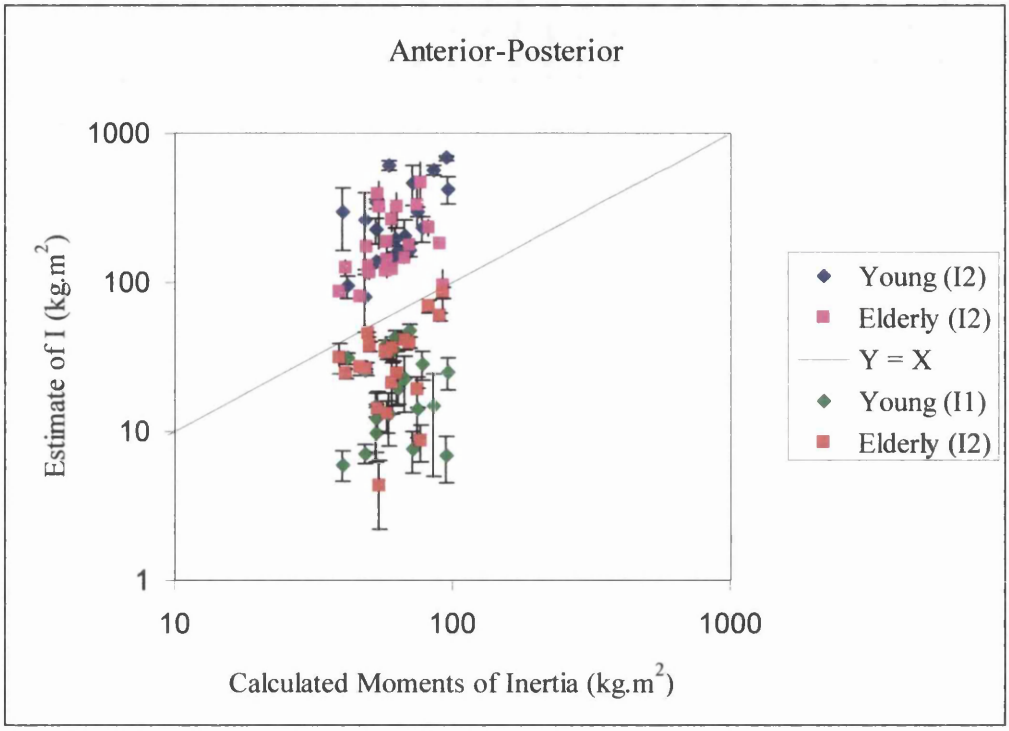
These were determined using a gamma-scanner and 100 young male subjects aged 23.8 ± 6.2 years (mean ± SD). The distance 'r' between the centre of gravity of each segment and the ankle, was estimated using body segment parameters (% stature) derived from the work of Dempster (1955), which entailed dismembering eight male cadavers aged 52 to 83 years. Tables of the estimated inertial characteristics of each subject in the A/P and M/L plane are to be found in Appendices A3-A6.

The values obtained for the whole body moment of inertia for each subject can only be a rough estimate of the true value. This is partly due to the parameters being derived from young men in the case of Zatsiorsky and Seluyanor (1983) and from a small number of older male cadavers in the case of Dempster (1955). Differences in the distribution of mass due to sex and age have not been fully accounted for. In addition, bone, muscle and fat have different densities and are distributed dissimilarly within individuals of the same age and sex. These differences in body type have also been ignored.

*Comparison of the two estimates for the body's moments of inertia*

Is the estimate of the body's moment of inertia calculated from linear regression analysis of angular acceleration and residual torque similar to that obtained using anthropometic parameters? The values obtained for $I_1$ and $I_2$ for each subject trial underwent a logrithmic transformation and the results for the three trials were averaged to give a single value for $I_1$ and $I_2$ in the A/P and M/L axes for each subject. Figures 3.1.15a and 3.1.16a illustrate plots of $I_1$ and $I_2$ against the estimate of 'I' calculated from the anthropometrc parameters in the A/P and M/L axes. The transformed values of $I_1$ and $I_2$ plotted in Figures 3.1.15a and 3.1.16a were then averaged and the antilog of the mean provided an estimate of 'I' in the M/L or A/P axis for each subject. In figures 3.1.15b and 3.1.16b the geometric mean values for each subject are plotted against their individual moment of inertia values calculated using anthropometric parameters.

A)




B)

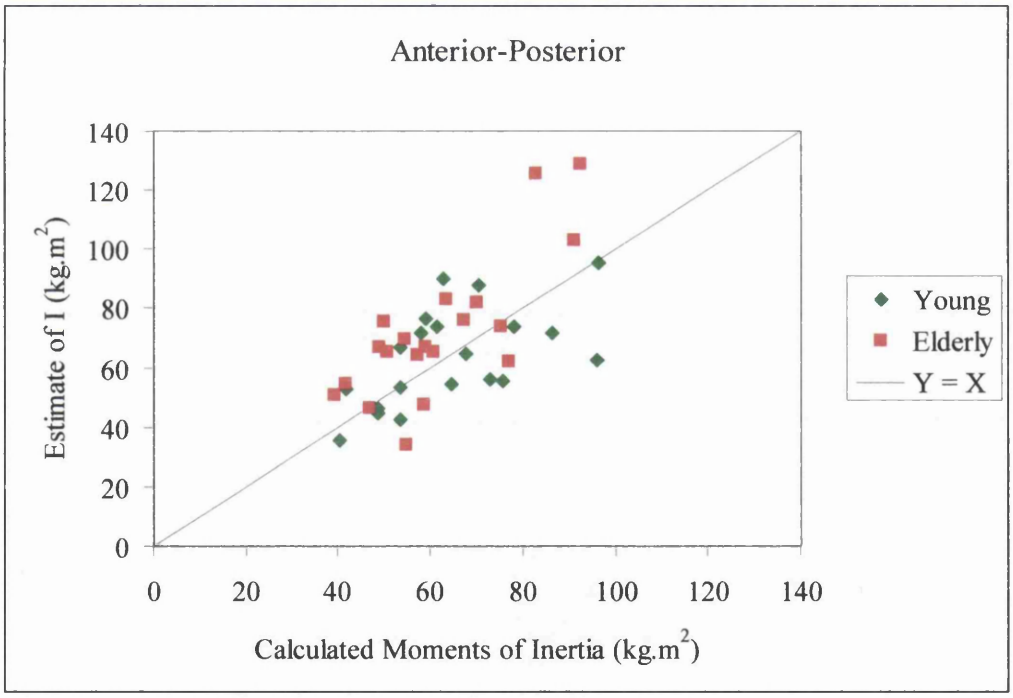
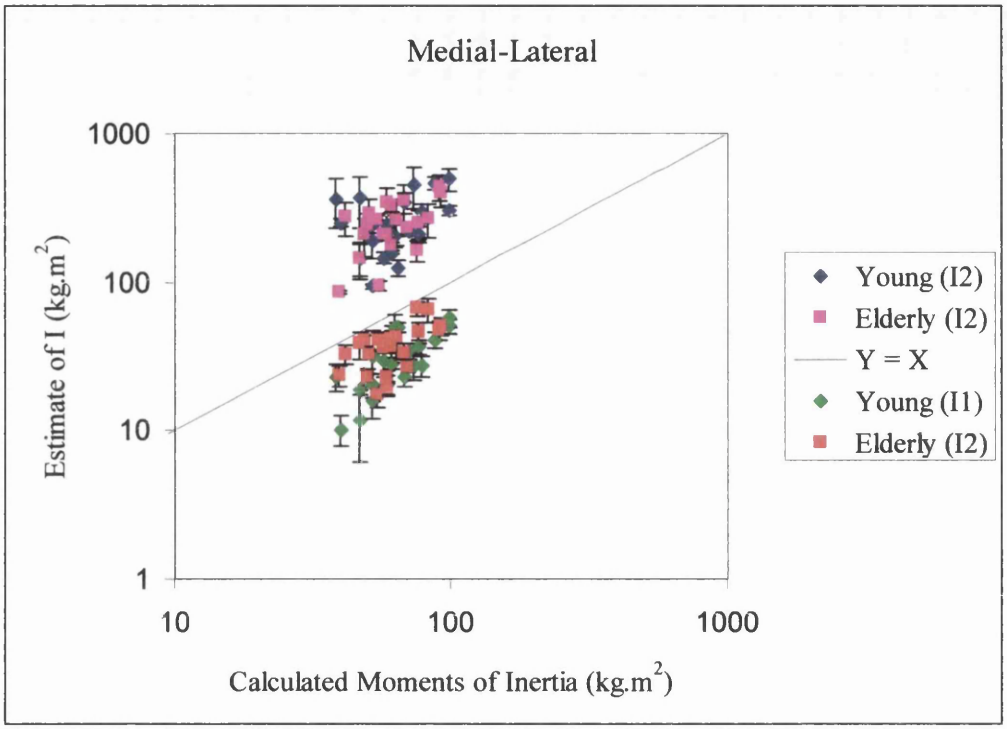



**Figure 3.1.15:** Anterior-posterior axis A) Individual moment of inertia estimates $I_1$ and $I_2$ calculated from the regression coefficients plotted against 'I' calculated using anthropometric parameters (double log axes). B) Geometric mean of $I_1$ and $I_2$ plotted against 'I'. For the young subjects, N=20 and for the elderly, N=21.

A)




B)

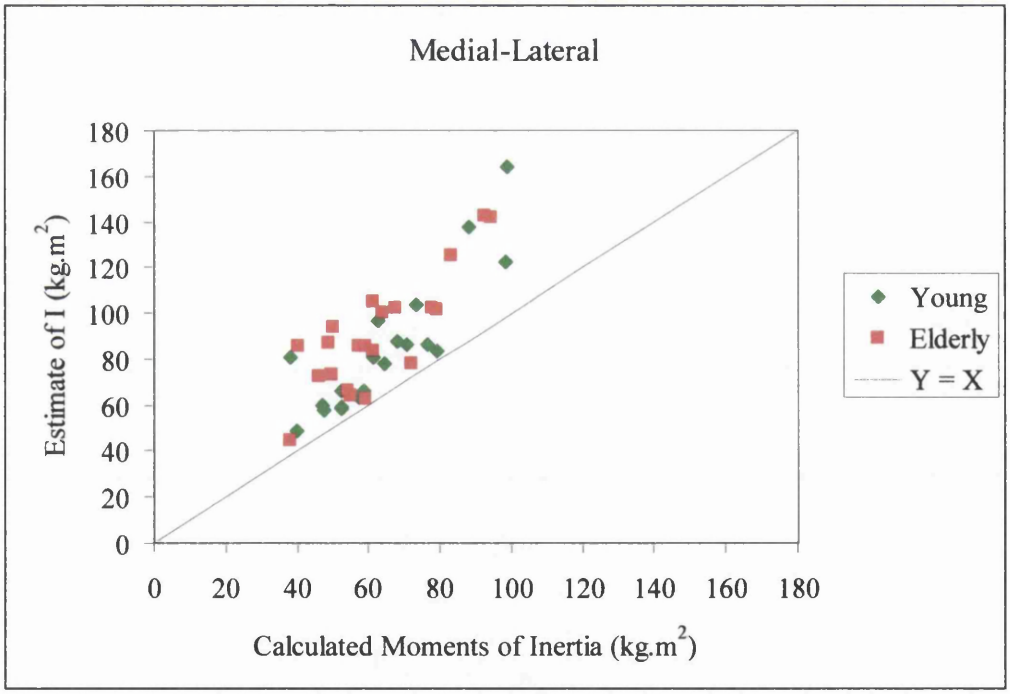


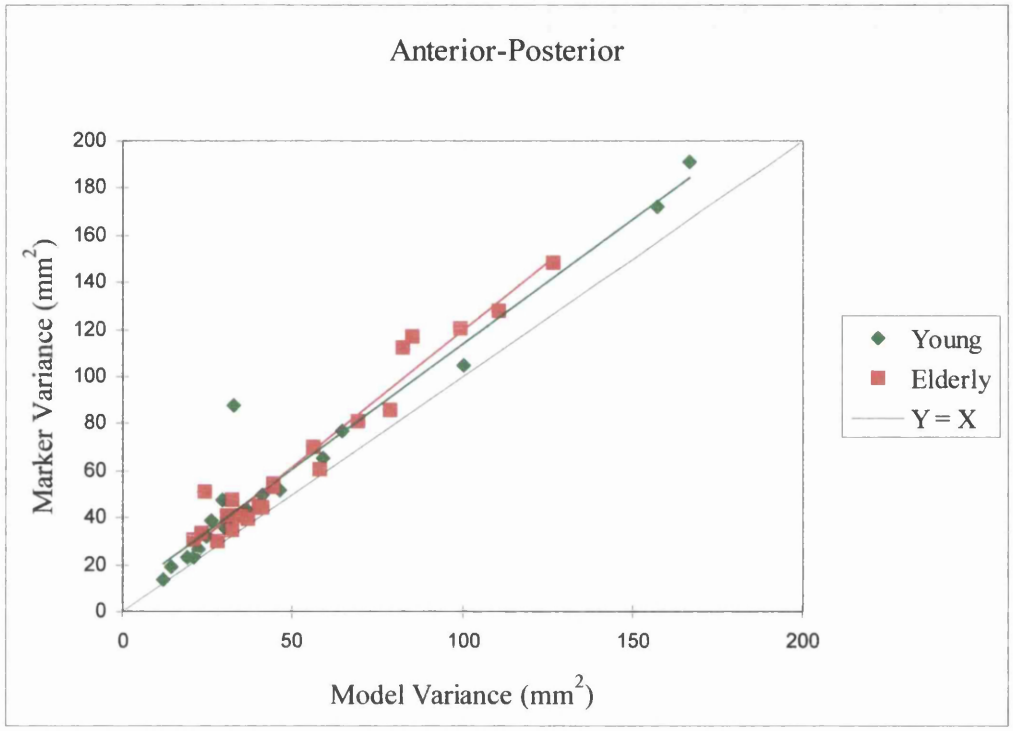

**Figure 3.1.16**: Medial-Lateral axis. **A)** Individual moment of inertia estimates $I_1$ and $I_2$ calculated from the regression coefficients plotted against 'I' calculated using anthropometric parameters (double log axes). B) Geometric mean of $I_1$ and $I_2$ plotted against 'I'. For the young subjects, N=20 and for the elderly, N=21.

Considering that the body's moment of inertia calculated using anthropometric parameters is in itself only an approximation for the body's true moment of inertia there appears to be reasonable agreement between the two estimates of 'I'. In the M/L axis the estimate for the body's moment of inertia calculated from the slope of the regression line is always larger than that calculated using anthropometric parameters. From Figure 3.1.16a it is apparent that the values estimated for $I_1$ are very close to those estimated from the anthropometric data suggesting that comparatively little of the error in 'I' is from the residual torque record. In contrast the errors in '$\alpha$' are relatively large, probably due to the differentiation process used to obtain the record of angular acceleration. In the M/L axis the movement of the body's COG is quite small and therefore the percentage of noise arising from the differentiation of the angular position of the model will be proportionally large. The difference in the estimates for 'I' in the M/L axes may also be due in part to the different control mechanisms in operation in the two axes. In the A/P axis, synchronous activity in the left and right ankle plantar-flexors and dorsi-flexors generate the residual torque recorded by the force plate in this direction. In the M/L axis the relationship between body mass parameters and body sway are more complex due to the two points of support.

*Does the inverted- pendulum model explain all of the marker movement?*

To investigate how much of the movement of the 14 markers from their mean position was explained by the angular position of the inverted pendulum, the variance of the records was calculated. For each marker, at each instant, and on each axis there is an actual movement from position ($q$) and a movement predicted by the model ($m$). The variance of '$q$' and '$m$' is calculated for each marker and the results combined to give the marker and model variance for the set of 14 markers. For each subject, the marker movement variance was plotted against the inverted pendulum model variance. (Figure 3.1.17a and b). Summaries of the linear regressions calculated are given in Table 3.1.8 including the regression coefficient and the $R^2$ values.

a)




b)

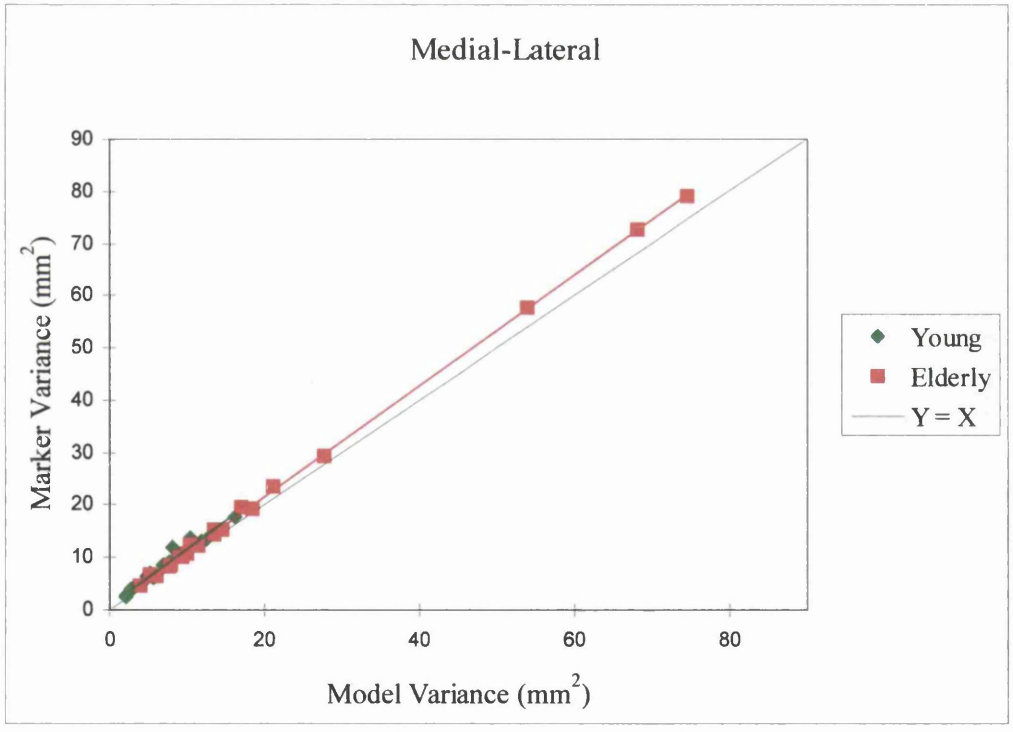



**Figure 3.1.17:** Proportion of marker movement variance explained by the inverted pendulum model in the A) Anterior-Posterior and B) Medial-Lateral axes. Each point represents the mean variance obtained for an individual from 3 trials. Also shown are the linear regression lines calculated for the young (N = 20) and elderly (N = 22). The regression coefficients and $R^2$ values calculated for the lines are presented in Table 3.1.8.

**Table 3.1.8:** Regression coefficients and $R^2$ values obtained for the regression of marker movement variance on model variance for the young and elderly subject groups.

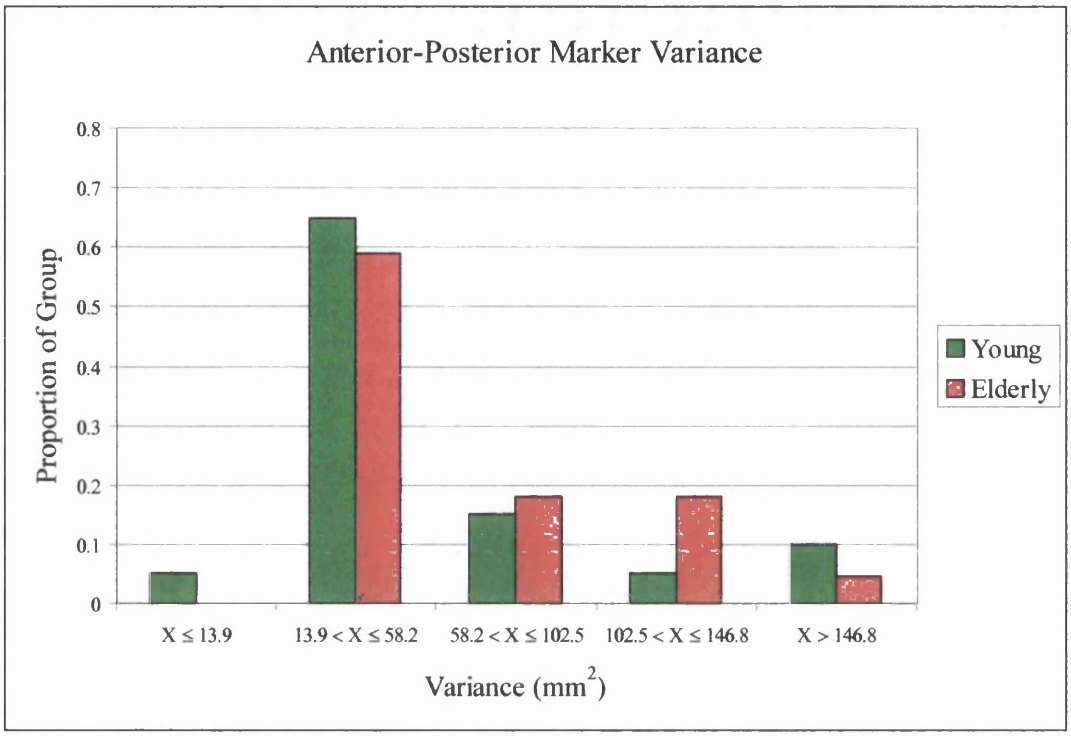
| AXIS | YOUNG (N = 20) | | ELDERLY (N = 22) | |
|---|---|---|---|---|
| | Slope | $R^2$ | Slope | $R^2$ |
| A/P | 1.063# | 0.940# | 1.156 | 0.955 |
| M/L | 1.067 | 0.972 | 1.056 | 0.999 |

# In the A/P axis the model variance calculated for one young subject was much less than the marker variance and the results for the slope and $R^2$ for the young group were somewhat influenced by this subject's result. The greater variance calculated for the markers in this subject were probably due to incorrect placement of the pelvis markers on the abdomen rather than the anterior superior iliac spine. In this situation respiration may have greatly increased the movement variance of the pelvis markers and movement of the inverted pendulum model could not explain these respiratory movements of the pelvis markers. If this individuals results were excluded the slope of the regression line for the young subjects becomes 1.085 with an $R^2$ value of 0.991, (N = 19).

The calculated regression coefficients indicate that most of the marker movement variance can be explained by the angular position of the inverted-pendulum model for both young and elderly subject groups and in both the A/P and M/L axes.

A Mann-Whitney Rank Sum Test was used to compare the median values for marker and model variance obtained for the two groups in the A/P and M/L axes. In the A/P axis, there was no statistically significant difference between the young and elderly groups for marker variance ($p$ = 0.186, N(young) = 20, N(elderly) = 22) or model variance ($p$ = 0.155). In the M/L axis, the median marker variance of the elderly group was significantly greater ($p$ = 0.016) than that of the young group. Similarly, the M/L model variance was significantly greater for the elderly group ($p$ = 0.006). Histograms illustrating the frequency distribution of the marker and model variance for the two groups are presented in Figures 3.1.18a and b.

a)




b)

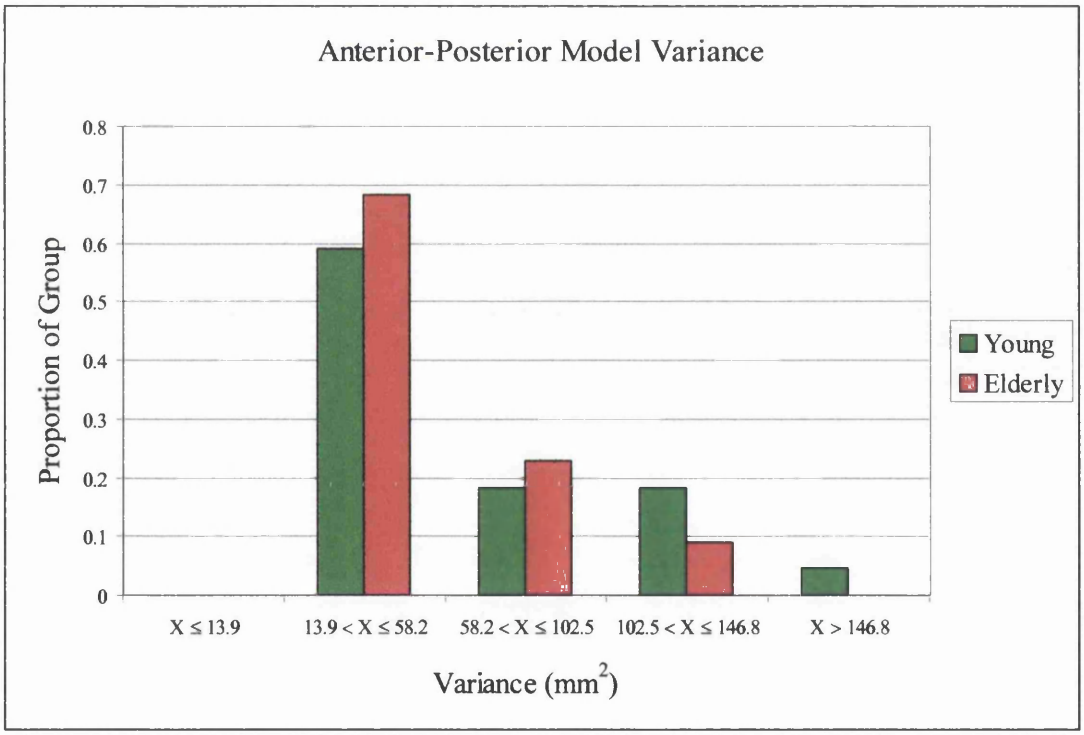
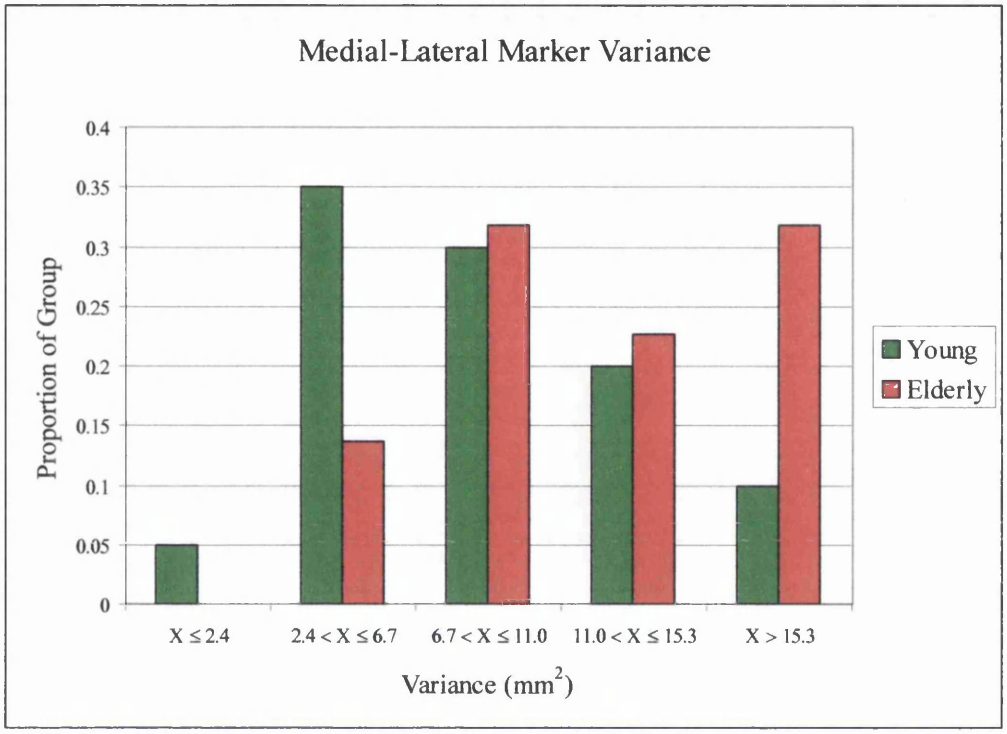



**Figure 3.1.18:** Histogram of anterior-posterior a) marker variance and b) model variance. Area of bar is equal to the proportion of the group whose variance values fall within the limits of the bin, N(Young) = 20, N(Elderly) = 22.

a)




b)

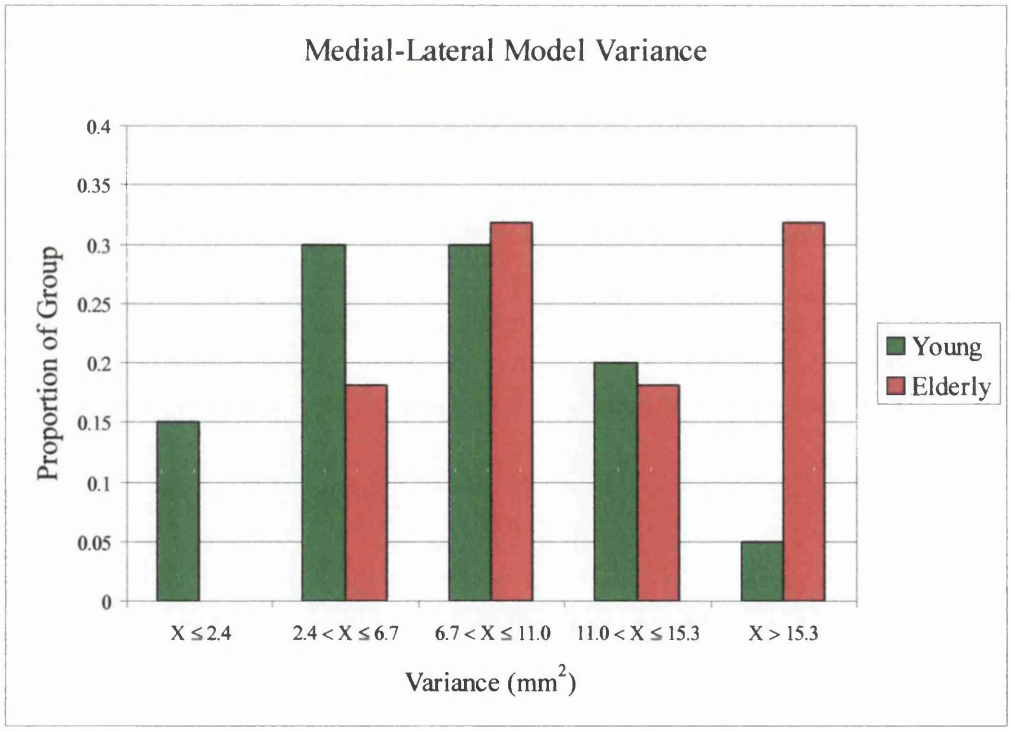



**Figure 3.1.19:** Histogram of medial-lateral a) marker and b) model variance. Area of bar is equal to the proportion of the group whose variance values fall within the limits of the bin. N(Young) = 20, N(Elderly) = 22.

In the A/P axis (Figure 3.1.18) both the young and elderly groups show a positively skewed frequency distribution with similar proportion of the group in each bin. In the M/L axis (Figure 3.1.19) the histograms clearly show why a significant difference was observed between the two groups for the M/L marker and model variance (Mann-Whitney Rank Sum Test). A greater proportion (40%) of the young group is concentrated in the 2 lowest bins compared to only 14% of the elderly group. In addition, the frequency distribution of the M/L marker variance for the elderly appeared to be bimodal suggesting that the elderly group may contain two separate populations. The histogram of the M/L model variance also shows clear differences between the distributions of the two groups. The frequency distribution of the young group is positively skewed with a large proportion (45%) of the variance values contained in the lowest 2 bins while the distribution of the elderly group is negatively skewed with 68% of the values in the 2 highest bins.

Subgroup analysis

The variance of the marker movement record is a measure of how far the respective body segments shift from their mean position. Similarly the variance of the model record reflects the excursions of the body's COG. If the excursions of the COG are sufficiently large, a subject's balance may become compromised and more demands will be placed on the postural control system. Some of the elderly subjects had obvious balance impairment as judged by their performance on the tandem gait, one legged stance and foam eyes closed tests. The marker and model variance calculated for the subgroup of 10 'balance impaired' (BI) elderly subjects was compared to that of a subgroup of 6 elderly subjects with good balance abilities, (GBA), and no known medical conditions which could affect their balance. The BI and GBA elderly subgroups were also compared to a group of 6 young subjects matched as closely as possible for sex, weight and height to the GBA elderly. The data underwent a logarithmic transformation prior to testing for significant differences between

the means of three groups using a one way ANOVA. When a significant difference was found a Tukey test was used to make pair-wise multiple comparisons between the groups. The results are summarised in Table 3.1.9.

**Table 3.1.9:** Results from one way ANOVA tests comparing the transformed marker and model variance in the A/P and M/L axes. Young adults 'Y' (N = 6), elderly with good balance ability 'GBA' (N = 6), 'balance impaired' elderly 'BI' (N = 10).

| VARIANCE | Y - GBA | Y - BI | GBA - BI |
|---|---|---|---|
| A/P, marker | NS | NS | NS |
| A/P, model | NS | NS | NS |
| M/L, marker | NS | * | * |
| M/L, model | NS | * | NS |

* Statistically significant differences, $P<0.05$. NS = no statistically significant difference.

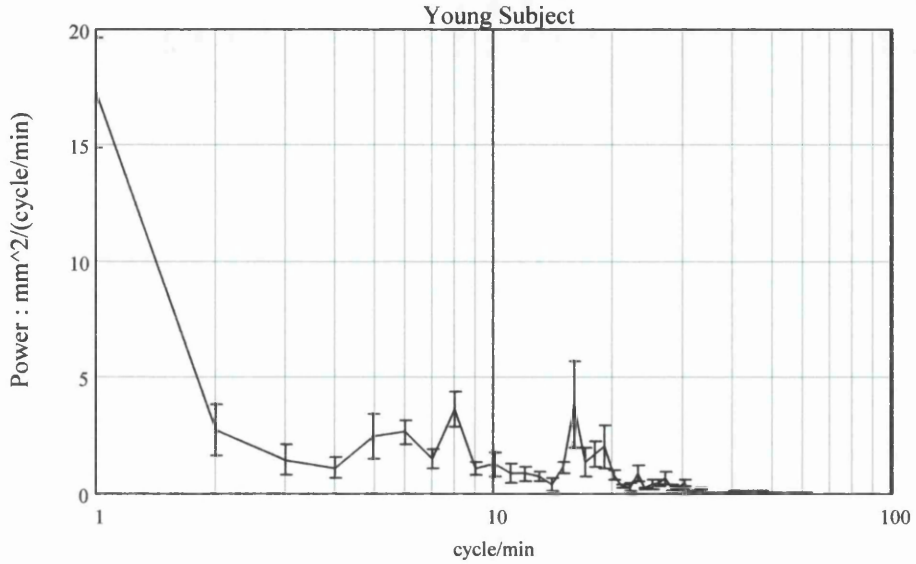
An inability to perform balance tests such as a one-legged stance or tandem gait suggests the subject has difficulty with medial-lateral postural control and this is borne out by the results of the one way ANOVA and pair-wise comparisons. In the A/P plane the marker and model variance was not significantly different between the three groups. However, in the M/L axis, the marker variance of the balance-impaired elderly was significantly greater than that of the young subjects and the elderly subjects with good balance. The M/L model variance of the BI elderly subjects was also significantly greater than that of the young subjects.

*Results of FFT analysis*

Power spectral analysis of the COP movement for the young and elderly subjects indicated that the principal power was contained below 1 Hz for both A/P and M/L axes with peak power occurring below 0.1 Hz. The energy was distributed across all frequencies up to approximately 0.5 Hz and there was rarely a sign of a prominent or consistent peak in the spectrum. Figures 3.1.20 and 3.1.21 represent the spectral signatures (mean and S.E.M of the three individual trials) for a young subject and an elderly subject in the A/P and M/L axes respectively. The individual subjects spectral signatures were averaged together with equal weighting to obtain a representative spectral envelope for the young and elderly groups, in the A/P and M/L axes (Figures 3.1.22a and b). The spectral envelopes were similar for the young and elderly groups in the A/P plane but in the M/L axis elderly subjects exhibited greater power at frequencies less than 0.25 Hz.

The amplitude of the COP movement in low frequency (0.0167 - 0.251 Hz) and high frequency (1.01 - 4.99 Hz) bandwidths was calculated for each subject's spectral signature in the A/P and M/L axes. A Mann-Whitney Rank Sum test was used to compare the median values of the young and elderly groups. In the A/P axis there was no significant difference in the amplitude of the COP movement between the groups in either the low ($p = 0.606$) or high ($p = 0.054$) frequency bandwidths. In the M/L axis the amplitude was significantly greater in the elderly group for the low frequency bandwidth ($p = 0.016$) but there was no difference between the two groups in the high frequency bandwidth ($p = 0.320$).

a)




b)

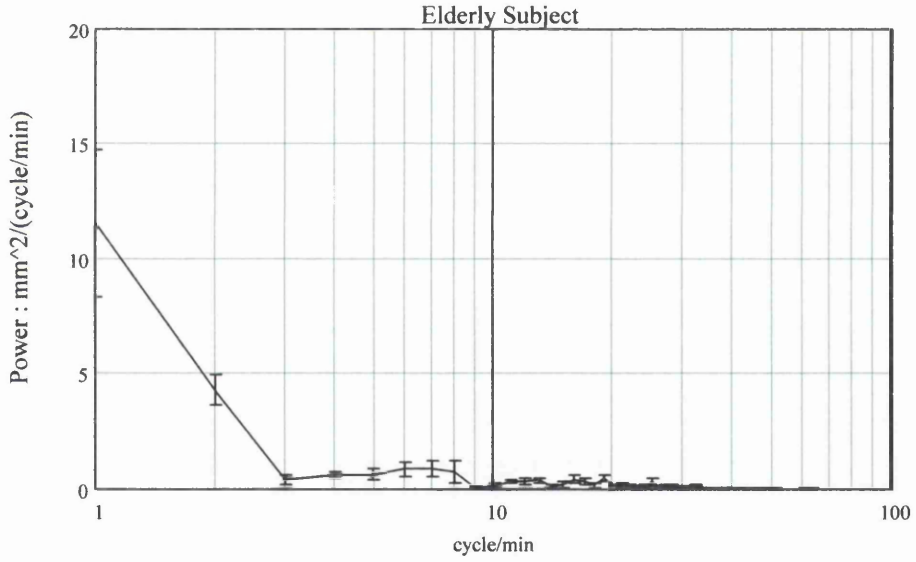
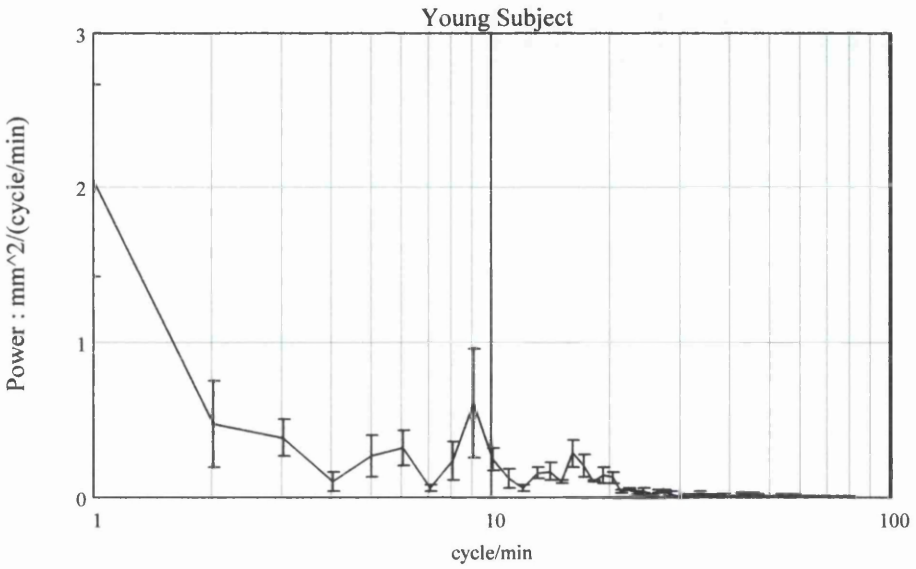



**Figure 3.1.20:** Power spectral analysis of COP movement in the A/P axis. Individual spectral signatures of a young subject (a) and an elderly subject (b). Results are the mean ± S.E.M. of three trials. 1 Hz = 60 cycles/minute.

a)




b)

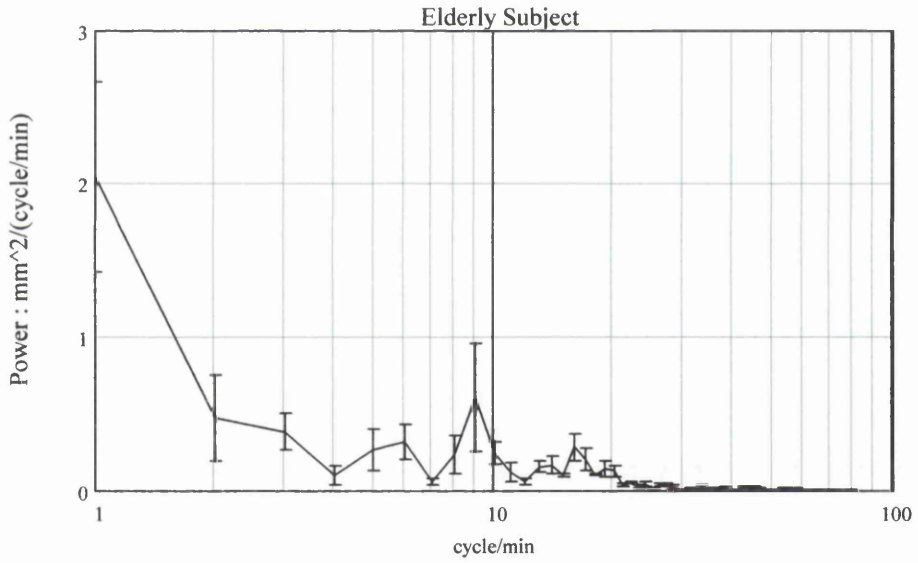
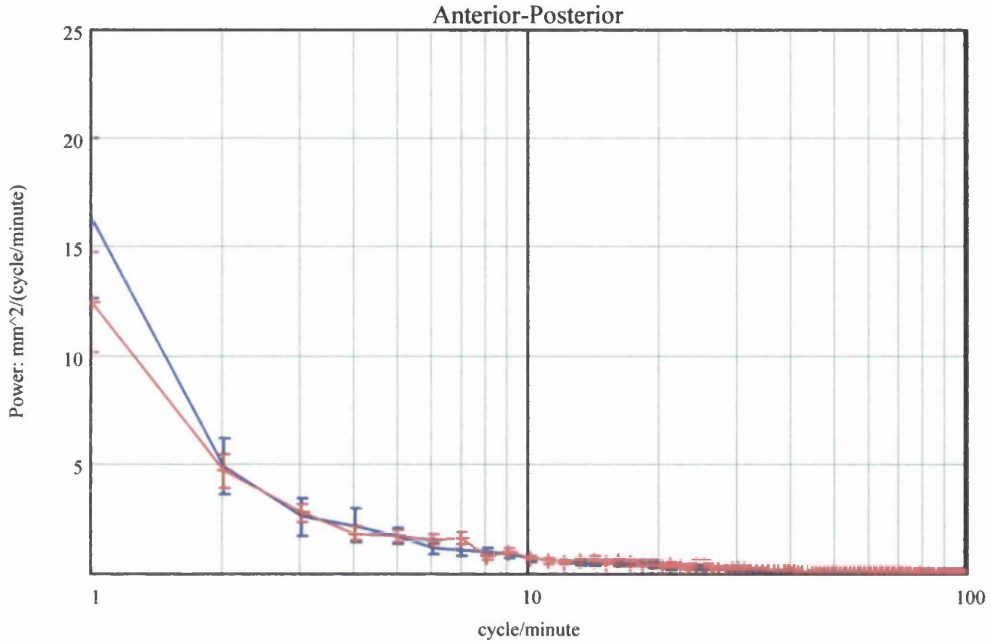



**Figure 3.1.21:** Power spectral analysis of COP movement in the M/L axis. Individual spectral signatures of a young subject, (a) and an elderly subject (b). Results are the mean ± S.E.M. of three trials. 1 Hz = 60 cycles/minute.

a)




b)

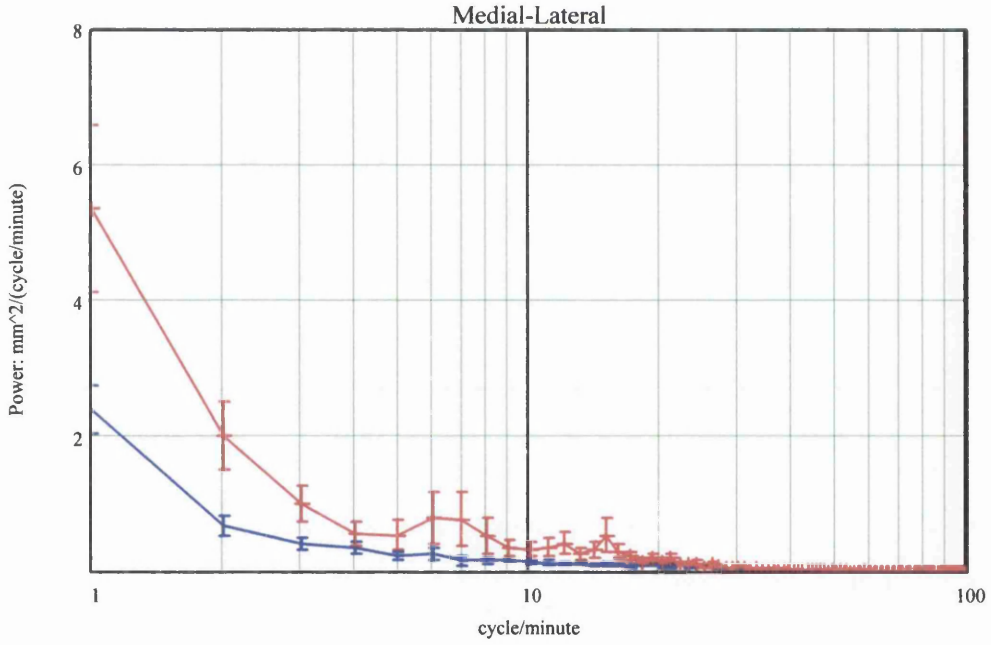


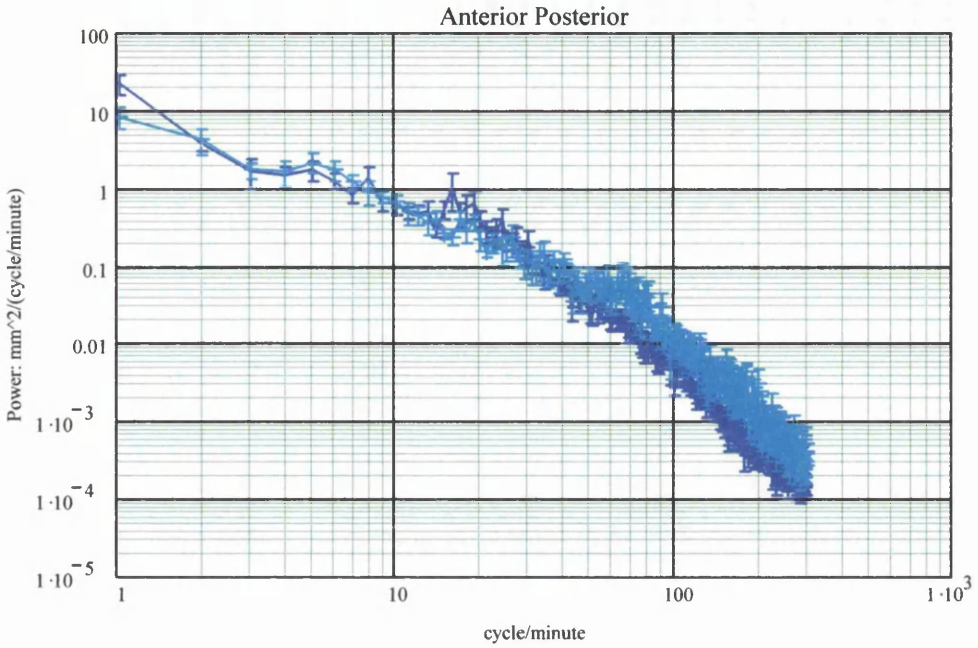

**Figure 3.1.22:** Spectral envelopes of COP movement generated by averaging individual spectral signatures. Results are the mean ± S.E.M. for the young group (N = 20, Blue traces), and the elderly group (N = 22, Red traces).1 Hz = 60 cycles/minute.

*Subgroup analysis*

A subgroup analysis of the spectral signatures was conducted using the same subgroups identified in the previous section. Spectral envelopes generated for each subgroup were plotted on double-log axes. Figures 3.1.23a and b represent the spectral signatures of the young and 'balance impaired' elderly subgroups. The 'balance impaired' elderly showed a trend towards greater power at low frequencies (less than 0.25 Hz) in the M/L axis similar to that observed in the spectra generated for the elderly group as a whole (Figure 3.22b). The balance-impaired elderly also showed a trend towards increased power at frequencies above 1.0 Hz in both the A/P and M/L axes compared to the young subjects. Figures 3.1.24a and b represent the results for the 'BI' elderly and 'GBA' elderly. The results for the 'GBA' elderly are very similar to those of the young subgroup in that they show less power at low frequencies in the M/L axis and less power above 1.0 Hz than the 'BI' elderly. The close match of the spectral envelopes for the GBA elderly and young subgroups (Figures 3.1.25a and b) also illustrates the similarity between the results of the two subgroups.

The Mean Spectral Frequencies (MSF) of the A/P and M/L COP movement was calculated for each subject trial and a resultant value obtained for each subject. The mean values for the three subgroups were compared using a one-way ANOVA and the results showed there was no significant difference between the groups in the A/P axis ($F = 0.857$, $p = 0.444$) or M/L axis ($F = 0.873$, $p = 0.438$).

a)




b)

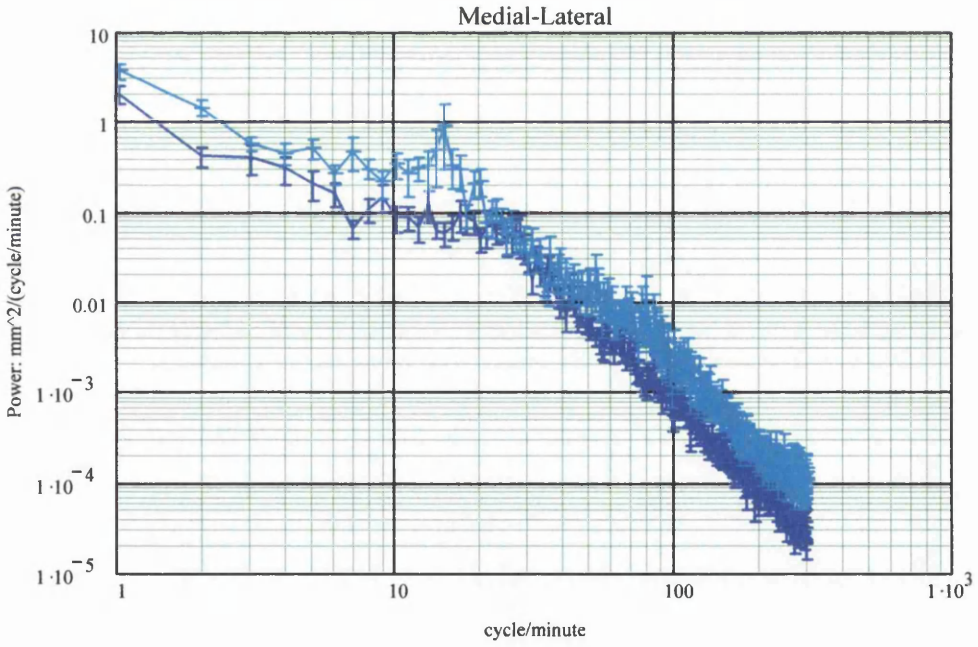
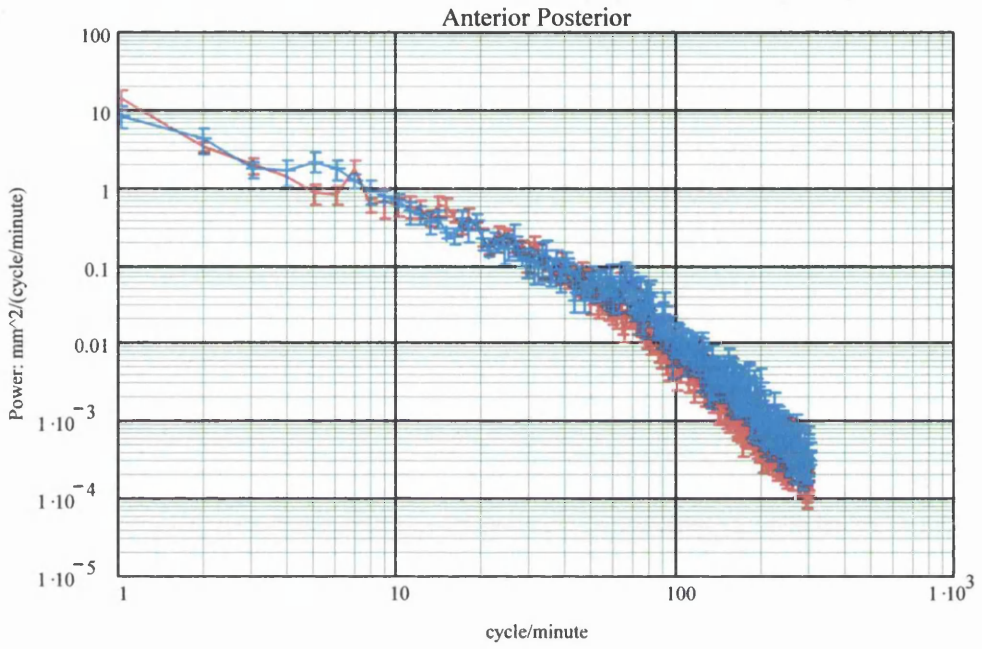



**Figure 3.1.23:** Spectral envelopes of COP movement generated by averaging individual spectral signatures. Results are the mean ± S.E.M. for the young (N = 6, blue traces) and 'balance impaired' elderly subgroups (N = 10, cyan traces). Data plotted on log-log scale to highlight the presence of some low-power, high frequency sway between 1 and 5 Hz in the elderly subgroup. 1 Hz = 60 cycles/minute.

a)




b)

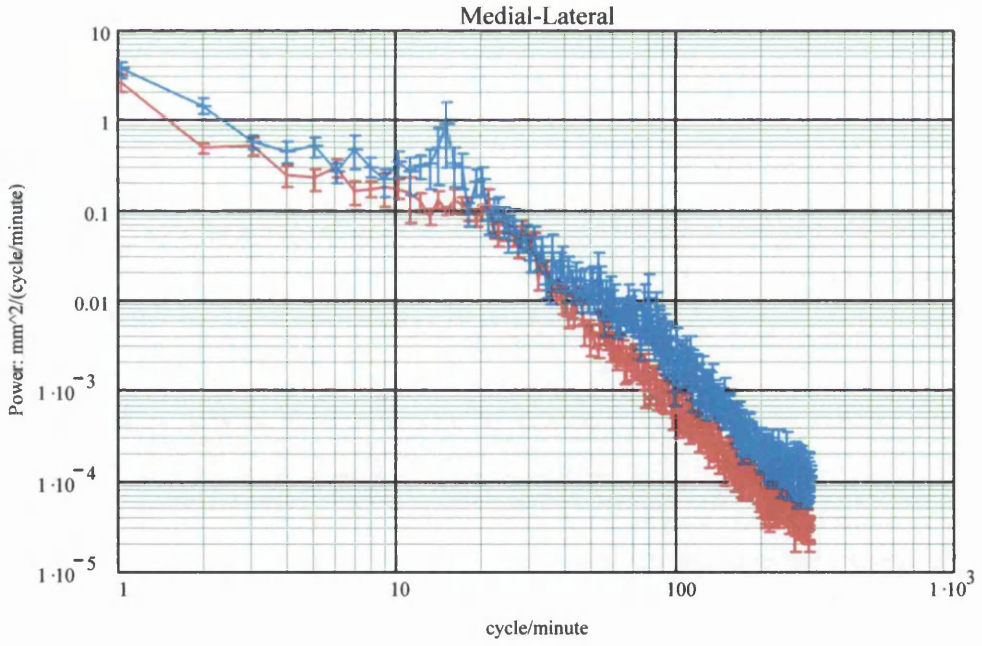
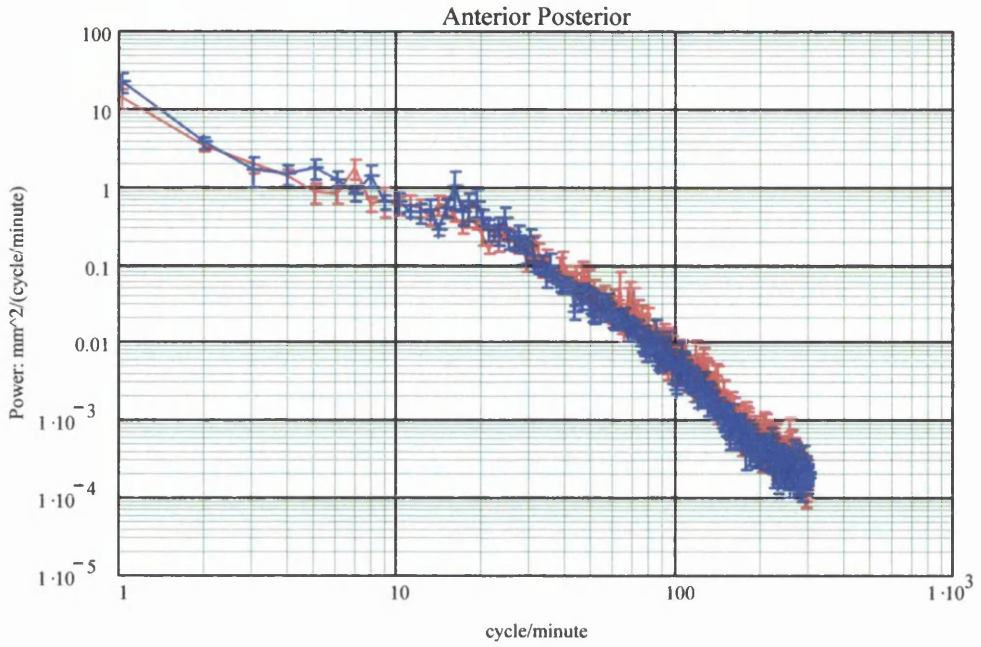



**Figure 3.1.24:** Spectral envelopes of COP movement generated by averaging individual spectral signatures. Results are the mean ± S.E.M. for the 'GBA' elderly (N = 6, red traces) and 'BI' elderly subgroups (N = 10, cyan traces). Data plotted on log-log scale to highlight the presence of some low-power, high frequency sway between 1 and 5 Hz in the BI elderly subgroup. 1 Hz = 60 cycles/minute.

a)




b)

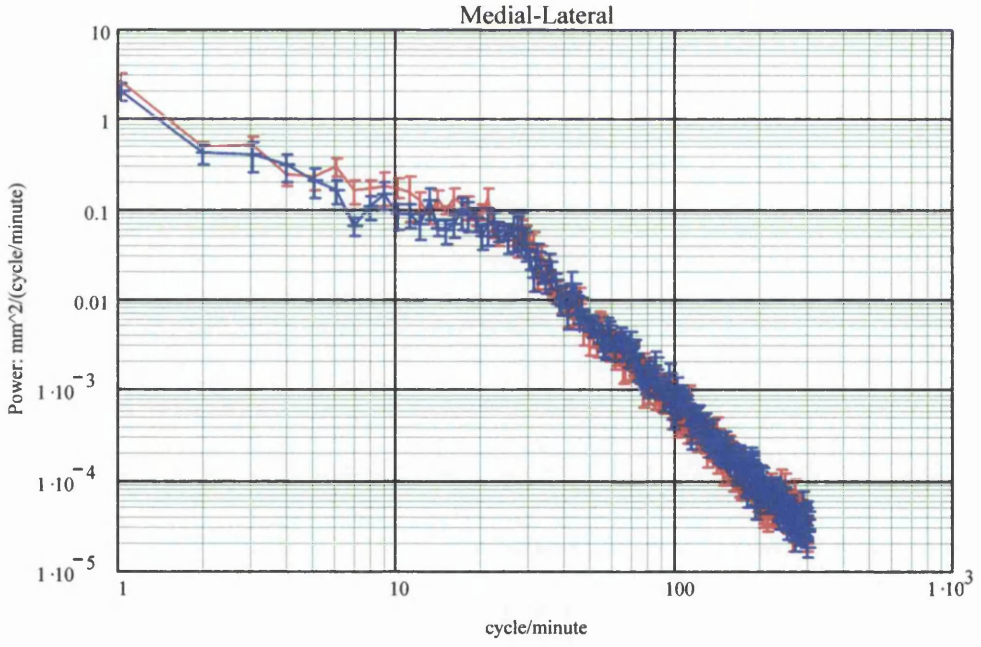



**Figure 3.1.25:** Spectral envelopes of COP movement generated by averaging individual spectral signatures. Results are the mean ± S.E.M. for the young (N = 6, blue traces) and 'GBA' elderly (N = 6, red traces). 1 Hz = 60 cycles/minute.

The amplitude of the COP movement in low frequency (0.0167 - 0.251 Hz) and high frequency (1.01 - 4.99 Hz) bandwidths was calculated for each subject's spectral signature in the A/P and M/L axes. One way analysis of variance was used to compare the means of the three groups. A pair-wise multiple comparison procedure (Tukey Test) was used to isolate group(s) that differed. A summary of the results is presented in Table 3.1.10.

**Table 3.1.10:** Results from one way ANOVA tests comparing the amplitude of the COP movement in the low (0.0167 - 0.251 Hz) and high frequency (1.01 - 4.99 Hz) bandwidths. Young adults 'Y' (N = 6), elderly with good balance ability 'GBA' (N = 6), 'balance impaired' elderly 'BI' (N = 10). * Statistically significant differences P<0.05. NS = no statistically significant difference.

| AMPLITUDE | Y - GBA | Y - BI | GBA - BI |
|---|---|---|---|
| A/P, low frequency | NS | NS | NS |
| A/P, high frequency | NS | NS | NS |
| M/L, low frequency | NS | * | NS |
| M/L, high frequency | NS | NS | NS |

In the A/P axis there was no significant difference between the three groups for the mean amplitude in the low ($F = 0.250$, $p = 0.781$) or high frequency ($F = 2.133$, $p = 0.146$) bandwidths. In the M/L axis, there was a significant difference in the mean amplitude between the BI elderly and the young subjects in the low frequency bandwidth ($p < 0.05$) but no significant difference was observed between the BI and GBA elderly or the GBA elderly and the young subjects. In the high frequency bandwidth there was a significant difference in the mean values among the subgroups ($F = 3.790$, $p = 0.041$) but the pair-wise multiple comparison of the groups (Tukey Test) failed to identify any group that differed significantly from another.

Case Studies

The spectral signatures of the two elderly subjects classified as having severe balance impairment were compared graphically to the spectral envelope of the 'GBA' elderly subgroup. (Figure 3.1.26a and b).

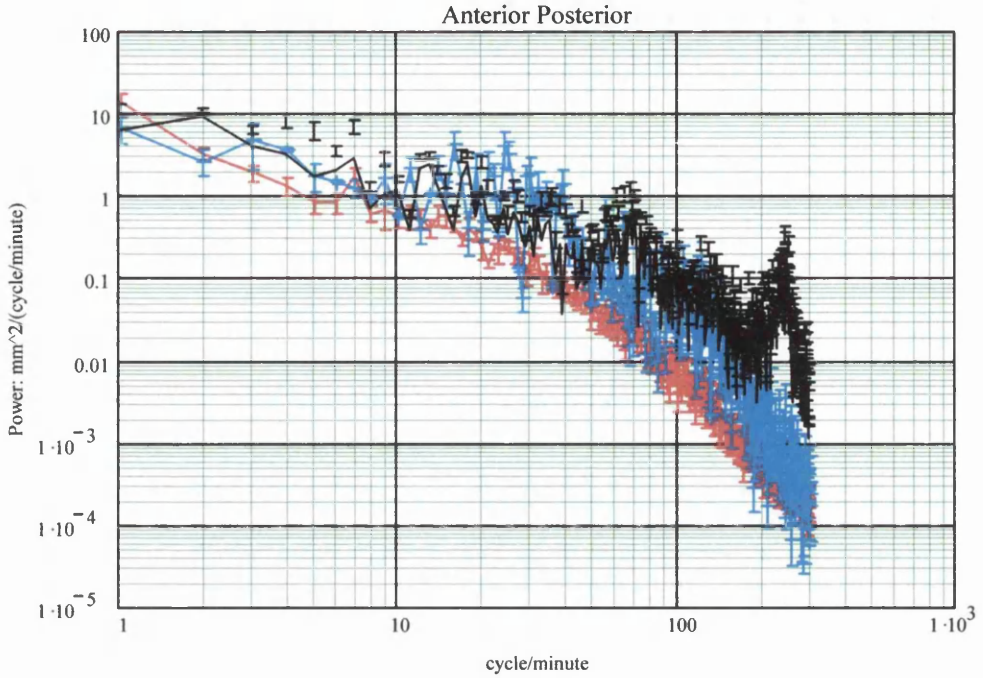
*Case 1*

Mr G. (blue trace) was an elderly male aged 74 years diagnosed as having tachycardia for which Amiodarone had been prescribed. The subject also complained of weakness in the left leg and tremor in the right hand. Side effects of Amiodarone include peripheral neuropathy and myopathy, hypo- and thyroid dysfunction, tremor, fatique, and ataxia. In the A/P axis, the subject's spectral signature exhibited some low power, high frequency components around 1 Hz. Similar high frequency instability around 1 Hz has been observed in the power spectrum of subjects with peripheral neuropathies (Mauritz *et al.* 1980). There was also an accentuation of power at approximately 3 Hz. Mauritz *et al* (1979) suggested that a 3 Hz A/P postural tremor is characteristic of cerebellar anterior lobe dysfunction and A/P instability is due to the anterior lobe control of proprioceptive postural reflexes operating in the sagittal plane. In the M/L axis there was greater power exhibited across all frequencies but particularly at low frequencies (less than 0.25 Hz) similar to that observed in the 'balance impaired' elderly subgroup.

*Case 2*

Mr W. (black trace) was an elderly male aged 68 years, moderately obese, with a BMI of 30.7%. He has a diagnosis of Type II diabetes an was taking an oral antidiabetic drug, (sulphonylurea) to augment insulin secretion. In the A/P axis the spectral signature of this subject was not very different from that of the 'healthy' elderly subgroup. However, in the M/L axis the spectral signature showed greater power at low frequencies (less than 0.25 Hz) as well as high frequencies (1.0 - 4.99 Hz).

a)




b)

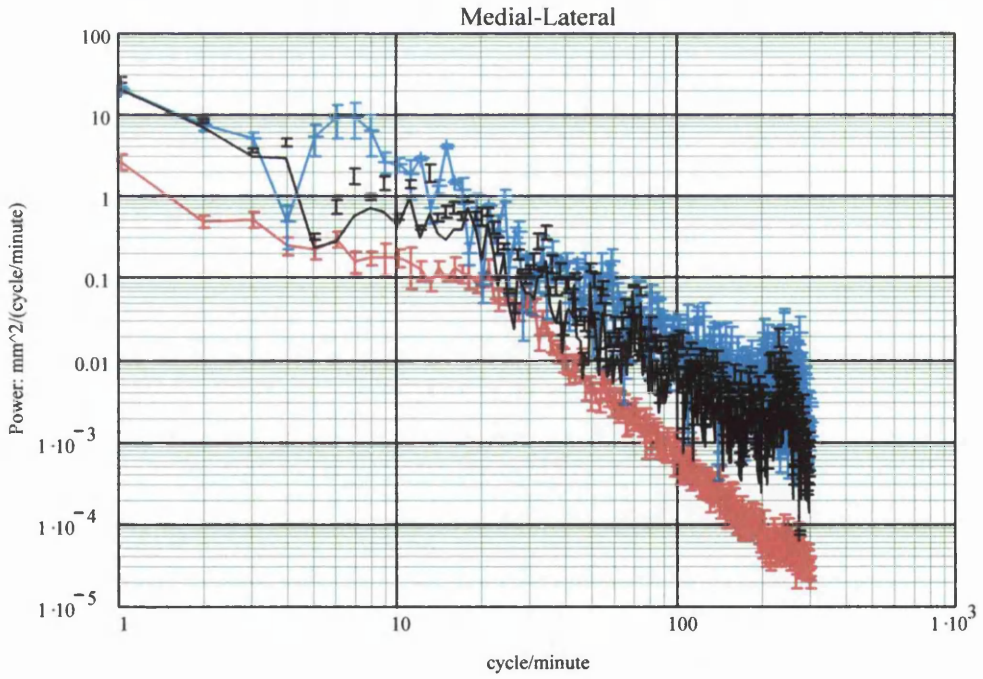



**Figure 3.1.26:** Comparison of the spectral signatures of two elderly subjects classified as having severe balance impairment with the spectral envelope of the GBA elderly subgroup (N = 6, red traces). Case 1 - black traces, Case 2 – cyan traces (mean ± S.E.M. of three trials). Data plotted on log-log scale to highlight the presence of some low-power, high frequency sway above 1 Hz. 1 Hz = 60 cycles/minute.

*Stabilogram-diffusion analysis*

*Stabilogram-diffusion* plots (Collins and DeLuca, 1995) were computed from the records of movement of COP, movement of COG, and the residual movement (COP - COG). See Figure 3.1.27 for an example. The values obtained for $D_{js}$ (short-term diffusion coefficient) of the COP, COG and residual plots for the young and elderly subject groups were normalised using a logarithmic transformation. A summary of the geometric mean (± 1 S.E.M.) for the two groups and the results of a *t*-test are comparing the two groups is presented in Table 3.1.11. The two elderly subjects with severe balance impairment were excluded from the analysis.

**Table 3.1.11** Summary of the geometric mean (± 1 S.E.M.) $D_j$ calculated for the COP, COG and residual records in the A/P and M/L planes for young (N = 20) and elderly (N = 20) groups. The result of two-tailed student's t-tests comparing the two groups is also shown.

| | GEOMETRIC MEAN (± 1 S.E.M.) | | *P* |
|---|---|---|---|
| $D_j$ | Young | Elderly | Young = Elderly |
| A/P COP | 22.28<br>(19.36, 25.66) | 25.94<br>(21.96, 30.65) | 0.493 |
| A/P COG | 11.09<br>(9.66, 12.73) | 9.95<br>(8.11, 12.22) | 0.659 |
| A/P Residual | 8.69<br>(7.29, 10.36) | 13.31<br>(11.00, 16.10) | 0.230 |
| M/L COP | 5.68<br>(4.76, 6.76) | 5.52<br>(4.40, 6.93) | 0.923 |
| M/L COG | 2.24<br>(1.87, 2.70) | 2.04<br>(1.51, 2.75) | 0.786 |
| M/L Residual | 2.19<br>(1.83, 2.62) | 1.90<br>(1.54, 2.34) | 0.601 |

The results of the *t*-test show that the mean value of $D_{js}$ calculated from the COP, COG and residual plots are not statistically different for the two groups in either the A/P or M/L axes. Examples of *stabilogram-diffusion* plots for individual subject trials are shown in Figures 3.1.27 and 3.1.28. Records of movement of the COP contain two components, a static component related to the movement of the COG and a dynamic component due to inertial forces as well as cardiac and respiratory effects. The relative proportions of each component in the COP record influences the $D_{js}$ of the COP *stabilogram-diffusion* plot. The value of $D_{js}$ calculated from the COP plot is always greater than that calculated from the COG plot due to the contribution from dynamic components. In contrast, the long-term slope of the COP plot more closely reflects the long-term slope of the COG plot. Often the COG *stabilogram-diffusion* plot is a fairly straight line with no distinct critical time point (Figure 3.1.28a) while the residual *stabilogram-diffusion* plot often has a steep short-term slope and a value for the long term slope close to zero (Figure 3.27b). If the influence of the dynamic components of stance on the COP record is minimal, than the slopes of the COP and COG records are very similar (Figures 3.1.27a and 3.1.28b). However, if there is a relatively large contribution from dynamic components than the slope of the COP *stabilogram-diffusion* plot is much greater than that of the COG (Figure 3.1.27b). Large values observed for the $D_j$ of the COP record in some elderly subjects are in most cases due to the high values obtained for the $D_j$ of the residual record, reflecting an increase in the dynamic, high frequency components of stance in these subjects. In contrast, large values observed for $D_j$ of the COP record in a few young subjects coincides with large values for $D_j$ of the COG record, suggesting an increase in the low frequency components of stance. In general, the dynamic components accounted for approximately half the short-term slope of the COP *stabilogram-diffusion* plot in the A/P and M/L axes (Table 3.1.11).

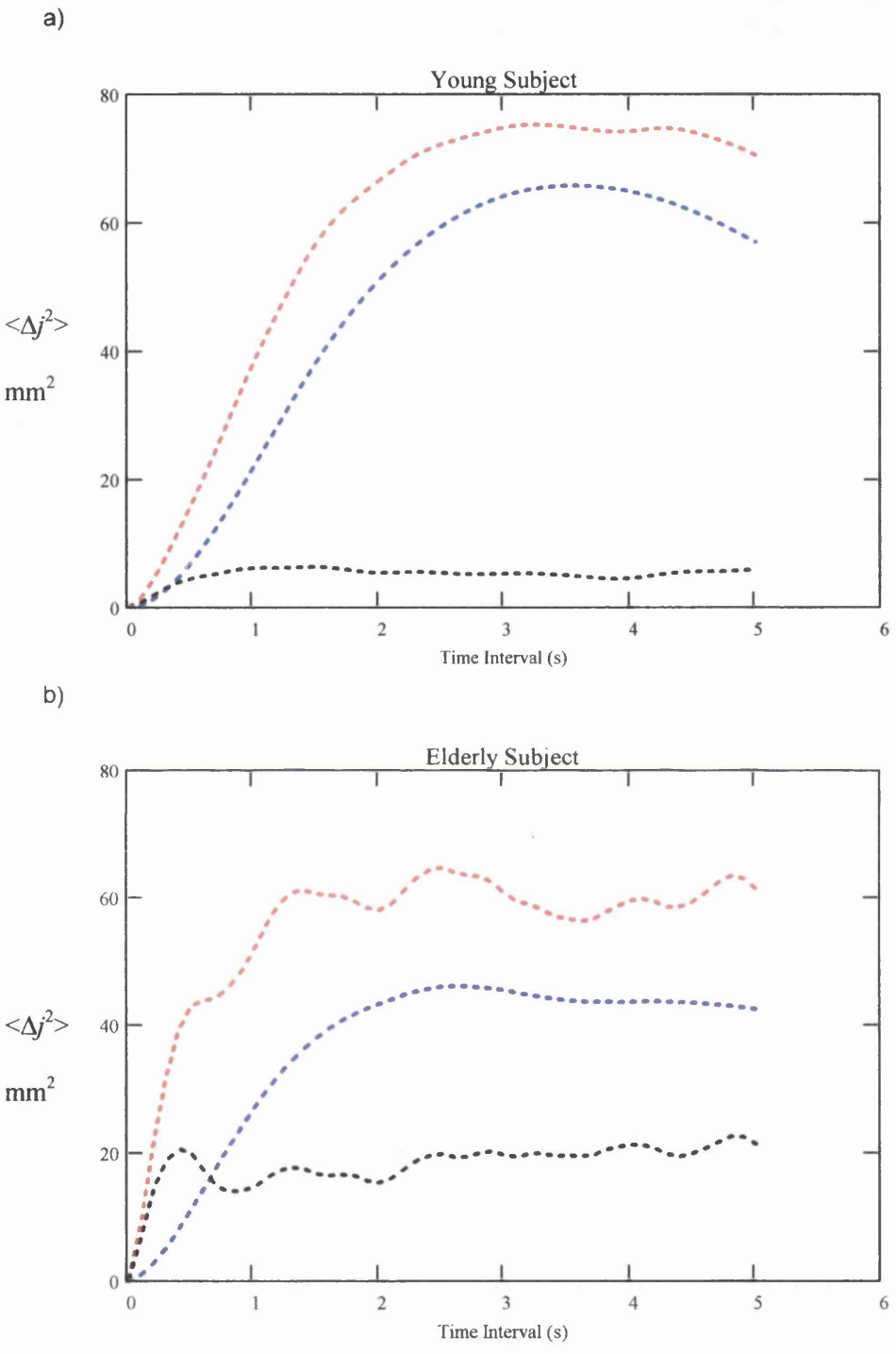



**Figure 3.1.27:** *Stabilogram-diffusion* plots of movement in the A/P axis of the COP (red trace), the COG (blue trace) and the residual movement (COP – COG, black trace). The data plotted are the results obtained during a single trial for a young subject (a) and an elderly subject (b). See text for further details.

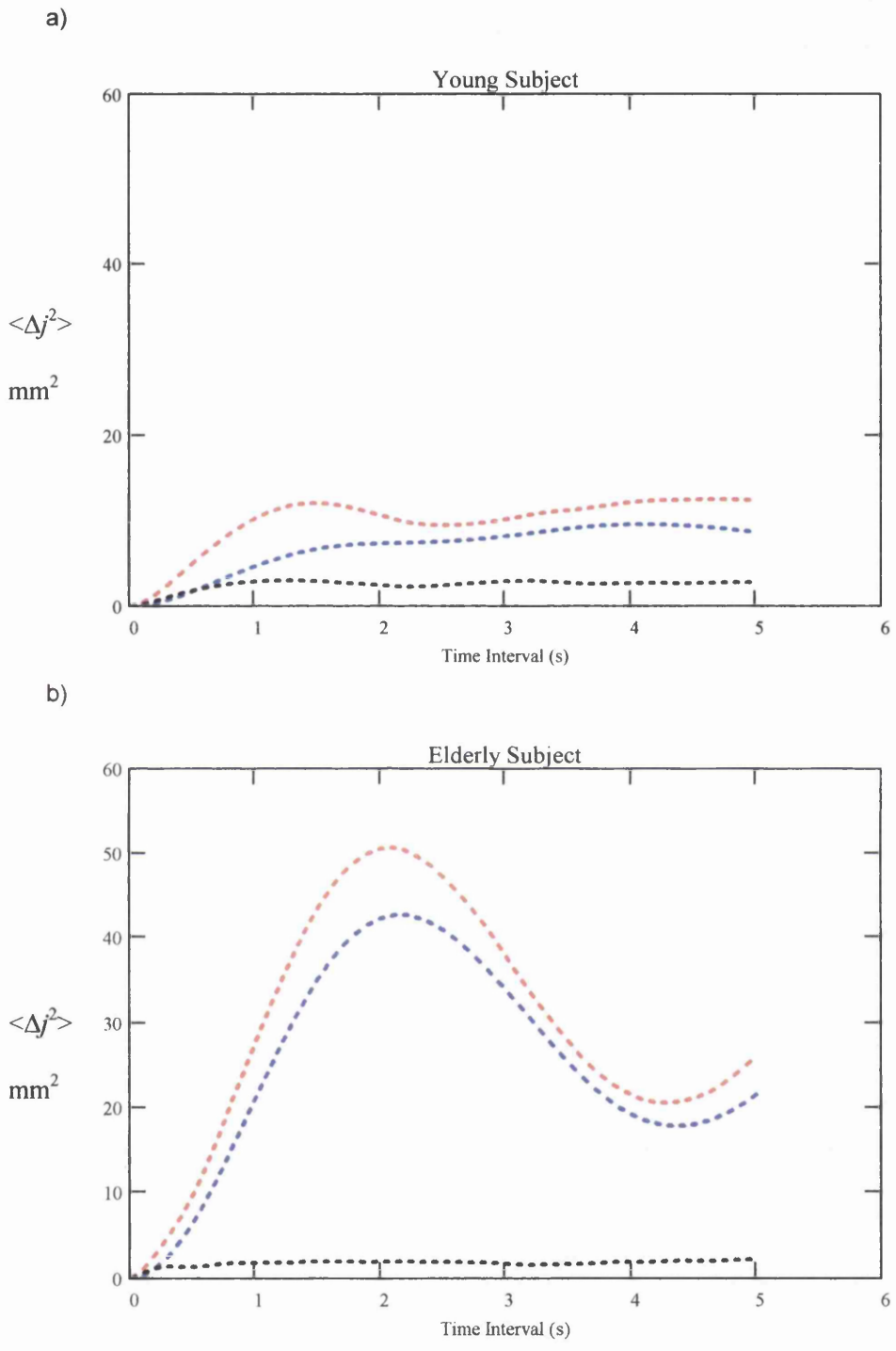



**Figure 3.1.28:** *Stabilogram-diffusion* plots of movement in the M/L axis of the COP (red trace), the COG (blue trace) and the residual movement (COP – COG, black trace). The data plotted are the results obtained during a single trial for a young subject (a) and an elderly subject (b). See text for further details.

## 3.2 *Postural stability during walking*

*Subjects*

The 20 young subjects and 22 elderly subjects that participated in the previous experiment also participated in this related experiment.

*Deviations from the mean path during walking*

Typically 15 strides were selected to create two data sets in which the A/P, M/L and vertical positions of the markers at each point during the stride were referenced to the ankle of the supporting leg i.e. one data set was referenced to the left ankle and the other to the right ankle. This took account of the forward progression across the room in the 'A/P' axis and any general sideways deviations from a straight path in the 'M/L' axis. The marker data of the selected strides was interpolated so that individual stride times were converted to 100% of a stride. The data was then averaged to obtain the 'mean stride path' described by the markers in the 'A/P' and 'M/L' axes for each subject. This 'mean stride path' corresponds to the 'mean position' described previously for quiet stance. A minimum of 10 strides was used to obtain the average. For 3 of the young and 1 elderly less than 10 strides could have been used from the combined record so these subjects were excluded from the analysis.

A record of 'marker movement from mean path' in the A/P and M/L axes was obtained for each marker by subtracting the 'mean stride path' of the marker from its total excursion during each of the strides selected for analysis. These records are the equivalent to the A/P and M/L movement records described previously for quiet stance. Figure 3.2.1a (A/P) and b (M/L) illustrate for a single marker, the actual movement of the marker relative to the supporting ankle during a single stride (aqua and dark blue traces), the mean stride path of the marker (pink and red trace) and the marker movement from mean path (black trace).

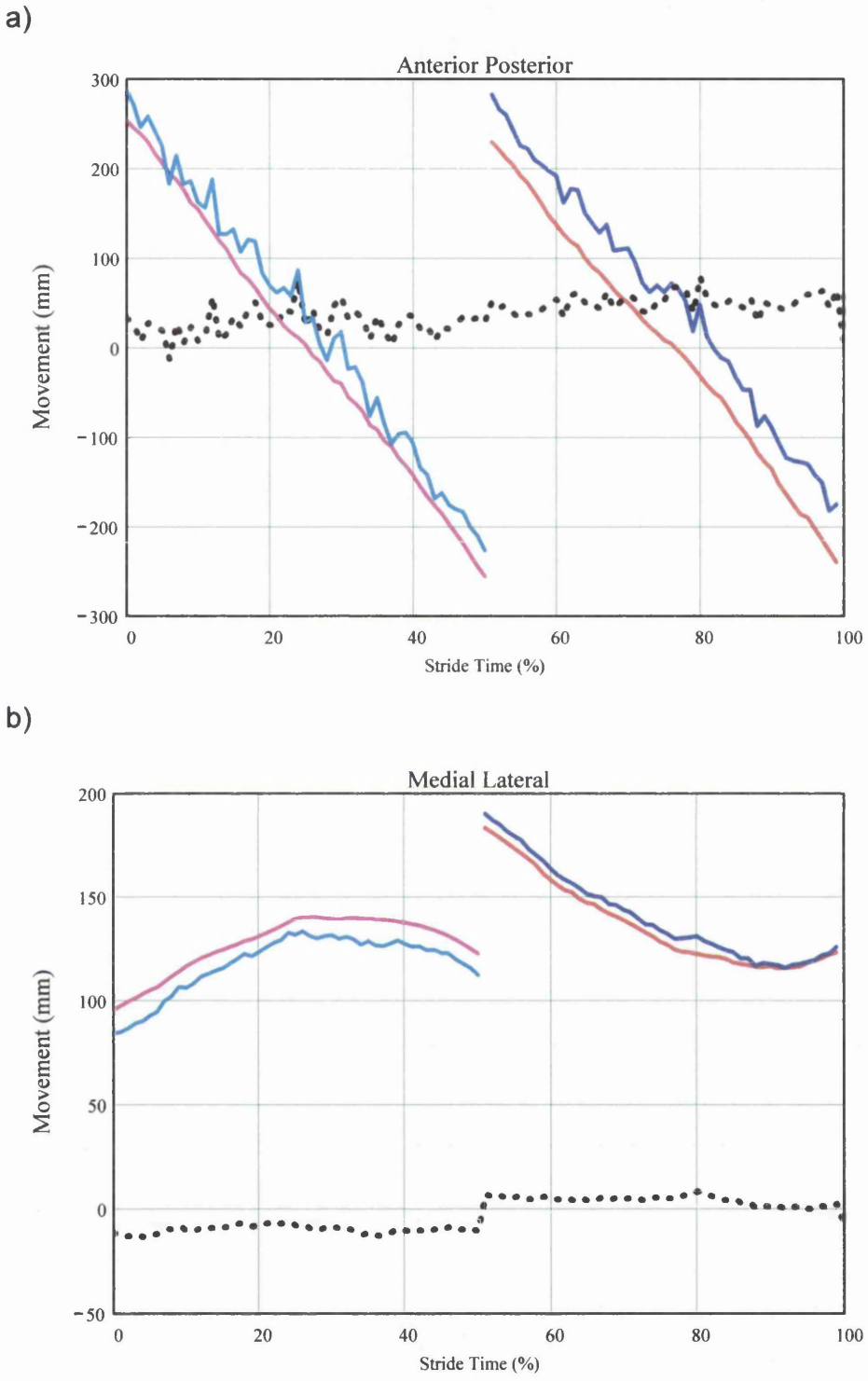



**Figure 3.2.1:** Actual movement of a single head marker, relative to the supporting ankle during a single stride (aqua and dark blue traces), the mean stride path of the marker (pink and red traces) and the movement of the marker from the mean path (black trace). A) Anterior-Posterior, B) Medial-Lateral.

Figure 3.2.2a and b illustrate the movement from mean stride path in the A/P and M/L axes for all the selected strides of a single shoulder marker. In the A/P axis, marker movement from the mean path is due mainly to variation in step length. In the M/L axis, abrupt shifts in marker position represent strides where the subject has placed the supporting foot to the left (or right) of the mean path and then overcompensated by placing the next supporting foot to the right (or left) of the mean path. On occasion, placement of the supporting foot to the left (or right) of the mean path may not be compensated for and the next supporting foot is also placed down to the left (or right) of the mean path for that foot. There is no regular pattern to the movements and they occur at a low frequency, i.e. the movements are not entrained by the step frequency.

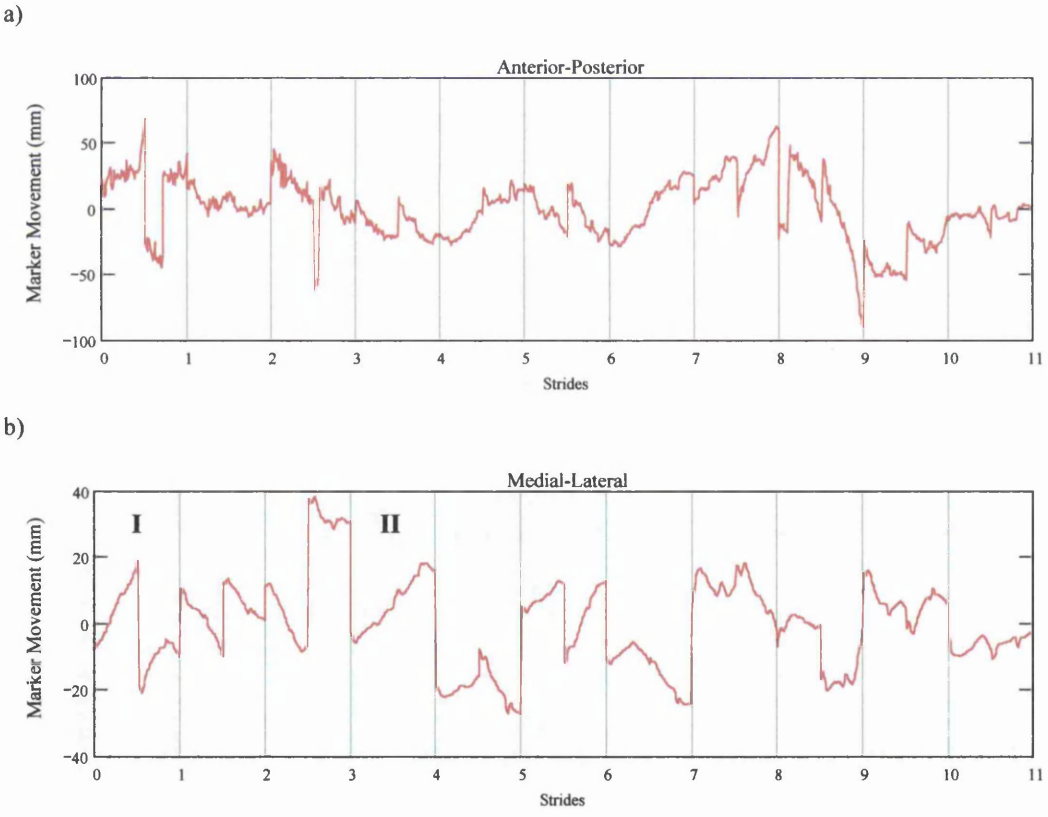



**Figure 3.2.2:** The movement from mean stride path of a single shoulder marker in the A/P (a) and M/L (b) axes. Each stride consists of two steps, one with each foot. In (I) the first supporting foot has been placed to the left of the mean path and then the subject has over compensated by placing the second supporting foot to the right of the mean path. In (II) there has been no attempt to compensate for the deviation from mean path and the second support foot has also been placed to the left of the mean path.

*Correlation between movements of the pelvis and shoulder markers*

During standing, the pelvis and shoulder markers showed a tendency to move together in the same direction. To determine if a similar tendency could be observed for the markers during walking, records of movement from mean path for the two pelvis markers were averaged together to obtain a mean pelvis movement record in the A/P and M/L axis. Mean movement records were also obtained for the shoulders and head. An example of the correlation between the mean pelvis and mean shoulder movement in the A/P and M/L axes for one subject is illustrated in Figure 3.2.3a and b respectively.

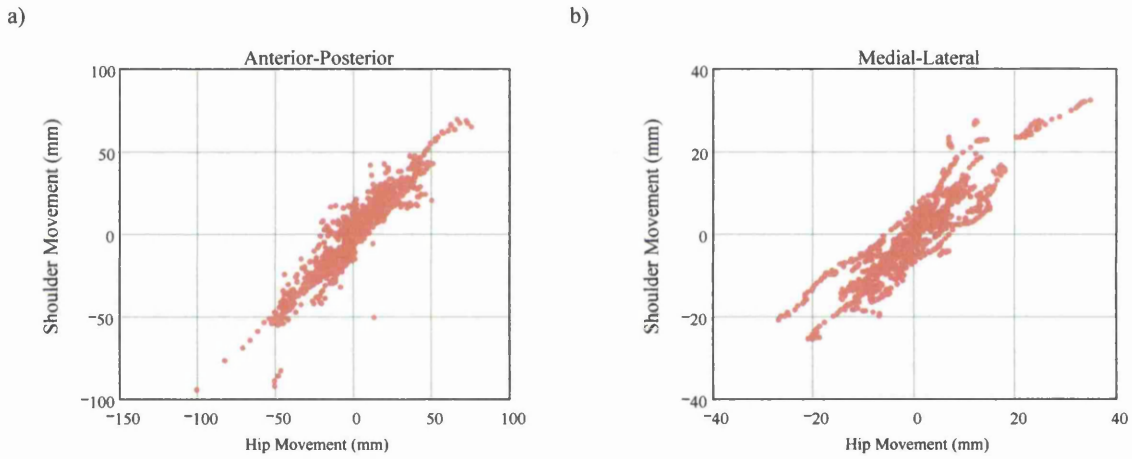



**Figure 3.2.3:** Correlation between pelvis and shoulder movement from mean position in the a) A/P and b) M/L axes for one subject. The correlation coefficient '*r*' is equal to 0.94 (A/P) and 0.91 (M/L) respectively.

The association between the deviation of the pelvis and shoulders (and pelvis and head) from their mean path in the A/P and M/L axes was measured by calculating the correlation coefficient, '*r*' for each subject. The individual subject values were transformed using the procedure outlined in the Methods (Chapter 2). A summary of the mean (± 1 S.E.M) '*r*' values for the young and elderly groups is presented in Table 3.2.1 along with the results of a two-tailed t-test used to detect for differences in the means of the two groups

**Table 3.2.1:** Summary of the correlation coefficients calculated for shoulder and head movement correlated with pelvis movement for the young (N=17) and elderly (N=21). Mean and 1 S.E.M. calculated from transformed data. Also shown are the results of two-tailed *t*-test (d.f. = 36) comparing the means of the two groups.

| AXIS | GEOMETRIC MEAN ( ± 1 S.E.M) | | *P* |
|---|---|---|---|
| | Young | Elderly | |
| Pelvis/Shoulder A/P | 0.89<br>(0.87,0.90) | 0.88<br>(0.87,0.90) | 0.899 |
| Pelvis/Shoulder M/L | 0.85<br>(0.83,0.87) | 0.86<br>(0.84,0.88) | 0.709 |
| Pelvis/Head A/P | 0.86<br>(0.83,0.88) | 0.83<br>(0.81,0.85) | 0.502 |
| Pelvis/Head M/L | 0.79<br>(0.77,0.81) | 0.77<br>(0.74,0.80) | 0.630 |

The results in Table 3.2.1 show that a large positive correlation exists between the movement of the pelvis and shoulders in the A/P and M/L axes in both subject groups. A large positive correlation was also observed for the movement of the pelvis and head markers in the A/P and M/L axes, although the correlation dropped off slightly in the M/L direction. There was no statistically significant difference between the means of the two groups for either the pelvis/shoulder correlation or the pelvis/head correlation and therefore the same type of movements are apparent in both groups

*Inverted-pendulum movements during walking*

The inverted pendulum model of the body during walking was based on the model described previously for quiet stance. Although the movement of all 14 markers were used in the linear regression which defined the inverted-pendulum model for stance, only the movement of the markers on the head, trunk and pelvis (total of 8 markers) relative to the supporting ankle were used in defining the model for walking. The movements of the swing leg and the two arms were not considered as part of the model because their movement from the mean path may be expected to have independent sources of variation. The slope of the linear regression line constrained though zero (the supporting ankle) is equal to:

$$b = \frac{\sum_{i=1}^{n} x_i \cdot y_i}{\sum_{i=1}^{n} (x_i)^2}$$

Where $x_i$ is the mean vertical height of a marker and $y_i$ is the movement of the marker from the mean path.

As in the model of quiet stance, the angle the inverted pendulum model of the body deviates from its mean position during walking is assumed to be relatively small (i.e. $\tan(\theta) \sim \theta$) and therefore $\theta$ is equal to the slope of the regression line. In other words, the slope of the regression line defines the angle of sway of the inverted pendulum model of the body during walking.

*The relationship between marker movement and the inverted pendulum model*

The record of movement of the head from its mean path was compared graphically to the movement of the head predicted by the inverted-pendulum model (product of angle and head height) in the A/P and M/L axes (Figure 3.2.4a and b). The blue trace on Figure 3.2.4a and b

which represents the movement of the head according to the inverted pendulum model, closely matches the actual movement of the head (red trace) suggesting that the inverted-pendulum model is a good approximation for movements of the upper body during walking.

To determine how inverted-pendulum motions of the body could explain much of the movement of the markers from their mean path, the variance of the marker movement was calculated. As in the analysis of quiet stance, for each marker, at each instant and on each axis there is an actual movement from the mean path ($q$) and a movement predicted by the model ($m$). The variance of '$q$' and that of '$m$' were calculated for each of the 8 'Top' markers and the results combined to give the marker and model variance for the set of markers. The variance of $q$-$m$ for the Top markers was also calculated.

The success of the inverted-pendulum model in explaining the movements of the Top after subtracting the mean path can be judged by the correlation coefficient '$r$' which is defined as:

$$r = \sqrt{\frac{V_{Top} - Vres_{Top}}{V_{Top}}}$$

Where $V_{Top}$ equals the total movement variance calculated for all the Top markers and $Vres_{Top}$ equals the variance unexplained by the model ($q$ - $m$).

Each correlation coefficient calculated underwent the standard transformation described in the Methods. The geometric mean (± 1 S.E.M.) values obtained for the young and elderly groups are presented in Table 3.2.2. A students t-test was used to compare the geometric means of the two groups and a summary of the results is also shown in Table 3.2.2.

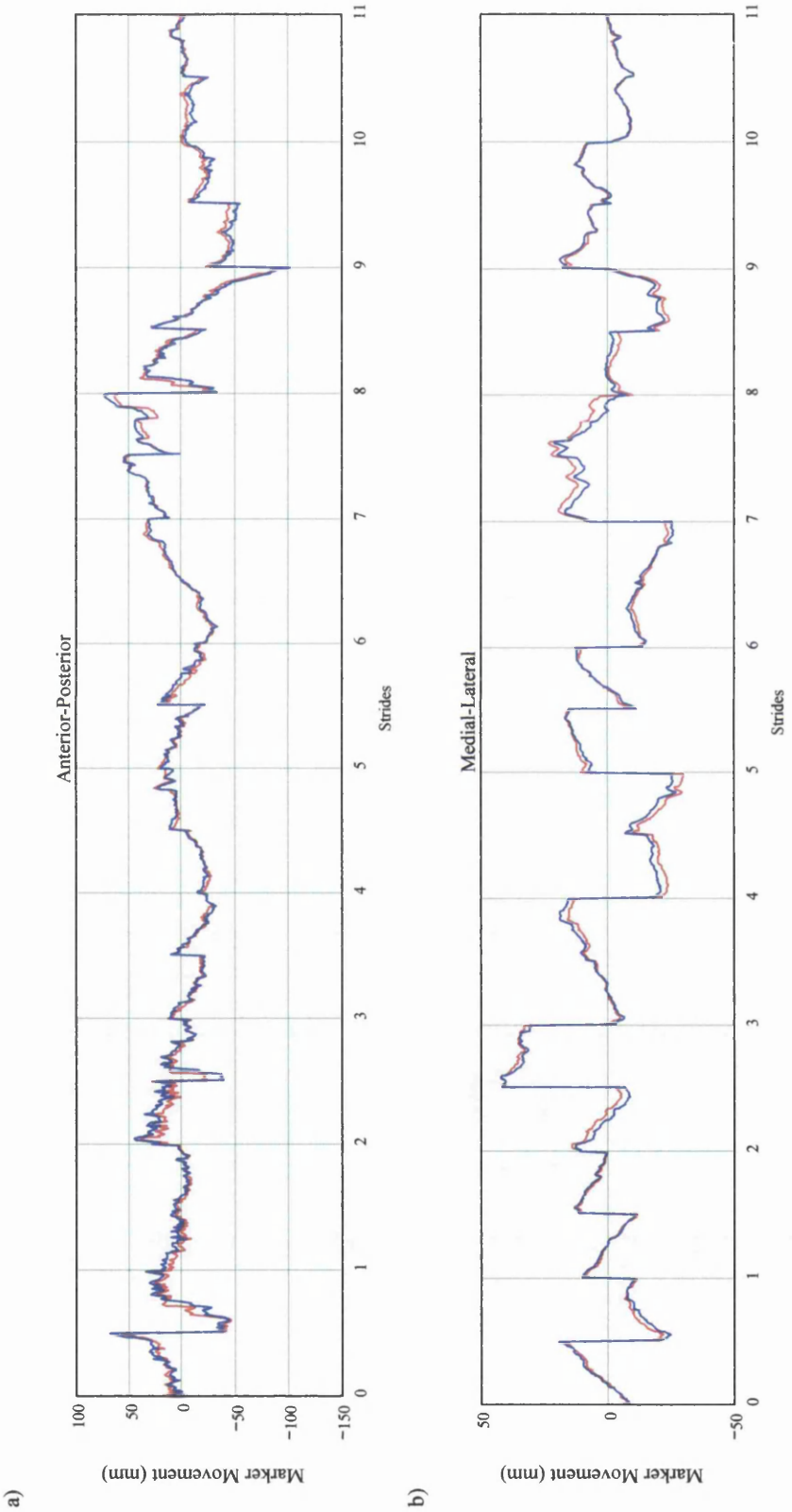



**Figure 3.2.4:** Comparison of the actual movement of the head from its mean path (red trace) with the movement of the head predicted by the inverted pendulum model (blue trace) in the A/P (a) and M/L (b) axes.

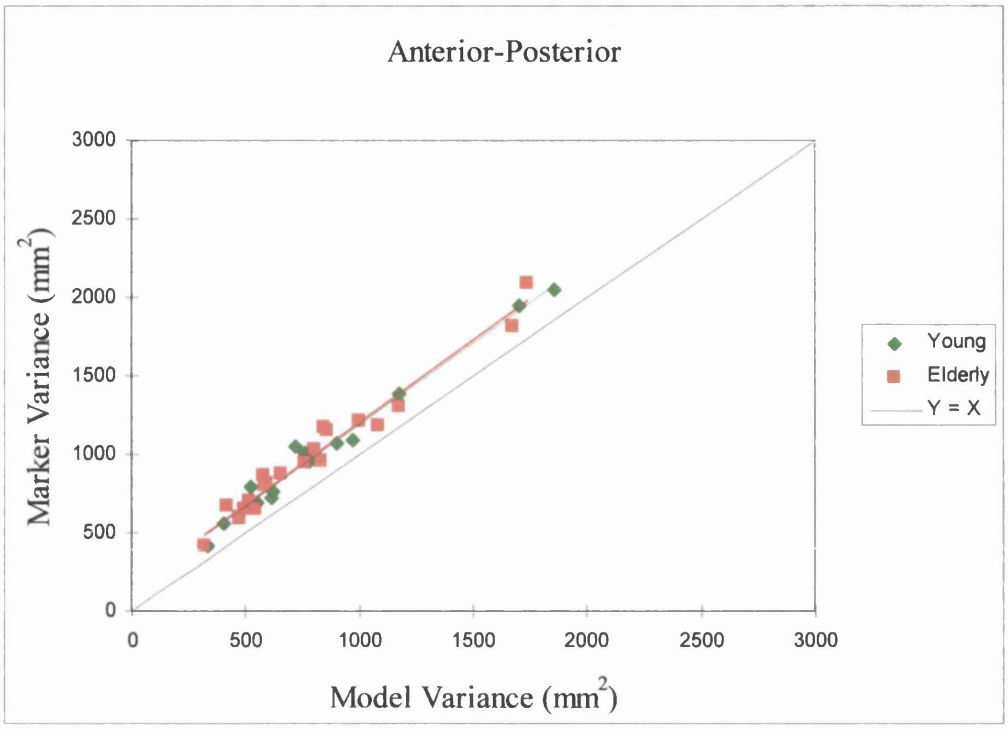
**Table 3.2.2:** Summary of the correlation coefficients calculated for the movement of the markers from their mean path with the movement of the inverted-pendulum model for walking. N(young) = 17, N(elderly) = 21. Mean (± 1 S.E.M) calculated from transformed data. Also shown are the results of a two-tailed *t*-test comparing the means of the two groups (d.f. = 36)

| AXIS | GEOMETRIC MEAN (± 1 S.E.M) | | *P* |
|---|---|---|---|
| | Young | Elderly | Young = Elderly |
| Anterior-Posterior | 0.90<br>(0.89, 0.91) | 0.90<br>(0.88, 0.90) | 0.767 |
| Medial-Lateral | 0.93<br>(0.92, 0.94) | 0.94<br>(0.94, 0.95) | 0.204 |

Large positive correlation between the movement of the markers and the movement of the model was observed in both the A/P and M/L axes and in both the young and elderly subject groups. The results also show that there was no significant difference between the means of the two groups in either the A/P or M/L axes.

For each subject, the variance of the marker movement was plotted against the inverted pendulum model variance (Figure 3.2.5a and b). A summary of the linear regression calculations calculated are given in Table 3.2.3 including the regression coefficient (slope of the regression line) and the $R^2$ values.

A)




B)

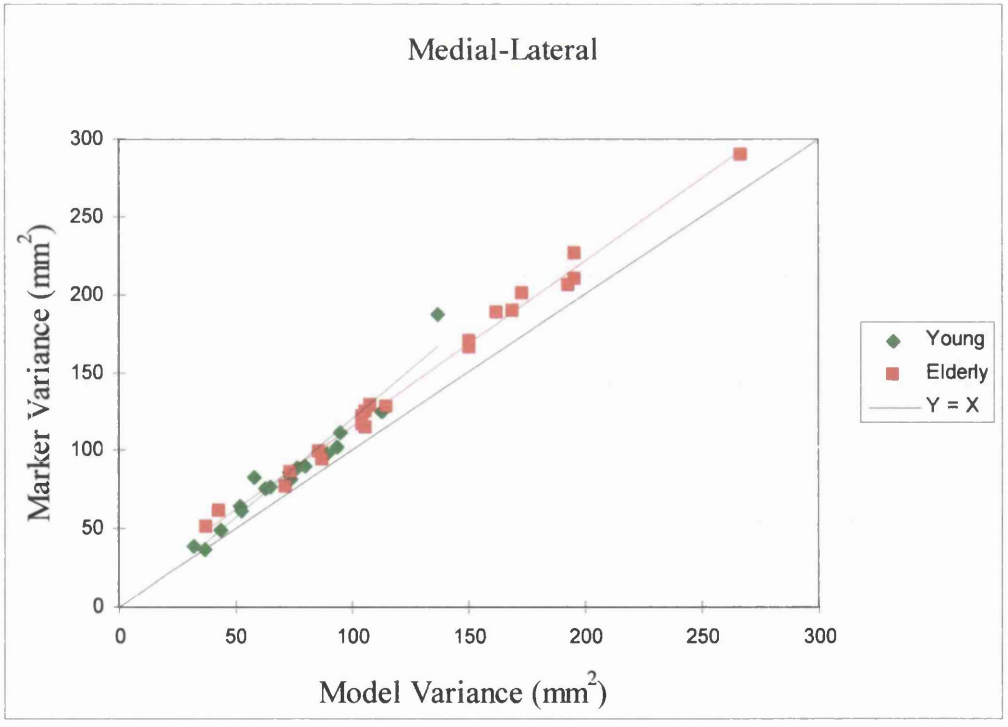



**Figure 3.2.5:** Marker variance explained by the inverted pendulum model in the A) A/P and B) M/L axes. Each point represents the variance (head, trunk and pelvis markers only) calculated for a single subject The linear regression lines calculated for the young (N=17) and elderly (N=21) are also shown. The regression coefficient and $R^2$ values are presented in Table 3.2.3.

**Table 3.2.3:** Regression coefficients and $R^2$ values obtained for the regression of marker variance on model variance for the young and elderly subjects (Figures 3.2.5a and b).

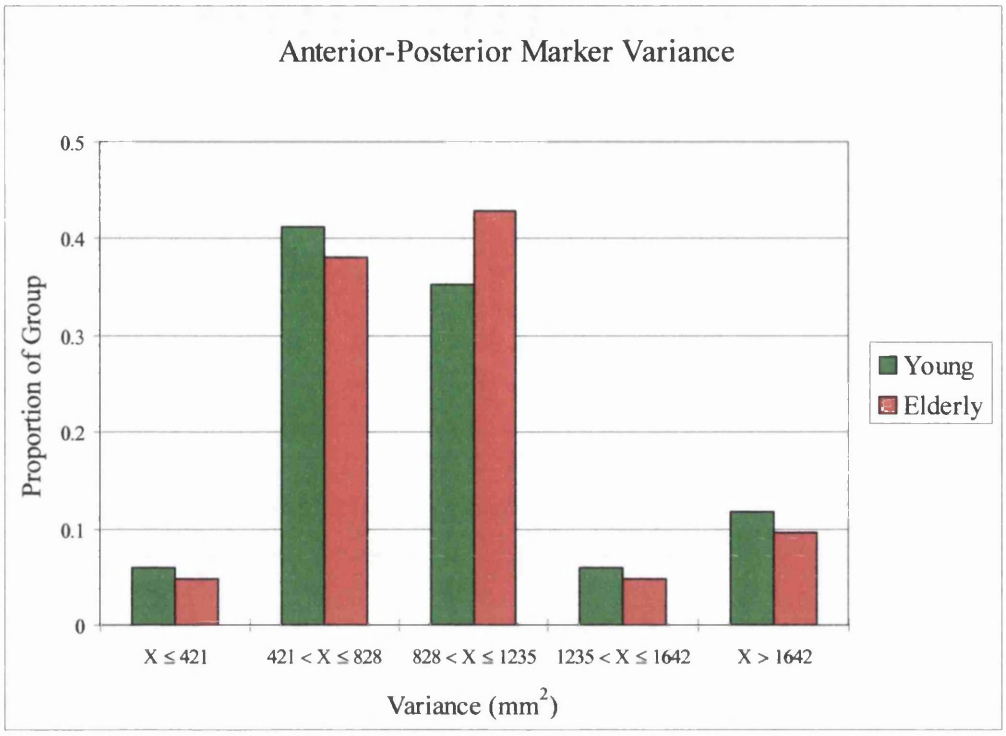
| AXIS | YOUNG (N = 17) | | ELDERLY (N = 21) | |
|---|---|---|---|---|
| | Slope | $R^2$ | Slope | $R^2$ |
| A/P | 1.04 | 0.98 | 1.05 | 0.97 |
| M/L | 1.26 | 0.94 | 1.06 | 0.99 |

The calculated regression coefficients indicate that most of the marker movement variance can be explained by the angular position of the inverted-pendulum model for both the young and elderly subject groups and in both the A/P and M/L axes. Values for the slope close to 1.0 indicate that even when the amplitude of the variance increases, the inverted-pendulum model is still valid.

*Is the variance observed for the elderly larger than that observed for young subjects?*

The values obtained for the marker and model variance for each subject were normalised using a logarithmic transformation. The mean variance values obtained for the young and elderly groups in the A/P and M/L axes were compared using a two-tailed Student's *t*-test. In the A/P axis there was no statistically significant difference between the means of the two groups for marker variance ($t = 0.0487$, $p = 0.961$, d.f. = 36) or model variance ($t = 0.144$, $p = 0.886$, d.f. = 36). However in the M/L axis the mean marker variance of the elderly group was significantly greater than that observed for the young group ( $t = -3.628$, $P < 0.001$, d.f. = 36). A similar result was observed for the M/L model variance ($t = -3.621$, $P < 0.001$, d.f. = 36). Histograms illustrating the frequency distribution of the un-transformed marker and model variance for the two groups are presented in Figures 3.2.6 and 3.2.7.

a)




b)

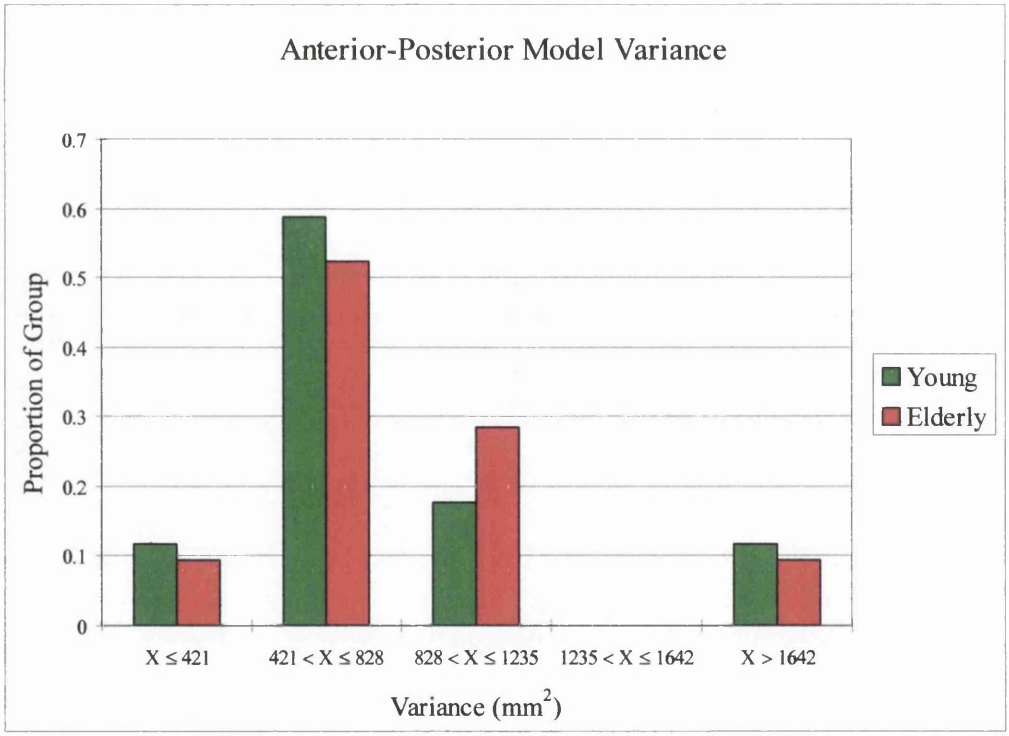
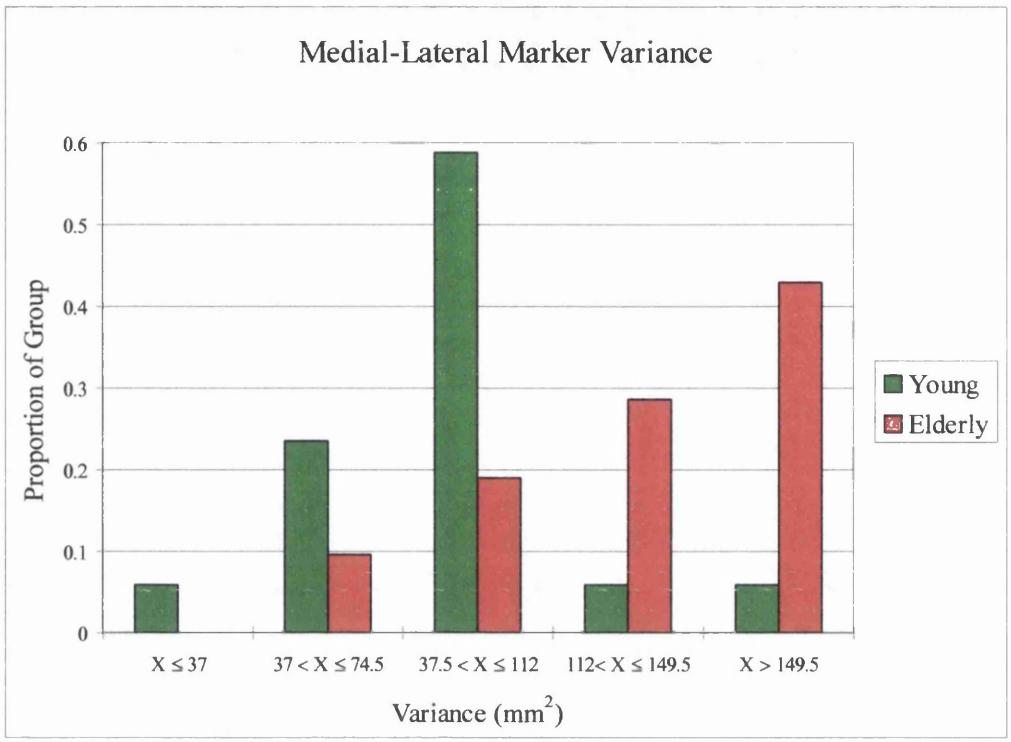



**Figure 3.2.6:** Histogram of anterior-posterior a) marker and b) model variance during walking. Area of bar is equal to the proportion of the group whose variance values fall within the limits of the bin. N(Young) = 20, N(Elderly) = 21.

a)




b)

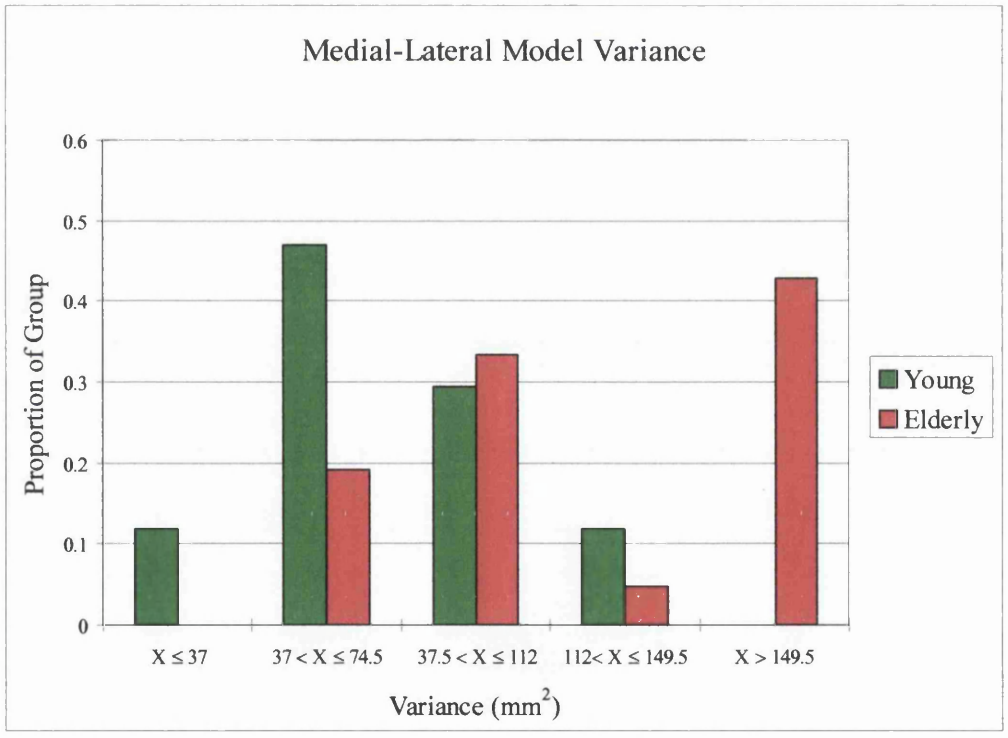



**Figure 3.2.7:** Histogram of medial-lateral a) marker and b) model variance calculated for walking. Area of bar is equal to the proportion of the group with variance values falling within the limits of the bin. N(Young) = 20, N(Elderly) = 22.

In the A/P axis, the distribution of marker and model variance for the two groups was very similar (Figure 3.2.6). However, in the M/L axis a much greater proportion of the young group had marker and model variance values in the lower three bins (88.2% in both cases). In the elderly group, only 28.6% (marker) and 52.4% (model) of the group had variance values in the lower three bins (Figure 3.2.7). Mann-Whitney Rank Sum Tests were performed on the un-transformed data and the results of these tests confirmed that there was no significant difference between the young and elderly groups in the A/P axis for the marker ($p = 1.000$) or model movement ($p = 0.849$) from mean path. However there was a significant difference between the young and the elderly groups in the M/L axis. The median variance value for the elderly group was significantly greater than that of the young group for the marker ($p = 0.001$) and model ($p = 0.001$) variance.

Subgroup Analysis

In the previous experiment investigating postural stability during standing, a selection of the elderly subjects were classified as being 'balance-impaired' (BI) or having good balance abilities (GBA) based on their performance on tandem gait, one-legged stance and foam eyes closed balance tests. Values obtained for the marker and model variance during quiet stance for the two groups were compared to values obtained for a subgroup of young subjects. A similar analysis was performed for walking. The variance data underwent a logarithmic transformation and then a one way ANOVA was used to detect for differences in the mean values of the three groups. A summary of the mean marker and model variance values calculated for each of the young, GBA and BI subgroups is presented in Table 3.2.4.

**Table 3.2.4:** Summary of geometric mean (± SEM) marker and model variance calculated for the young (N = 6), GBA elderly (N =6) and BI elderly (N = 9) subgroups.

| AXIS | Y | GBA | BI |
|---|---|---|---|
| A/P marker | 855.1 (721.1,1013.9) | 837.5 (665.4, 1054.1) | 863.0 (767.7, 970.1) |
| A/P model | 668.3 (555.6, 803.9) | 645.7 (509.3, 818.5) | 653.1 (567.8, 751.3) |
| M/L marker | 85.9 (77.5, 95.2) | 126.2 (112.1, 142.1) | 138.7 (115.2, 166.9) |
| M/L model | 75.0 (66.8, 84.2) | 112.2 (98.7, 127.6) | 120.5 (98.8, 147.0) |

The results of the one way ANOVA showed that in the A/P axis there was no significant difference between the mean marker variance ($p$ = 0.992) or mean model variance ($p$ = 0.991) for the three groups. In the M/L axis there was a trend towards increased variance in the two elderly subgroups compared to the young but the difference in mean marker and model variance between the subgroups was not significant ($p$ = 0.117 and 0.151 respectively). The failure to find a significant difference between the groups may have been partly due to the power of the tests, 0.257 (M/L marker) and 0.206 (M/L Model) being below the desired value of 0.8.

*Do subjects that sway more during quiet stance also sway more during walking?*

The original hypothesis for this experiment was that elderly subjects who made large inverted-pendulum rotations about the ankle in the M/L axis during standing would also make large lateral sway movements during walking. To investigate the relationship between standing and walking the marker variance calculated for each subject for walking (Top

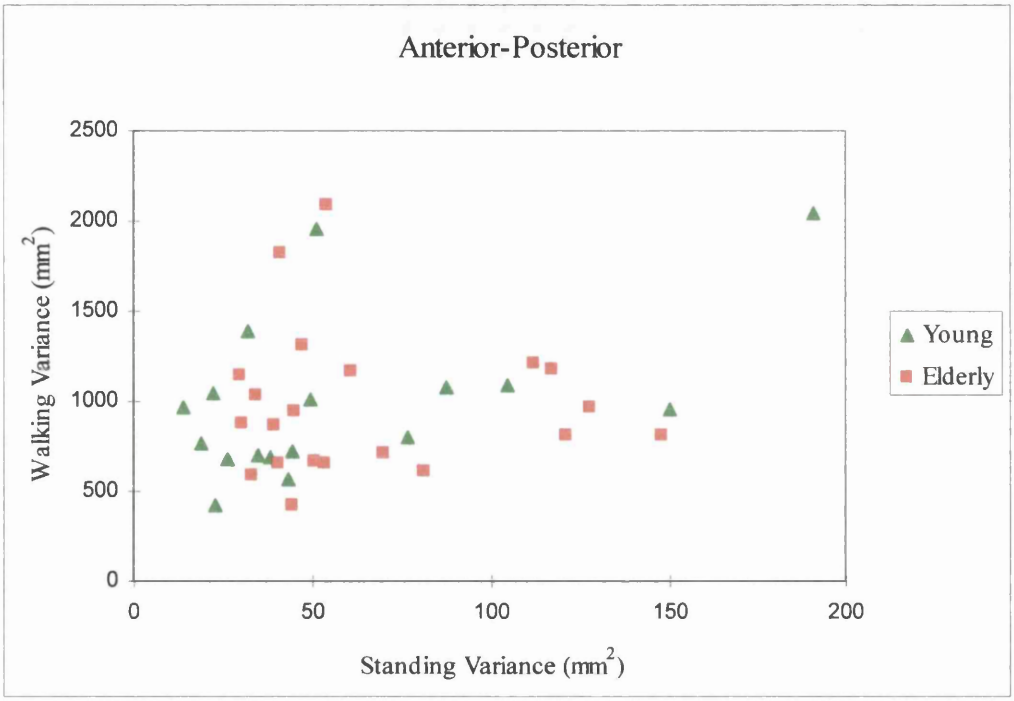
markers only) was plotted against the variance calculated for standing (all 14 markers) in the A/P and M/L axes (Figures 3.2.8a and b). In addition, a Spearman Rank Order Correlation was used to look for an association between walking variance and standing variance for the two groups. Table 3.2.5 summarises the computed correlation coefficient of the ranks and *p* values.

**Table 3.2.5:** Summary of the correlation coefficients calculated for the correlation of walking variance with standing variance. Values for '*r*' were calculated using a Spearman Rank Order Correlation. N(young) = 17, N(Elderly) = 21.

| AXIS | YOUNG | | ELDERLY | |
|---|---|---|---|---|
| | *r* | *P* | *r* | *P* |
| A/P | 0.439 | 0.076 | 0.087 | 0.703 |
| M/L | -0.0858 | 0.737 | 0.204 | 0.370 |

The variance values obtained for walking in the young and elderly are much greater than those obtained for standing in both the A/P and M/L directions (approximately 130% and 240% increase respectively, Figures 3.2.8a and b). The results suggest that in both the A/P and M/L axes there is no clear relationship between the variance of the marker movement during standing and the variance of the marker movement during walking for young or elderly subjects. This was confirmed by the results of the Spearman Rank Order Correlation tests that showed that there was no significant correlation between the two variables for either group.

A)




B)

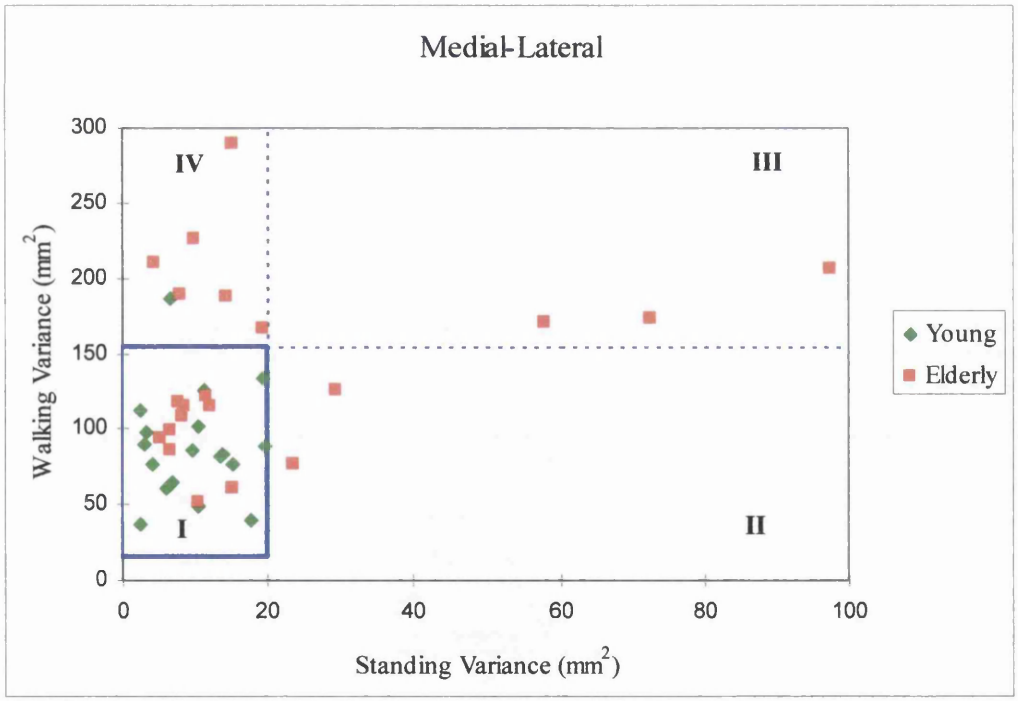



**Figure 3.2.8:** Scatterplot of marker variance calculated for walking plotted against marker variance calculated for quiet stance. Each point represents the results for a single subject. N(Young) = 17, N(Elderly) = 21. Solid blue box represents 95% confidence limits for the young group, dotted lines are an extrapolation of the upper limits of the walking and standing variance for the young group. Regions (I) to (IV) see text.

Figure 3.2.8b shows the 95% confidence limits for standing and walking variance of the young (blue box) and the dotted blue lines are an extension of the upper limits. These lines effectively divide the elderly subjects into 4 subgroups: I) 10 elderly subjects with variance values similar to the young subjects; II) 2 elderly individuals with increased standing variance but walking variance equal to young subjects; III) 3 elderly individuals with increased standing and walking variance and IV) 6 elderly subjects with 'normal' standing variance but increased walking variance. These results show that the amount of M/L sway observed during quiet stance is not a good predictor of the M/L sway during walking.

*Is there increased step width variability in the elderly?*

The mean step width (lateral distance between the medial mallioli markers in meters) during the double stance period and the stride-to-stride variability of the separation (standard deviation) was calculated for each subject (See methods). Both the mean and standard deviation values for each subject were divided by their height (m). Table 3.2.6 summarises the normalised mean step width (± S.E) and the mean stride to stride variability (S.D ± S.E) of the young and elderly groups. A *t*-test was used to compare the mean and SD values for the two groups.

**Table 3.2.6:** Summary of normalised mean step width (mean/height ± S.E) and the mean stride to stride variability (S.D/height ± S.E) for the young (N = 17) and elderly (N = 21) groups. Also shown are the results of t-tests comparing the mean values for each group.

| VARIABLE/HEIGHT | YOUNG | ELDERLY | *P* |
|---|---|---|---|
| Mean / height (± S.E) | 4.56 (±0.373) .10 $^{-2}$ | 4.79 (±0.275) .10 $^{-2}$ | 0.616 |
| S.D / height (± S.E) | 0.656 (±0.0494) .10 $^{-2}$ | 0.890 (±0.0676) .10 $^{-2}$ | 0.011* |

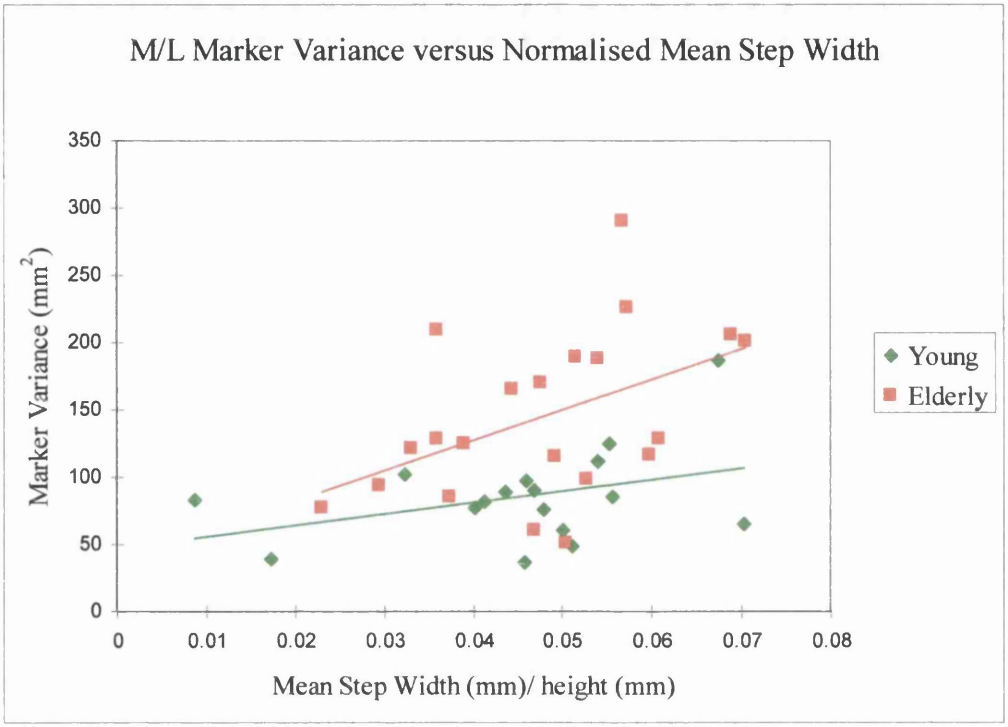
The results showed there was no statistically significant difference between the young and elderly groups for mean step width ($t = 0.506$, $P = 0.616$, d.f. = 36). However the stride-to-stride variability of the step width was significantly increased in the elderly group compared to that observed in the young group ($t = 2.675$, $P = 0.011$, d.f. = 36).

*The relationship between step width and M/L marker variance*

To determine whether the increased lateral movements of the body observed in the elderly group were associated with either a wider or more variable step width, M/L marker variance values were plotted against the mean step width (Figure 3.2.9a) and against the stride-to-stride variability in step width (Figure 3.2.9b) for both young and elderly individuals. Linear regressions were performed on the data for the young and elderly subject groups separately. For the elderly subjects, the M/L marker variance increased significantly with mean step width and with stride-to-stride variability. The slope of the regression lines were significantly different from zero, $t = 2.269$, $p = 0.035$ and $t = 4.728$, $p < 0.001$ respectively. However, for the young subjects M/L marker variance increased significantly only with stride-to-stride variability, $t = 2.855$, $p = 0.012$ and was not dependent on mean stride width ($t = 1.546$, $p = 0.143$).

The association between M/L marker variance and mean step width and M/L marker variance and stride-to-stride variability was measured by calculating the correlation coefficient '*r*' for both young and elderly subject groups. A summary of the *r*-values is presented in Table 3.2.7 along with the results of significance tests (*p* values) of the association.

a)




b)

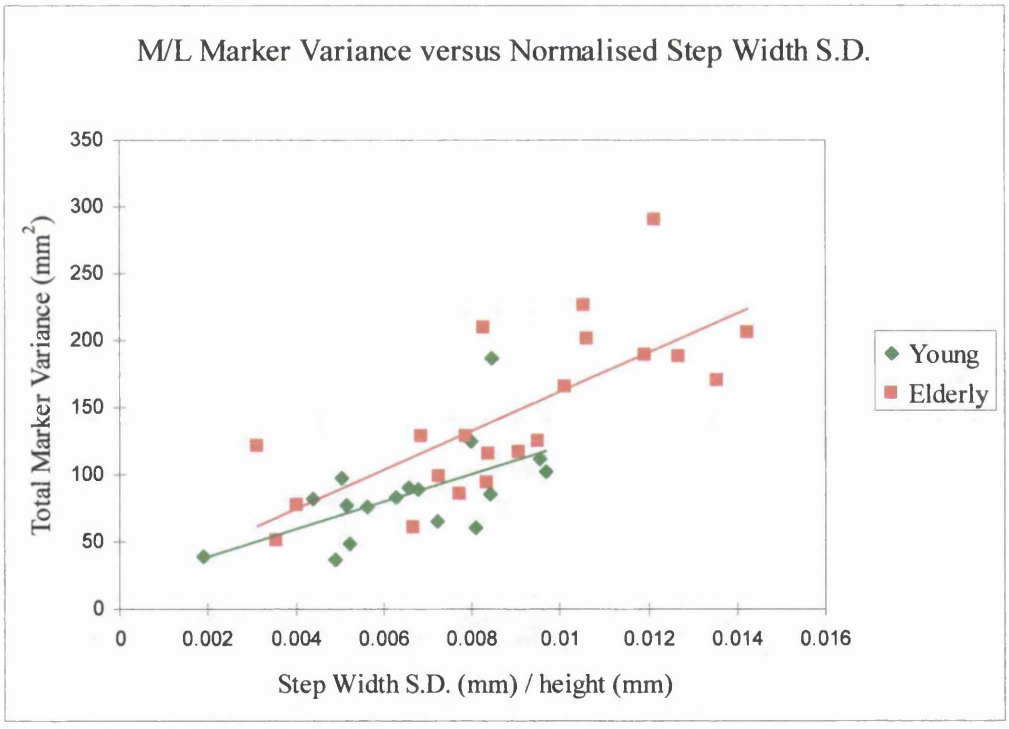



**Figure 3.2.9:** The relationship between M/L marker variance and (a) Mean step width. Equation of regression line for the young: y = 852.8x + 46.9, $R^2$ = 0.14, elderly: y = 2253.2x+ 37.4, $R^2$ = 0.21. (b) Step width S.D. Equation of regression line for the young: y = 10,319x + 18.1, $R^2$ = 0.35, elderly: y = 14,568x+ 15.6, $R^2$ = 0.54.

**Table 3.2.7:** Summary of the correlation coefficients ($r$) calculated for the correlation of M/L marker variance with the mean and S.D of step width (normalised by subjects' height). Also shown are the results of a significance test on the $r$-values. N(Young) = 17, N(Elderly) = 21.

| | YOUNG | | ELDERLY | |
|---|---|---|---|---|
| | *r* | *P* | *r* | *P* |
| mean | 0.371 | 0.143 | 0.462 | 0.035* |
| S.D | 0.593 | 0.012* | 0.735 | < 0.001** |

*, ** Denotes significant association, $P < 0.05$, 0.001 respectively

A low but significant positive correlation was found between M/L marker variance and mean step width in the elderly group ($r = 0.462$, $p = 0.035$). It is possible that the increased step width in elderly subjects with increased lateral movements of the body is an adaptation to increase stability. An association between M/L marker variance and mean step width was not observed for the young group ($p > 0.05$). The strong correlation observed between M/L marker variance and stride-to-stride variability in step width for the elderly group ($r = 0.735$) shows that the elderly who make large lateral movements of the body have variable foot placement.

## *3.3 Relationship between movements of COP and MVF/CSA*

*Subjects*

Nineteen young subjects aged between 20 and 38 years (mean ± S.E.M., 28.9 ± 1.3 years) and 56 elderly subjects aged between 58 and 94 years (71.0 ± 0.9 years) participated in this experimental study. The general characteristics of the young and elderly subjects are presented in Table 3.3.1. A summary of the clinical characteristics and the level of physical activity of the elderly subjects are presented in Tables 3.3.2 and 3.3.3 respectively.

**Table 3.3.1:** General characteristics of subject groups

| | Young (N = 19) | Elderly (N = 56) | |
|---|---|---|---|
| Mean (± S.E.M.), Median (25%,75%) or Proportion (%) | | | *P* |
| Height | 172.2 (1.7) | 164.0 (1.2) | < 0.001* |
| Weight | 66.8 (59.6,87.5) | 66.7 (58.4,74.1) | 0.268 |
| No. of women | 10/19 (52.6%) | 33/56 (58.9%) | 0.14 |

# Tests for differences associated with age: Student's *t*-Test (height), Mann-Whitney Rank Sum Test (Weight), chi-squared test (No. of women).

* Statistically significant difference between the groups at $p<0.001$.

The young group was significantly taller than the elderly group but there was no significant difference in the median weight of the two groups or in the proportion of women subjects included in each group.

**Table 3.3.2:** Clinical characteristics of elderly participants (N = 56).

| | N (% of Group) | | N (% of Group) |
|---|---|---|---|
| **Diagnoses** | | **Medications** | |
| Cardiac disease | 1 (1.8) | No medication | 18 (32.1) |
| Hypertension | 8 (14.3) | Cardio-vascular | |
| Raynaud's disease | 1 (1.8) | *Diuretic* | 6 (10.7) |
| Respiratory disease | 7 (12.5) | *ACE inhibitor* | 2 ( 3.6) |
| Diabetes | 1 (1.8) | *Calcium channel blocker* | 3 (5.4) |
| Hypothyroidism | 3 (5.4) | *Antiplatelet* | 8 (14.3) |
| Stroke / TIA | 3 (5.4) | *Beta-blocker* | 1 (1.8) |
| Visual problems | 8 (14.3) | Respiratory | |
| (cataract, glaucoma, | | *Inhaled Bronchodilators* | 4 (7.1) |
| macular degeneration) | | *Inhaled Corticosteroids* | 5 (8.9) |
| Joint pain | | | |
| neck | 3 ( 5.4) | NSAID | 5 (8.9) |
| spine | 3 ( 5.4) | Opioid Analgesic | 3 (5.4) |
| arm/shoulder | 4 ( 7.1) | Lithium | 1 (1.8) |
| hand (excluding thumb) | 7 (12.5) | | |
| knee | 7 (12.5) | Endocrine | |
| foot/ankle | 4 (7.1) | *Sulponylurea* | 1 (1.8) |
| Low back pain | 12 (21.4) | *Thyroxine* | 3 (5.4) |
| Hip replacement | 1 (1.8) | *HRT* | 4 (7.1) |
| Old knee injury | 3 (5.4) | | |
| Diagnosed osteoporosis | 4 (7.1) | Gastro-intestinal | |
| History of neoplasia | 6 (10.7) | *Acid suppressing* | 3 (5.4) |
| (breast, prostrate, colon, | | *Anti-spasmodic* | 1(1.8) |
| meningioma) | | | |
| Dysequilibrium - cause | 3 (5.4) | Genito-urinary disorders | |
| unknown /uninvestigated | | *Oxybutynin* | 2 (3.6) |
| Psychotic illness | 1 (1.8) | | |

**Table 3.3.3:** Characteristics of elderly participants: mobility, experience of falling and activity level (N = 56).

| | N (% of Group) |
|---|---|
| **Use of mobility aid** | 2 (3.6) |
| **Falls in the past year** | |
| At least once | 21 (37.5) |
| More than one fall | 6 (10.7) |
| **Physical Activity** | |
| (1-2 times/week) | |
| Light | 21 (37.5) |
| *(walking < ½ hour)* | |
| Moderate | 22 (39.3) |
| *(walking > ½ hour, bowls, gardening)* | |
| Vigorous | 13 (23.2) |
| *(jogging, tennis, keep-fit, cycling, swimming)* | |

*Correlation between MVF and CSA in young and elderly subjects*

Significant correlation was found between MVF and CSA for both young and elderly subject groups. The correlation coefficient was higher for the young subjects ($r = 0.81$, $p < 0.001$, Pearson Product Moment Correlation) than for the elderly subjects ($r = 0.54$, $p < 0.001$, Spearman Rank Order Correlation). The MVF produced by the elderly was 20.3 ± 2.6 % (mean ± SEM) less than that predicted from their CSA using the regression line for the young subjects (Y = 25.867 + (0.109*CSA)).

*Decline in MVF/CSA with age*

There were no significant difference in MVF/CSA between the elderly women and the elderly men ($t = -0.17$, $p = 0.866$, d.f. = 54) so the results of the two groups were combined. Similarly there was no significant difference in MVF/CSA for the young women and young men ($t = 0.853$, $p = 0.406$, d.f. = 17) so their results were also combined. When the results of the total elderly group were compared to the total young group, a significant difference

between the means of the two groups was observed. The elderly group were significantly weaker than the young group ($t$ = 2.585, $p$ = 0.012, d.f. 72). A summary of the mean (± S.E.M.) of the MVF/CSA ratio for the two groups is presented in Table 3.3.4.

**Table 3.3.4:** A summary of the mean (± S.E.M.) of the MVF/CSA ratio for the young group (N = 19) and for the elderly group (N = 56). The result of a two-tailed t-test comparing the means of the two groups is also shown.

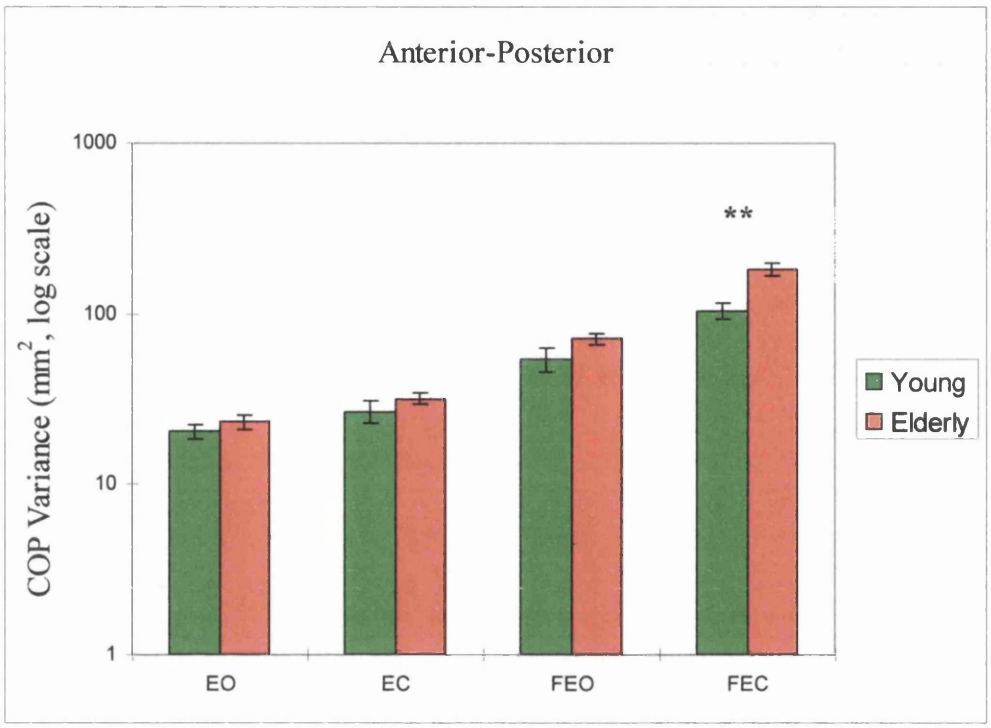
| | YOUNG MEAN(SEM) | ELDERLY MEAN (SEM) | *P* |
|---|---|---|---|
| Men & Women | 0.167 (0.008) | 0.141 (0.005) | 0.012* |

* Denotes significant difference, $p < 0.05$

*Comparison of COP variance in the young and elderly subjects*

The variance of the COP movements in the A/P and M/L axes was calculated for each young and elderly subject for each of the 4 test conditions, EO, EC, FEO and FEC. A logarithmic transformation was used to obtain a normal distribution of the data. The A/P and M/L geometric mean variance (± SEM) calculated for the young and elderly groups for each of the four test conditions (EO, EC FEO, FEC) is illustrated in Figures 3.3.1a and b. Two-tailed t-tests were used to test for differences in the mean values of the two groups. The A/P FEO and M/L EO data failed the normality and equal variance tests even after transformation, so Mann-Whitney Rank Sum Tests were used to compare the medians of the two groups. A summary of these results is presented in Table 3.3.5.

A)




B)

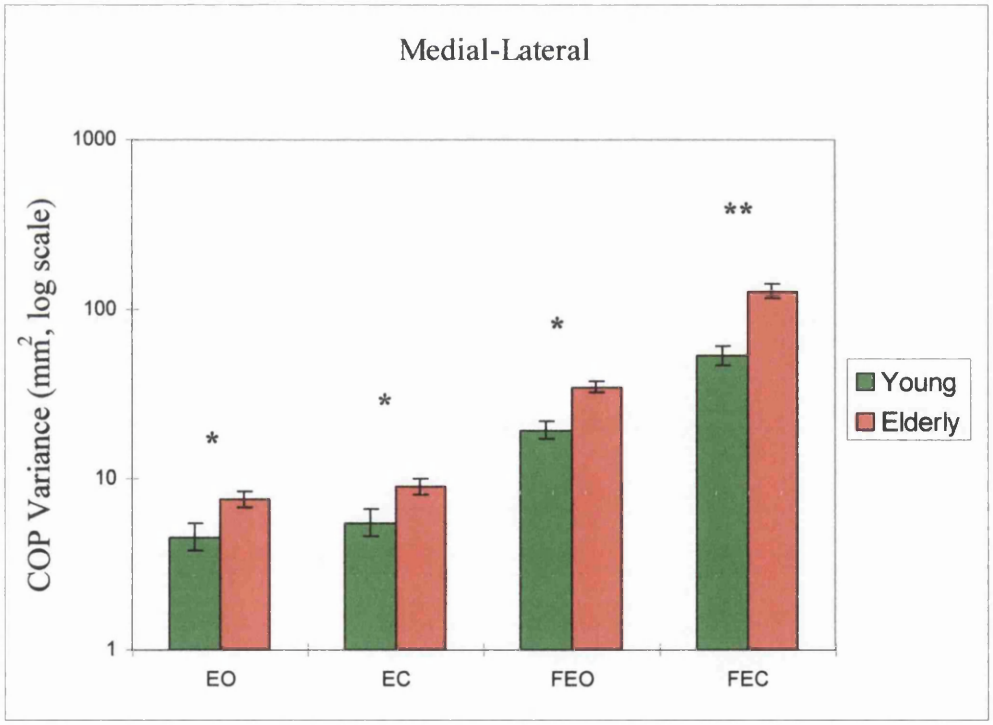



**Figure 3.3.1:** A) A/P and B) M/L COP variance (geometric mean ± S.E.M.) for the young and elderly groups under the 4 test conditions, eyes open (EO), eyes closed (EC), foam eyes open (FEO), foam eyes closed (FEC). N(Young) = 19 for all tests, N(Elderly) = 56 (EO), 55 (EC,FEO), 38 (FEC). *,** denotes significant difference, $p<0.05$, 0.001 respectively.

**Table 3.3.5:** Results from two-tailed *t*-tests and Mann-Whitney Rank Sum tests comparing the mean and median (#) COP variance values of the young and elderly groups. N(Young) = 19 for all tests, N(elderly) = 56 (EO), 55 (EC,FEO), 38 (FEC).

| TEST | DIRECTION OF COP | |
|---|---|---|
| | A/P | M/L |
| EO | N.S | #* |
| EC | N.S | * |
| FEO | #N.S | ** |
| FEC | ** | ** |

*, ** Denotes statistically significant difference $P < 0.05$, 0.001 respectively,

N.S. no statistically significant difference

Although there is a general trend towards increased COP variance in the elderly subjects in the A/P direction, a significant difference between the young and elderly subject groups was observed only in the FEC test condition i.e. when visual inputs, proprioceptive inputs from the ankles and cutaneous inputs from the soles of the feet were reduced. However, the power of the A/P EO and EC tests (0.05 and 0.078 respectively) was below the generally accepted value of 0.8 and therefore the negative findings for the A/P direction need to be treated cautiously. In the M/L direction, a significant difference was observed between the mean (and median) values for the young and elderly for all test conditions and the difference between the two groups was greatest for the FEC test condition.

*Comparison of COP variance (EC) results with those obtained in Section 3.1*

The movement of the COP during the eyes closed (EC) test condition has been recorded for the same 17 elderly subjects on two separate occasions, approximately 8 months apart. The data described in this section were collected prior to that described in Section 3.1. Although the duration of the test and the experimental protocol differed slightly, the expectation was

that the values obtained for COP variance on the two occasions would be similar. A Wilcoxon Signed Rank Test was used to detect for a change in the COP variance over time. The results showed that the median A/P and M/L COP variance calculated for the group on the first occasion (31.39 $mm^2$ and 7.75 $mm^2$ respectively) was not significantly different ( $p$ = 0.243 and 0.487) from that calculated on the second occasion (32.89 $mm^2$ and 8.692 $mm^2$). However, two of the elderly subjects showed a dramatic increase in the COP variance between the two test occasions, particularly in the M/L direction. One of these subjects had difficulty performing the eyes closed test on the second occasion because of unsteadiness and was able to complete only two of the three 60 second trials. This subject had a diagnosis of type II diabetes mellitus and was taking an oral antidiabetic drug (sulphonylurea) to augment insulin secretion. The dosage of his medication had doubled since the first visit and it is possible that the increased instability observed during the second visit may have been caused by hypoglycaemia, a recognised hazard of this medication when used in the elderly. In addition, the feet of diabetic patients are often affected by a combination of peripheral neuropathy and peripheral vascular disease and these conditions may have contributed to unsteadiness during the eyes closed stance test. The second subject was taking lithium carbonate to control for mania and had a previous history of syncope and giddiness. Aspirin had also been prescribed as an antiplatelet therapy. Therapeutic agents such as lithium and Aspirin can act on peripheral and/or central vestibular systems to produce equilibrium disorders (Matsuoka *et al.* 1986). The margin between the therapeutic and toxic serum-lithium concentration is relatively narrow and the elderly are particularly susceptible to toxicity due to decreasing renal function and hence elimination. Acute intoxication with lithium causes 'vermis syndrome' ataxia, vertical nystagmus and peripheral neuropathy (Yoshimoto, 1978). Other adverse effects associated with lithium toxicity include muscle weakness and tremor. Acute salicylate (Aspirin) poisoning can produce an increase in labyrinthine pressure as well as damage to hair cells. The resultant tinnitus and hearing loss are similar to those seen in Meniere's disease (Walter, 1955). The dose of aspirin used as

antiplatelet therapy is unlikely to have such adverse effects, however in a previous study it was noted that serum-lithium concentrations were increased in a patient receiving aspirin (Bendz and Feinberg, 1984) and thus the combination of the two medications may have serious consequences. Because of their health status and apparent change in postural stability from one test occasion to the next these two subjects were excluded from analyses investigating the association between variables measured on the two different occasions.

*Correlation between MVF/CSA and COP measures in the elderly*

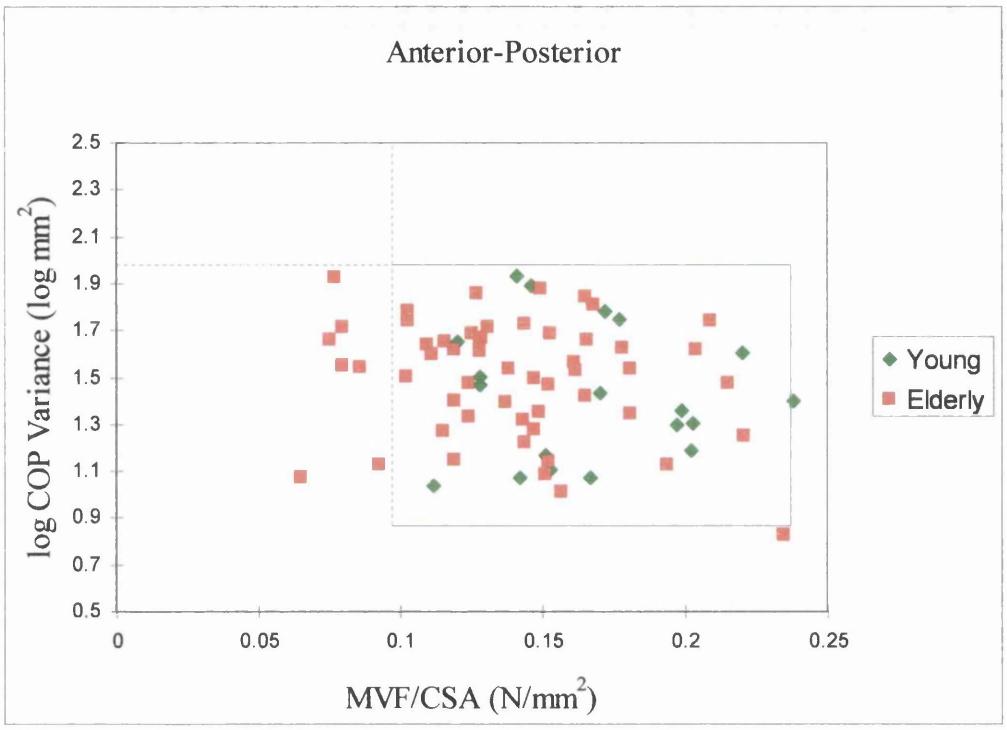
To determine whether there was an association between the age-related decline in MVF/CSA and increased COP variance in the elderly, log COP variance values in the A/P and M/L axes were correlated with values of MVF/CSA using Pearsons Product Moment correlation (or Spearman Rank Order correlation when data was found to be not normally distributed). A summary of the calculated correlation coefficient ($r$) values and the results of significance tests of the association ($p$ values) are presented in Table 3.3.6.

**Table 3.3.6:** Summary of the correlation coefficients calculated for log COP variance correlated with MVF/CSA for the elderly subjects. EO test (N = 56), EC Test (N = 55), FEO (N = 55), FEC (N = 38). # Spearman Rank Order Correlation used.

| TEST | AXIS | *r* | *P* |
|---|---|---|---|
| EO | #A/P | -0.059 | 0.666 |
| | M/L | 0.026 | 0.851 |
| EC | A/P | -0.214 | 0.117 |
| | M/L | 0.060 | 0.664 |
| FEO | A/P | 0.097 | 0.482 |
| | M/L | -0.152 | 0.267 |
| FEC | A/P | 0.310 | 0.058 |
| | M/L | 0.299 | 0.068 |

For three of the four test conditions (EO, EC, FEO) no significant association between MVF/CSA and log COP variance was observed in either the A/P or M/L direction. The majority of the calculated correlation coefficients (*r-values*) are very close to zero. The sign of the correlation coefficient (negative or positive) appears to be random, with 3 of the results positive and 3 negative. Scatterplots of log COP variance (EC) against MVF/CSA for the young and elderly subjects are presented in Figures 3.3.2a and b. In the A/P direction, 7 subjects have MVF/CSA values below the lower limits for the young but they have log COP variance values that fall within the 95% confidence limits of the young. In the M/L direction, the 55 elderly subjects can be divided into 4 rather unequal groups; I) 6 elderly subjects have low MVF/CSA values but COP values within the limits of the young, II) the majority of elderly subjects (N = 46) have MVF/CSA and COP values similar to the young subjects, III) 2 elderly have greater COP values than the young but have MVF/CSA values within the limits of the young, IV) one subject has both reduced MVF/CSA and greater COP values compared to the young. The distribution of the subjects between the groups suggest that in general, elderly with reduced MVF/CSA and elderly with increased postural sway are two separate populations.

A)




B)

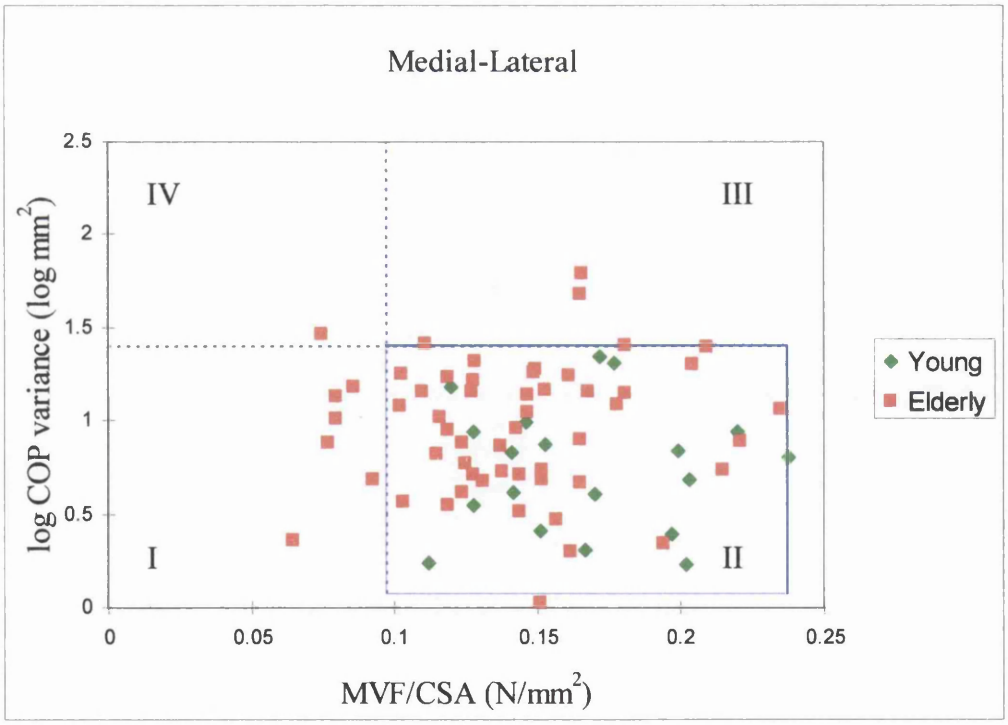



**Figure 3.3.2:** Scatterplots of log COP variance (EC) against MVF/CSA for young (N=19) and elderly (N=55) subject groups. Solid blue box represents the 95% confidence limits for the young group, the dotted blue lines are an extrapolation of the lower limit for MVF/CSA and the upper limit for COP variance of the young group.Regions (I) to (IV) see text.

In the FEC condition, a low, positive correlation was observed between MVF/CSA and COP variance in both the A/P and M/L direction but the values did not reach statistical significance. The prior expectation was that a negative correlation would exist between COP variance and MVF/CSA i.e. elderly subjects who were weak would also exhibit increased postural sway. Hence the suggestion of a positive correlation was rather surprising. However, only 38 of the 56 elderly individuals were able to complete this test and the mean MVF/CSA ratio for these 38 elderly subjects (0.147 ± 0.006, Mean ±SEM) was not statistically significant different from that of the young subjects. The mean MVF/CSA ratio for the elderly subjects who could not complete the FEC test was 0.1429 ± 0.009, (Mean ±SEM). This value is significantly less that the mean ratio for the young adults ($t$ = -2.993, d.f = 36, $p$ = 0.005) indicating that the elderly subjects excluded from the FEC analysis included those with reduced MVF/CSA. The exclusion of a number of the more balance impaired and/or weaker subjects may have had some influence on the sign and magnitude of the correlation. To investigate this, the 18 subjects that were unable to complete the test were assigned COP variance values equal to the mean + 3 SD of the value obtained for the 38 elderly subjects that were able to complete the FEC test (mean + 3 SD, A/P = 544 $mm^2$, M/L = 385 $mm^2$). A Spearman Rank Order Correlation was used to determine if there was an association between log COP variance and MVF/CSA for all 56 subjects. In the A/P direction, the inclusion of the estimated results for the 18 elderly subjects reversed the low positive correlation observed previously to a low negative correlation. This was also not statistically significant ($r$ = -0.114, $p$ = 0.403). In the M/L direction, there was no significant correlation between the two variables and the correlation coefficient was close to zero ($r$ = 0.0362, $p$ = 0.790).

In summary, there is no significant association between MVF/CSA and log COP variance for any of the four test conditions (EO,EC,FEO,FEC). Elderly individuals with increased COP variance are a separate population from those with reduced MVF/CSA.

*Correlation between MVF/CSA and walking variance in the elderly*

To determine if there was an association between MVF/CSA and the amount of movement of the body from the mean path during walking in the elderly, the MVF/CSA ratio was correlated with A/P and M/L walking variance. Fifteen elderly subjects (including 6 women) participated in both sets of experiments. A summary of the correlation coefficients (Pearson Product Moment Correlation) and *p* values are presented in Table 3.3.7.

**Table 3.3.7:** Summary of the correlation coefficients calculated for the correlation of MVF/CSA with A/P and M/L walking variance, elderly subjects only (N = 15).

| DIRECTION | *r* | *P* |
|---|---|---|
| A/P | -0.447 | 0.095 |
| M/L | 0.168 | 0.549 |

No significant association was observed between MVF/CSA and walking variance in either the A/P or M/L direction for the group of 15 elderly subjects. The group mean (± S.E.M.) MVF/CSA was 0.153 (± 0.043 $N/mm^2$) which was not significantly different ($t$ = -1.008, d.f = 32, $p$ = 0.321) from that calculated for the young adults (0.167 ± 0.036 $N/mm^2$). This suggests that for this group of relatively strong elderly subjects there is no association between balance during walking and MVF/CSA

*Correlation between COP variance and walking variance*

COP variance (untransformed data) for the 4 test conditions (EO, EC, FEO, FEC) was correlated with the A/P and M/L walking variance for the fifteen elderly subjects who participated in both sets of experiments. A Pearson Product Moment Correlation was used to calculate the *r* values, except in the case of M/L walking variance correlated against FEO M/L COP variance where a Spearman Rank Order Correlation was used. A summary of the correlation coefficients and *p* values are presented in Table 3.3.8.

**Table 3.3.8:** Summary of the correlation coefficients calculated for A/P and M/L walking variance correlated with COP variance measures for the elderly subjects (N = 15). A Spearman Rank Order Correlation was used to calculate the correlation between M/L walking variance and FEO M/L COP variance.

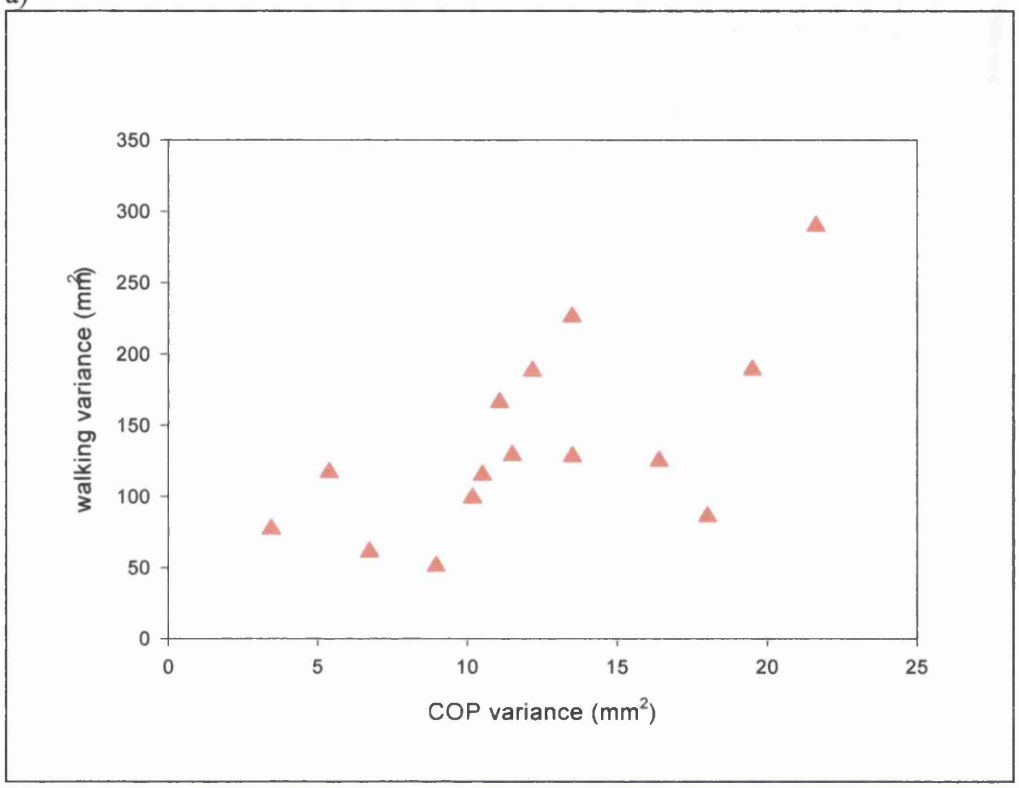
| TEST | AXIS | *r* | *P* |
|---|---|---|---|
| EO | A/P | -0.433 | 0.107 |
| | M/L | 0.645 | 0.009* |
| EC | A/P | -0.313 | 0.255 |
| | M/L | 0.465 | 0.080 |
| FEO | A/P | 0.239 | 0.392 |
| | M/L | -0.207 | 0.449 |
| FEC# | A/P | -0.191 | 0.573 |
| | M/L | 0.818 | 0.002* |

# N = 11

*, Denotes a significance of P < 0.01

The correlation between A/P walking variance and A/P COP variance was not significant for any of the test conditions. Similarly there was no significant correlation between M/L walking variance and M/L COP variance for the EC and FEO test conditions. However a significant correlation was observed between M/L walking variance and M/L COP variance for the EO and FEC test conditions. Scatter plots of the M/L walking variance against M/L EO and FEC COP variance are presented in Figures 3.3.3a and b.

a)




b)

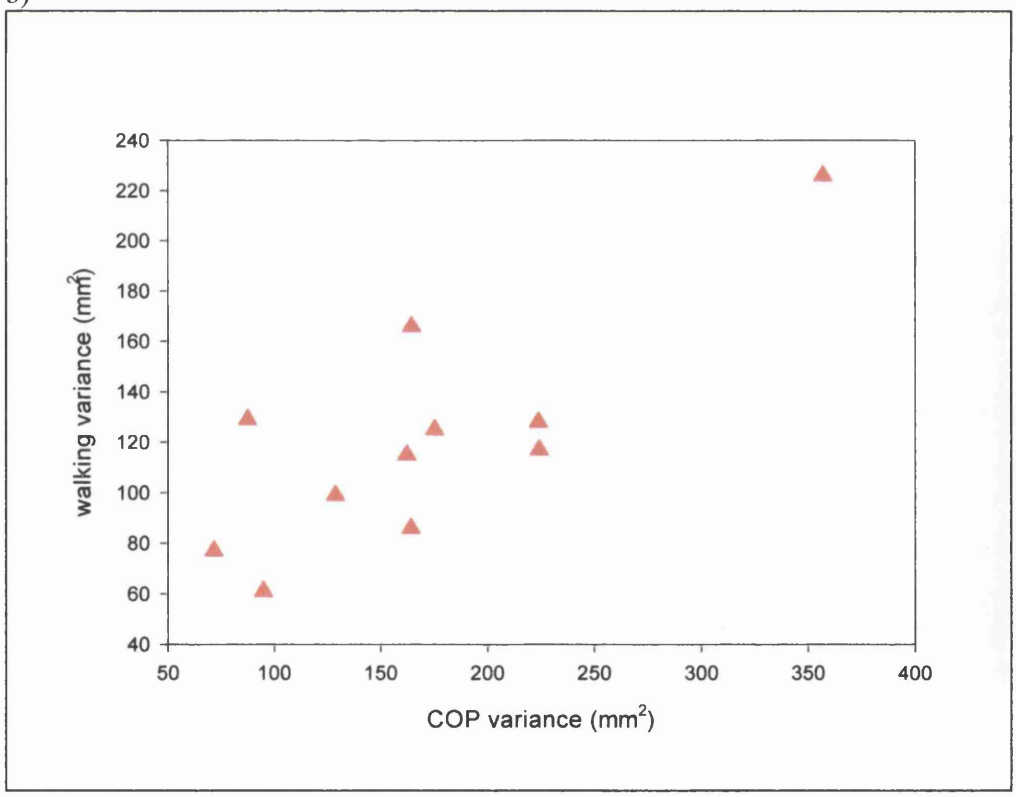



**Figure 3.3.3:** Scatterplots of M/L walking variance against M/L COP variance for the elderly subjects that participated in both experiments. (a) Eyes Open (N = 15) and (b) Foam Eyes Closed, (N = 11).

## 3.4 *Recovery responses to tripping*

*Subjects*

Nine young men and women (age: 20 - 34 years, mean 27.4 years) participated in this experiment. The general characteristics of each subject are listed in Table 3.4.1.

**Table 3.4.1:** General characteristics of the nine subjects.

| SUBJECT NO. | SEX | AGE (YEARS) | HEIGHT (M) | WEIGHT (KG) |
|---|---|---|---|---|
| 1 | male | 34 | 1.796 | 74 |
| 2 | male | 24 | 1.805 | 69 |
| 3 | male | 32 | 1.782 | 66 |
| 4 | male | 20 | 1.720 | 67 |
| 5 | male | 20 | 1.824 | 84 |
| 6 | male | 27 | 1.770 | 84 |
| 7 | male | 32 | 1.778 | 74 |
| 8 | female | 32 | 1.750 | 60 |
| 9 | female | 26 | 1.534 | 64 |
| **Mean** | | **27.4** | **1.751** | **71.3** |
| **(S.E.M.)** | | **(1.8)** | **(0.029)** | **(2.8)** |

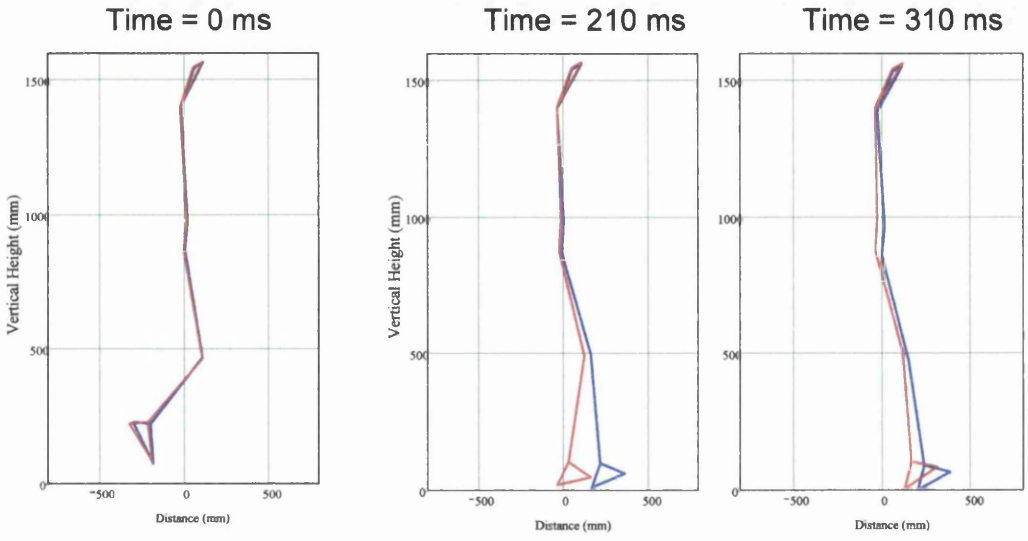
*Kinematics of the obstructed swing limb following the perturbation*

The gait perturbations were applied to the right limb at the beginning of the swing phase (toe off). Two different recovery strategies were observed in response to the perturbation. The most common movement outcome (7 out of 9 subjects) was for the subject to lengthen the swing phase of the perturbed limb so that heel strike was delayed in perturbed strides compared to non-perturbed strides (**Recovery Strategy 1**). In contrast, Subjects No.7 and 9 tended to return the obstructed foot to the treadmill earlier in perturbed strides compared to non-perturbed strides, thus shortening the swing phase (**Recovery Strategy 2**).

A series of sagittal plane stick figure diagrams illustrating the kinematics of the right limb, trunk and head during normal walking and after a 180 ms perturbation are presented in Figure 3.4.1. The red stick figures describe the kinematics of the body during a perturbed stride and are the mean of 5 perturbations. The blue stick figures represent the movement of the body during normal walking and are the mean of approximately 80 strides. The results for an individual (Subject No. 4) using **Recovery Strategy 1** are shown in Figure 3.4.1a and the results for Subject No. 9 (**Recovery Strategy 2**) are presented in Figure 3.4.1b. The first diagram (Time = 0 ms) in Figures 3.4.1a and b show the time point at which the peak perturbation force is recorded at the ankle. For **Recovery Strategy 1** (Figure 3.4.1a) the second diagram (Time = 210 ms) illustrates the time at which heel strike occurs during a normal walking stride and the third diagram (Time = 310 ms) shows when heel strike occurs in the perturbed stride. For subjects using **Recovery Strategy 2** the second diagram (Time = 230 ms) illustrates the earlier heel strike in perturbed strides compared to non-perturbed strides (third diagram, Time = 320 ms).

The mean non-perturbed stride time for the 7 subjects using **Recovery Strategy 1** was 1.12 ± 0.01 seconds (Mean ± SEM). In response to a 240 ms perturbation, heel strike was delayed by 0.06 ± 0.03 s and the perturbed step was 74.6 ± 19.3 mm shorter (Mean ± SEM, N = 7) than during normal walking. The two subjects using **Recovery Strategy 2** had a shorter non-perturbed stride time (1.03 s and 1.02 s for subjects 7 and 9 respectively) compared to subjects using **Recovery strategy 1** and this factor may have influenced their choice of recovery strategy. Following a 240 ms perturbation, heel strike was advanced by 0.07 ± 0.01 s (Mean ± SEM, N = 2). The perturbed step length was considerably shorter than normal particularly in response to the 240 and 180 ms perturbations. For example for Subject 9 the stride length was decreased by 321.4 mm (mean of 5 perturbations) after a 240 ms perturbation.

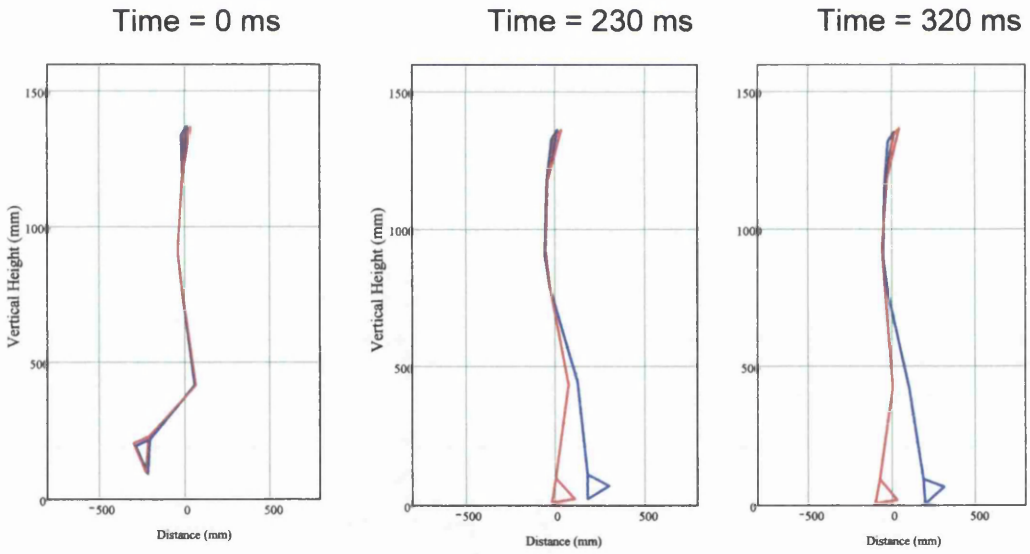
a) **Recovery Strategy 1**




Start of perturbation

Heel strike
Contact occurs *later* in perturbed (red) strides

b) **Recovery Strategy 2**




Start of perturbation

Heel strike
Contact occurs *earlier* in perturbed (red) strides

**Figure 3.4.1:** Kinematic changes in the right (obstructed) limb following a 180 ms perturbation of gait. Red stick figure represents the mean of 5 trips. Blue stick figure is the mean of approximately 80 unperturbed strides. a) The response observed for a subject using **Recovery Strategy 1.** b) The response of a **Recovery Strategy 2 subject**.

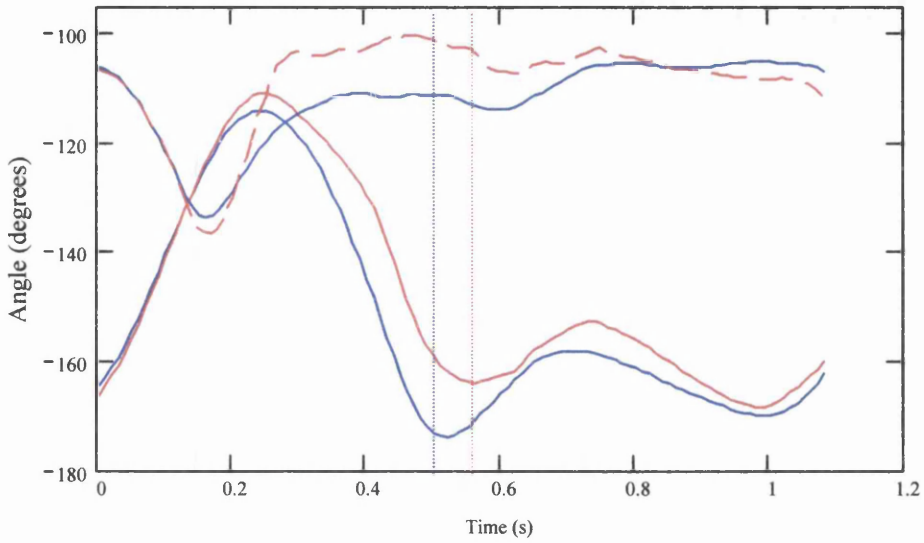
*Changes in knee and ankle angle following the perturbation*

Figure 3.4.2a illustrates the changes in knee and ankle angle for an individual using **Recovery Strategy 1** in response to a 180 ms perturbation. The braking of the foot at the heel tends to plantar-flex the foot. To counteract this, the subject increases the dorsi-flexion of the foot (red dashed line) and keeps it more flexed until after heel contact. The knee (solid red line) is also more flexed following the perturbation and remains slightly flexed even at heel strike. Figure 3.4.2b illustrates the changes in knee and ankle angle observed in Subject 9 in response to the 180 ms perturbation. There is a small increase in dorsi-flexion of the ankle following the perturbation (dashed red line), and as before, the knee is considerably more flexed at heel strike in the perturbed stride (solid red line). In addition, it is obvious from the plots of ankle and knee angle that the swing phase of the obstructed leg, and the stance phase of the contralateral leg have both been shortened following the perturbation as the next gait cycle has begun much earlier than in non-perturbed strides.

*Changes in the trajectory of the toe-marker*

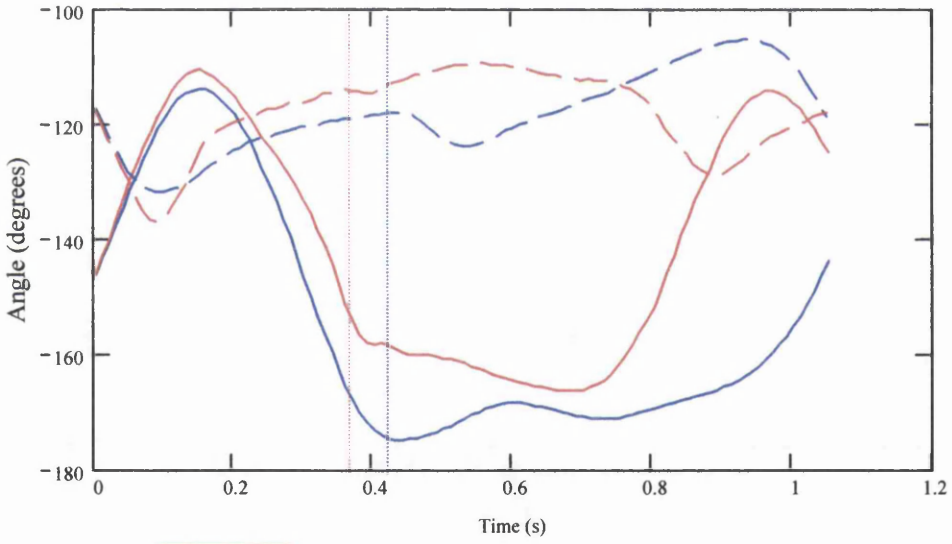
One notable feature of **Recovery Strategy 2** was a more flattened heel contact. During the swing phase of normal walking the foot is dorsiflexed just prior to heel strike and once heel strike occurs the foot is lowered to the ground in a controlled manner. Figure 3.4.3 illustrates for Subject 9 the trajectory of the toe marker during normal walking (blue traces) and during a perturbed stride (red traces). The double peaks observed in the toe trajectory of the non-perturbed stride prior to heel contact are reduced to a single peak following a perturbation. The second peak observed late in the plot of the perturbed stride represents the next 'toe off' by the right limb. For subjects using **Recovery Strategy 1,** the trajectory of the toe marker remained relatively unchanged following the perturbation, although there was a shift in the time frame to coincide with the delayed heel contact in perturbed strides.

A)




Perturbation

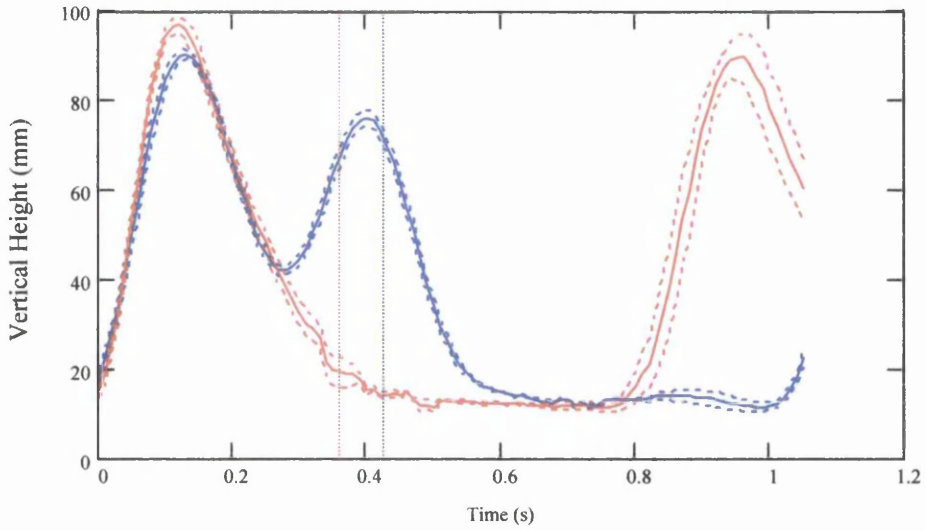
B)




Perturbation

**Figure 3.4.2:** Changes in knee angle (solid lines) and ankle angle (dashed lines) following a 180 ms perturbation. A) Results for Subject 4 (**Recovery Strategy 1**). B) Results for Subject 9 (**Recovery Strategy 2**). Blue lines represent the mean results for 84 non-perturbed strides and red lines represent the mean of 5 trips. Vertical dotted lines represent heel strike in the non-perturbed strides (blue) and perturbed strides (red) respectively.

**Figure 3.4.3**: Trajectory of toe marker during normal walking and following a 180 ms perturbation for an individual (Subject 9) using **Recovery Strategy 2**. Blue lines represent the mean and SEM of 84 unperturbed strides. Red lines represent the mean and SEM of 5 trips. Vertical dotted lines represent heel strike in the unperturbed (blue) and perturbed (red) strides respectively.




Perturbation

In summary, two distinct recovery strategies were observed in the young adults in response to a trip. The key changes in the kinematics of the obstructed swing limb for each **Recovery Strategy** are listed in Table 3.4.2.

**Table 3.4.2:** Key changes in the kinematics of the obstructed swing limb during a trip for **Recovery Strategy 1** (N = 7) and **Recovery Strategy 2** (N = 2).

| | STRATEGY 1 | STRATEGY 2 |
|---|---|---|
| Lengthened swing phase (Time) | Yes | No |
| Shortened swing phase (Time) | No | Yes |
| Delayed heel strike (Time) | Yes | No |
| Early heel strike (Time) | No | Yes |
| Shortened step length (Distance) | Yes | Yes |
| Flat foot landing | No | Yes |
| More flexed knee on landing | Yes | Yes |

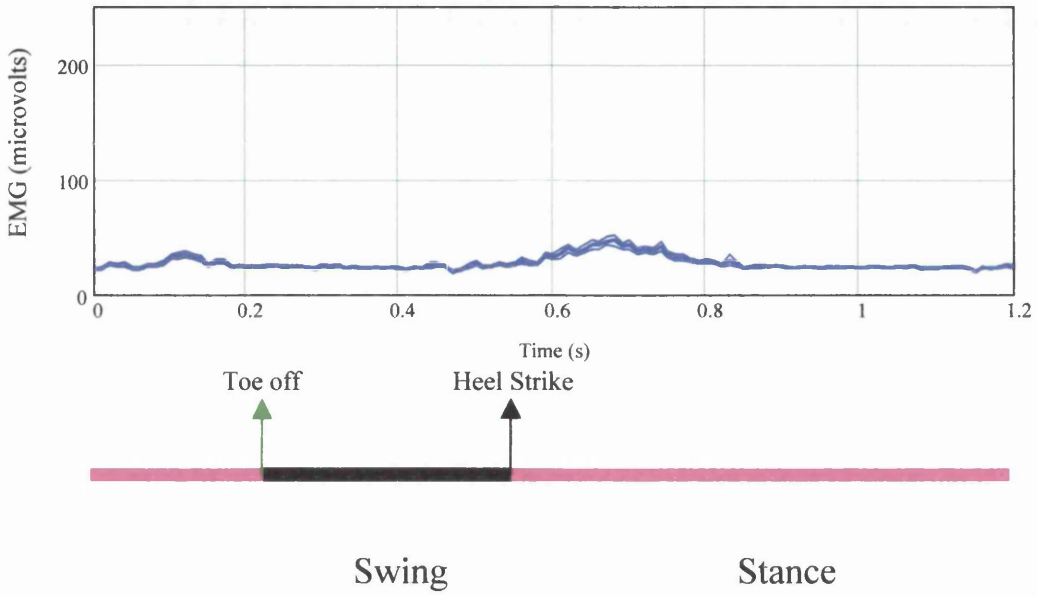
*EMG activity patterns*

The gait perturbation produced changes in the EMG of all four muscles studied, both in the obstructed swing limb (right) and the contralateral stance limb (left). The mean latencies of the responses to the 240 ms perturbation for the two subject groups (**Recovery Strategy 1** and **2**) are detailed below and are summarised in Table 3.4.3. The results for individual subjects and for the 120 ms and 180 ms perturbations are presented in Appendix B. Figures 3.4.4 to 3.4.11 illustrate for each muscle group the activity recorded following a 240 ms perturbation and during normal walking in a representative subject. In Figure 3.4.4 the mean and S.E.M. of the response to 5 trips is shown but for the remaining examples only the mean is shown for clarity.

*Quadriceps*

The perturbation was applied during a period of low activity for the both the right and left quadriceps. Successful recordings were obtained from the right quadriceps of 7 subjects. In response to the trip there was an activation of the right quadriceps in 6 of the subjects. Figure 3.4.4 illustrates the response observed in Subject 3 following a 240 ms perturbation. The mean latency of the response for the subjects using **Recovery Strategy 1** was 108 ± 19 ms (mean ± S.E.M, N = 4). The mean latency of the response for the two subjects using **Recovery Strategy 2** was similar (110 ± 20 ms). In the left quadriceps, the response was more variable among the subjects. For those subjects using **Recovery Strategy 1** a small activation was observed following the perturbation in 3 out of 4 subjects. An example of the pattern of activity recorded for one of these subjects (Subject 4) is illustrated in Figure 3.4.5. The mean latency of the response elicited was 117 ± 30 ms (N = 3). An activation of the left quadriceps was observed in only one of the subjects using **Recovery Strategy 2** (Subject 7). The magnitude of the response in Subject 7 was approximately four-fold greater than that observed for Subjects 2,4 and 8 but the latency of the response was similar (100 ms).

A) Unperturbed Stride (mean and S.E.M. of 84 strides)




B) Perturbed Stride (mean and S.E.M. of 5 trips)

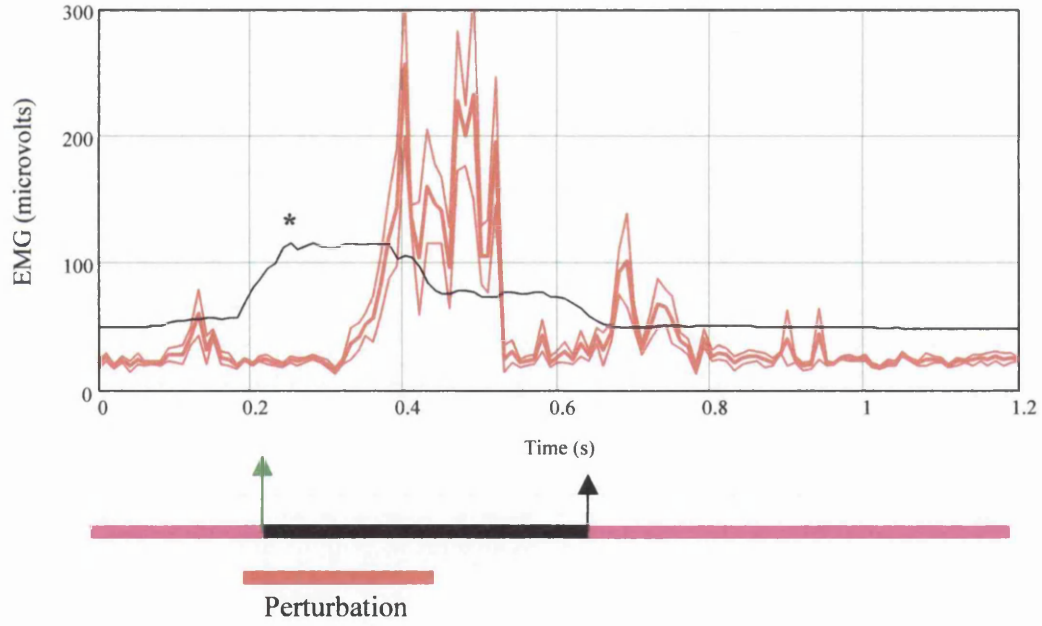
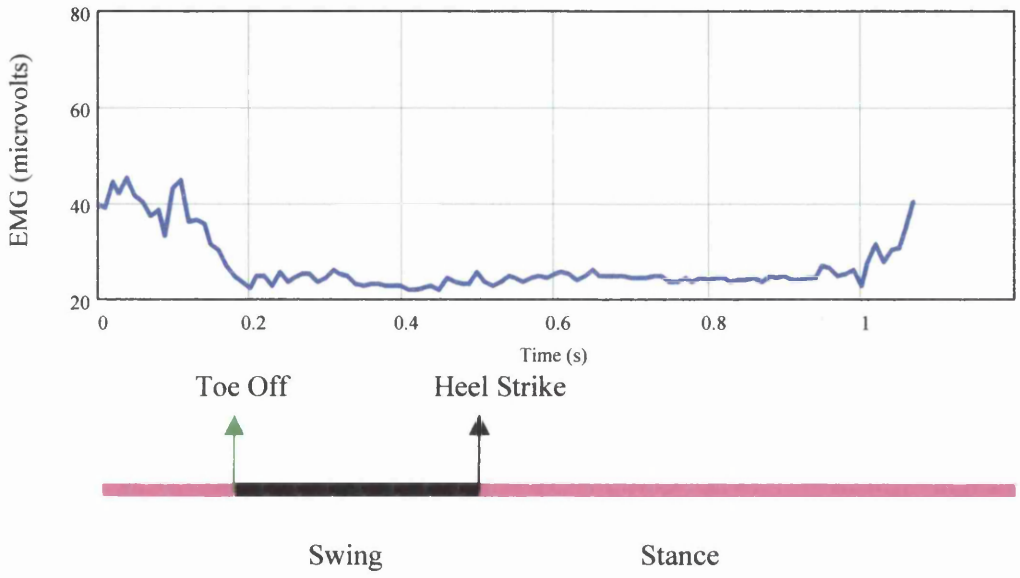



**Figure 3.4.4:** Muscular activity recorded from the **right quadriceps** (obstructed limb) following a 240 ms perturbation. The results are for a single individual (Subject 3) using **Recovery Strategy 1**. A) Normal walking, Blue trace represents the mean and S.E.M. of 84 strides B) Perturbed stride, showing the longer swing phase of the obstructed limb. Red traces are the Mean ± S.E.M of 5 trips, Black trace is the force experienced at the ankle with the baseline shifted by 50 units. From calibration curve, 50 Coda units equals 3.14 N. * Depicts the time from which the latency of the muscular response was measured.

A) Unperturbed Stride (mean of 84 strides)




B) Perturbed Stride (mean of 5 trips)

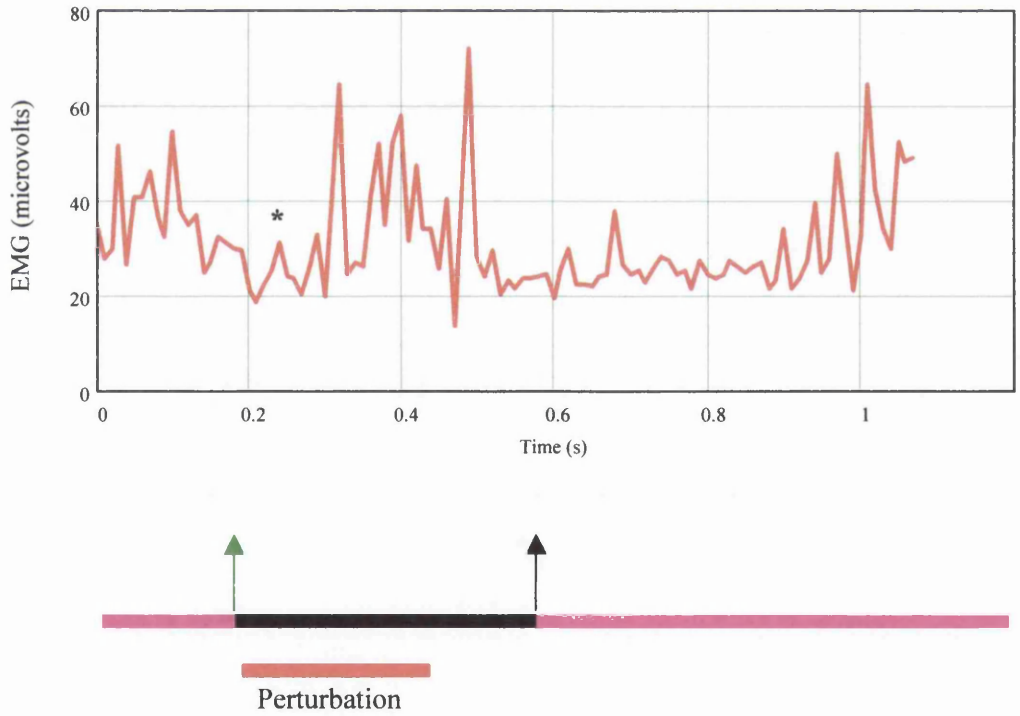



**Figure 3.4.5:** Muscular activity recorded from the **left quadriceps** (stance limb) following a 240 ms perturbation. The results are for a single individual (Subject 4) using **Recovery Strategy 1.** A) Normal walking, B) Perturbed stride, showing the longer swing phase of the obstructed swing limb. * Indicates the time point from which the latency of the muscular response was measured, determined from the force record.

*Hamstrings*

The perturbation was applied approximately 150 ms before activity would normally be observed in the right hamstrings and during a period of low activity for the left hamstrings. Successful recordings were obtained from all 9 subjects on the right side and from 6 subjects on the left side. Following the perturbation there was an *inhibition* of activity in the right hamstrings in subjects using **Recovery Strategy 1**. An example of this response in Subject 8 is shown in Figure 3.4.6. The inhibition is apparent at the start of when normal activity occurs in the muscle so the mean latency of the response is no more than 150 ms. For the two subjects using **Recovery Strategy 2,** no inhibition of activity was observed, rather there was an activation of the hamstrings and the latency of the response was 100 ± 10 ms (mean ± S.E.M., N = 2).

There was an activation of the left hamstrings following the perturbation recorded in 4 subjects using **Recovery Strategy 1**. The mean latency of the response was 63 ± 6 ms (N = 4). An example of the response in one of these 4 subjects (Subject 8) is illustrated in Figure 3.4.7. The activation of the hamstrings prior to heel strike appeared to be slightly delayed in perturbed strides suggesting there was a lengthening of the swing phase in the contralateral limb in these subjects. For the subjects using **Recovery Strategy 2,** a response to the perturbation was only observed in Subject 9. The latency of the response to a 240 ms perturbation was 90 ms. For Subjects 7 and 9 an early onset of the normal muscle activation prior to heel contact was observed. This suggests a shorter swing phase for the contralateral limb following the perturbation.

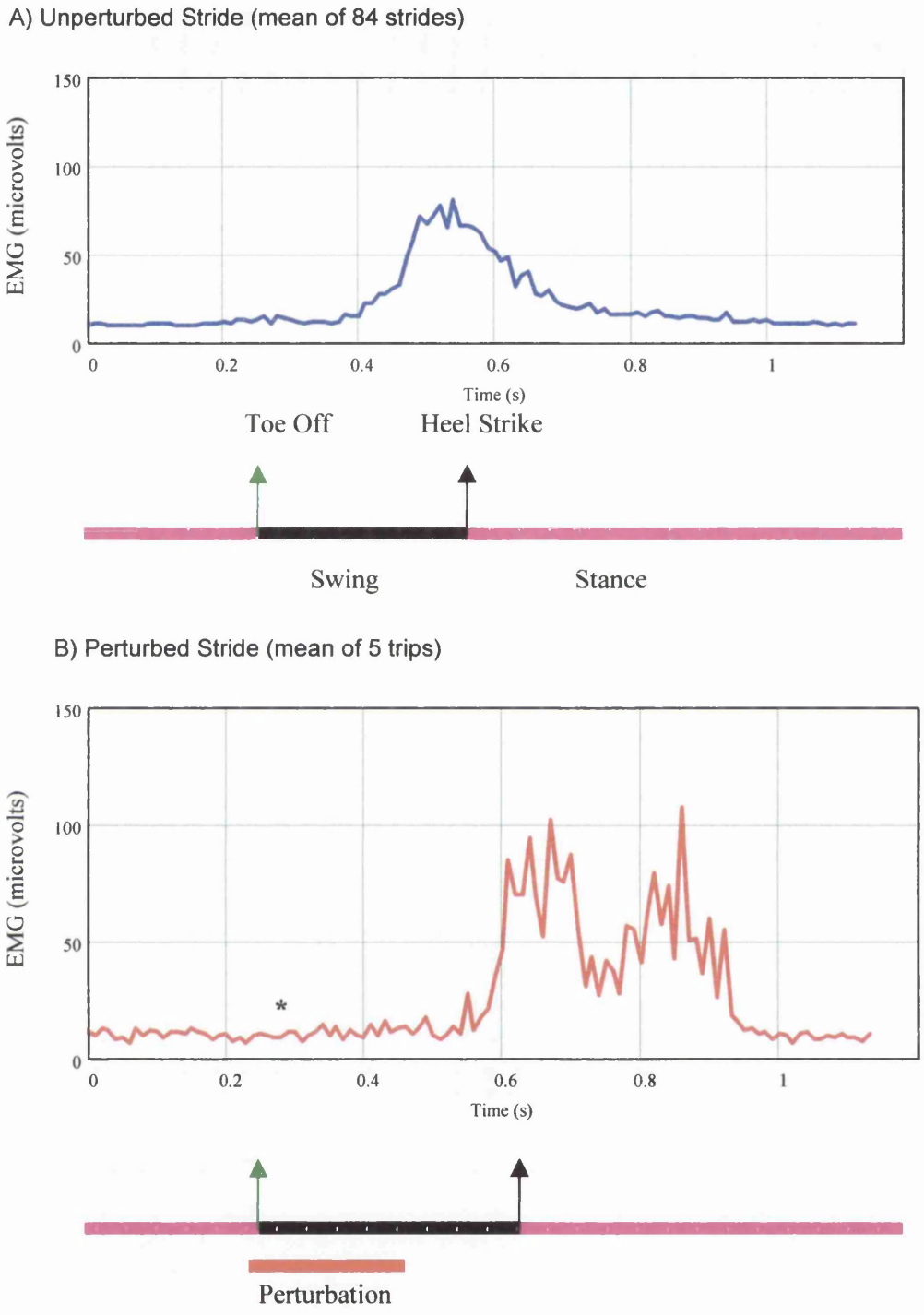



**Figure 3.4.6:** Muscular activity recorded from the **right hamstrings** (obstructed limb) following a 240 ms perturbation. The results are for a single individual (Subject 8) using **Recovery Strategy 1.** A) Normal walking, B) Perturbed stride, showing the longer swing phase of the obstructed swing limb. Note that there is inhibition of normal hamstrings activity following a trip. * Indicates the time point from which the latency of the muscular response was measured, determined from the force record.

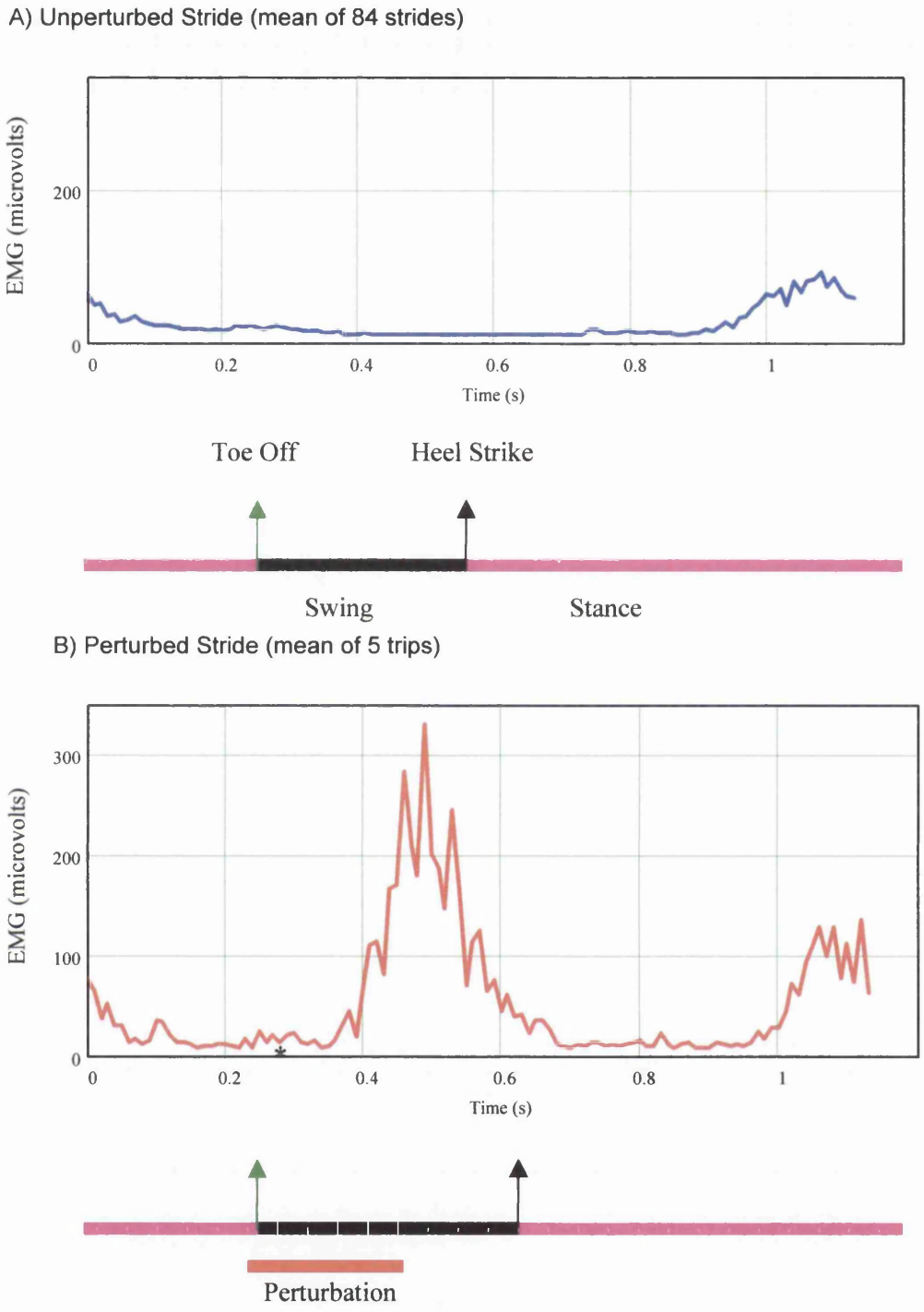



**Figure 3.4.7:** Muscular activity recorded from the **left hamstrings** (stance limb) following a 240 ms perturbation. The results are for a single individual (Subject 8) using **Recovery Strategy 1.** A) Normal walking, B) Perturbed stride, showing the longer swing phase of the obstructed swing limb. * Indicates the time point from which the latency of the muscular response was measured, determined from the force record.

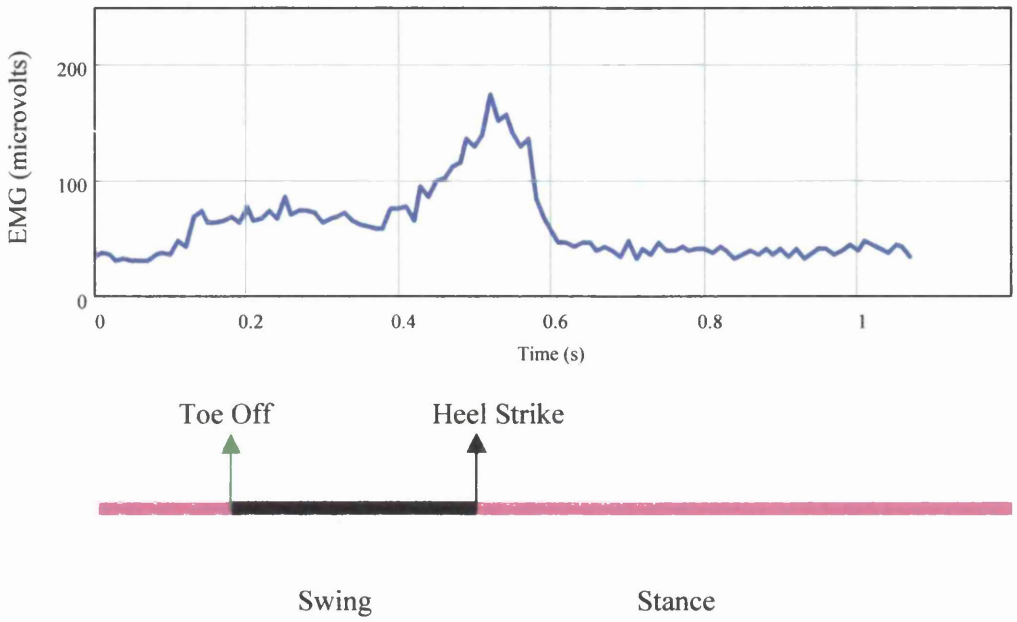
*Tibialis Anterior*

The right TA was active at the time the perturbation was applied (toe off), and there was increased activation of the muscle in response to the perturbation in all 8 subjects for whom recordings were obtained. Figure 3.4.8 illustrates the response recorded for Subject 4. The mean latency of the response for subjects using **Recovery Strategy 1** was 63 ± 5 ms (N = 6). A similar response latency was observed for the two subjects using **Recovery Strategy 2** (65 ± 5 ms). For the left TA, the perturbation was applied during a period of fairly low activity. The general response to the perturbation was an increase in muscle activation. An example of the response in Subject 3 (**Recovery Strategy 1**) is illustrated in Figure 3.4.9. The mean latency of the response was 97 ± 13 ms (N = 5). The duration of the activation was quite variable among subjects and in several cases continued for the remainder of the stride. For **Recovery Strategy 2** subjects, the latency of the response was 105 ± 5 ms.

*Gastrocnemius*

The perturbation was applied during a period of low activity in the right MGa. Successful recordings were obtained from 8 subjects. In 3 out of the 6 subjects using **Recovery Strategy 1** a small response with a mean latency of 70 ± 10 ms was observed. There was no obvious change in activity for the remaining three subjects. In one of the subjects using **Recovery Strategy 2,** (Subject 7) a distinct response with a latency of 40 ms was observed. Figure 3.4.10 illustrates the pattern of muscle activity recorded from Subject 7 following a 240 ms perturbation. The left MGa was active at the time the perturbation was applied and in 6 out of 8 subjects there was increased muscle activation following the perturbation. The mean latency at which the activity in perturbed strides exceeded that in non-perturbed strides for the 4 subjects using **Recovery Strategy 1** was 70 ± 15 ms. Figure 3.4.11 illustrates the responses elicited in the left limb of Subject 3. For subjects using **Recovery Strategy 2** the latency of the response was similar (60 ± 20 ms, N = 2) but the duration of muscle activation was shorter in perturbed strides compared to normal walking.

A) Unperturbed Stride (mean of 84 strides)




B) Perturbed Stride (mean of 5 trips)

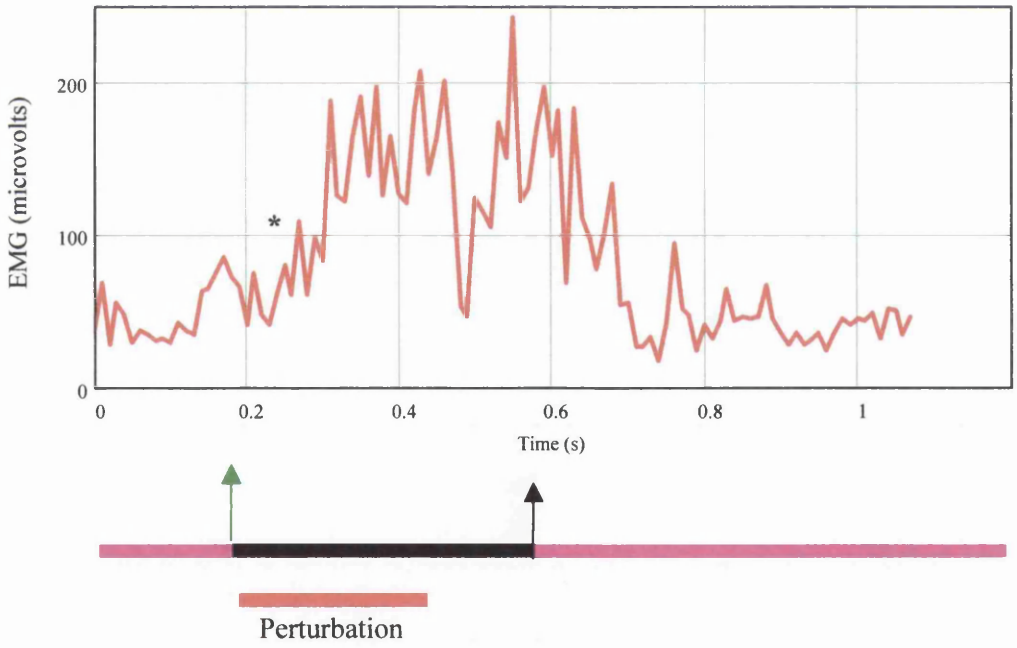
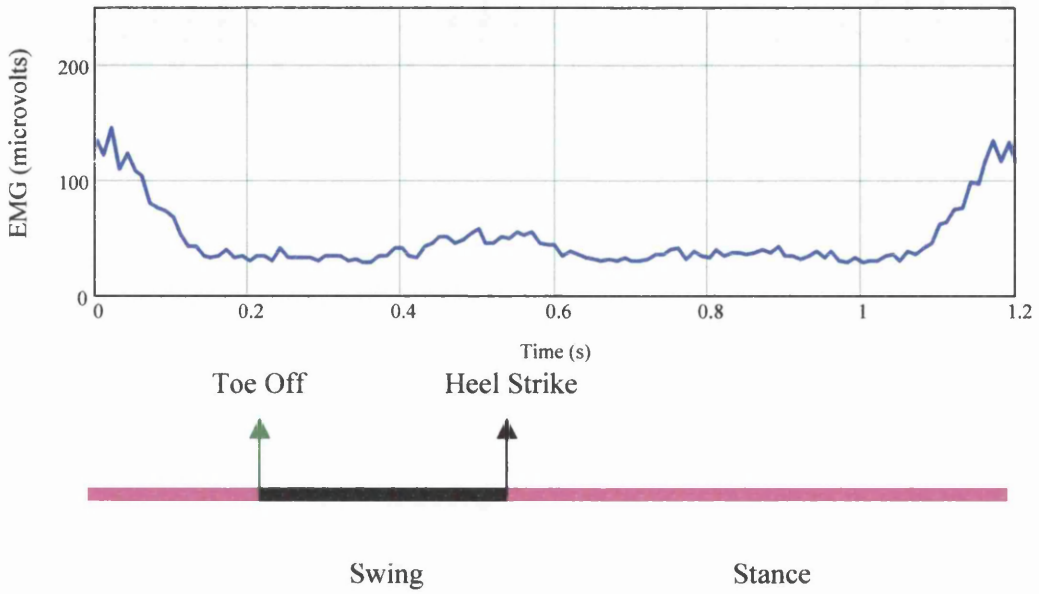



**Figure 3.4.8:** Muscular activity recorded from the **right Tibialis Anterior** (obstructed limb) following a 240 ms perturbation. The results are for a single individual (Subject 4) using **Recovery Strategy 1.** A) Normal walking, B) Perturbed stride, showing the longer swing phase of the obstructed swing limb. * Indicates the time point from which the latency of the muscular response was measured, determined from the force trace.

A) Unperturbed Stride (mean of 84 strides)




B) Perturbed Stride (mean of 5 trips)

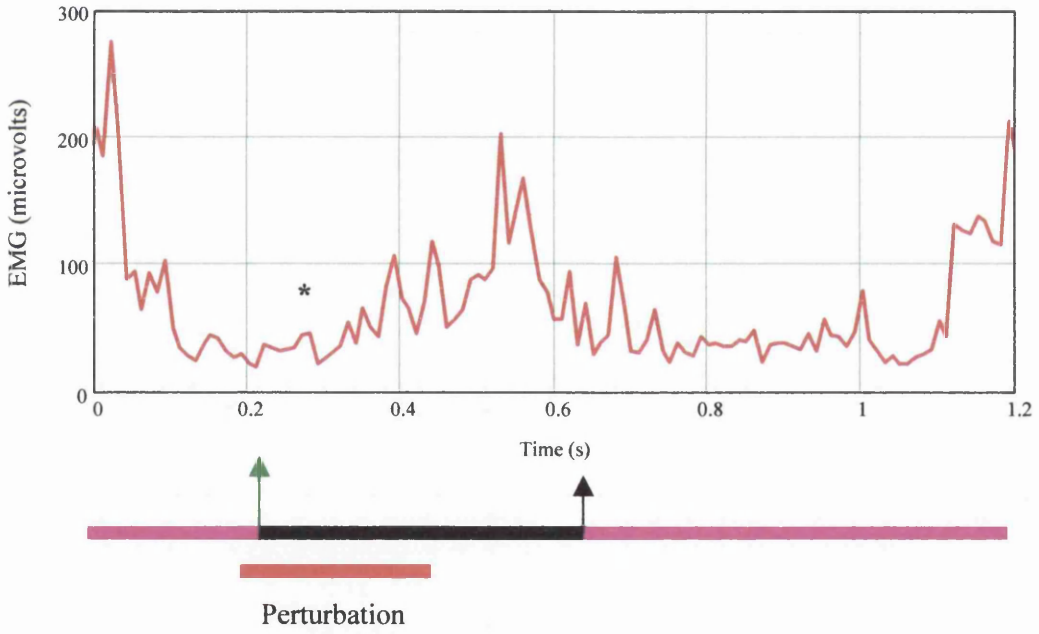



**Figure 3.4.9:** Muscular activity recorded from the **left Tibialis Anterior** (stance limb) following a 240 ms perturbation. The results are for a single individual (Subject 3) using **Recovery Strategy 1**. A) Normal walking, B) Perturbed stride, showing the longer swing phase of the obstructed limb. * Indicates the time point from which the latency of the muscular response was measured, determined from force record.

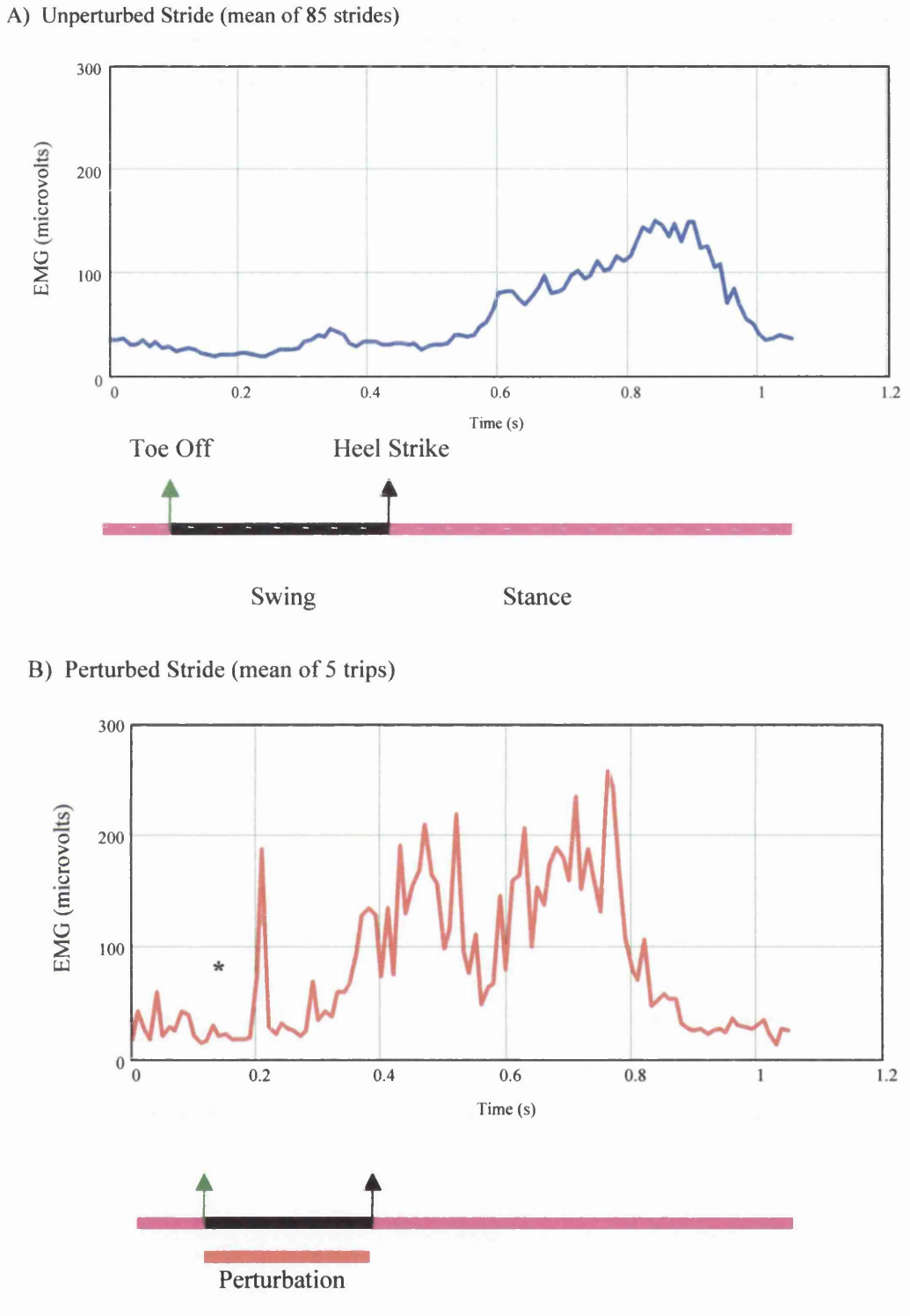
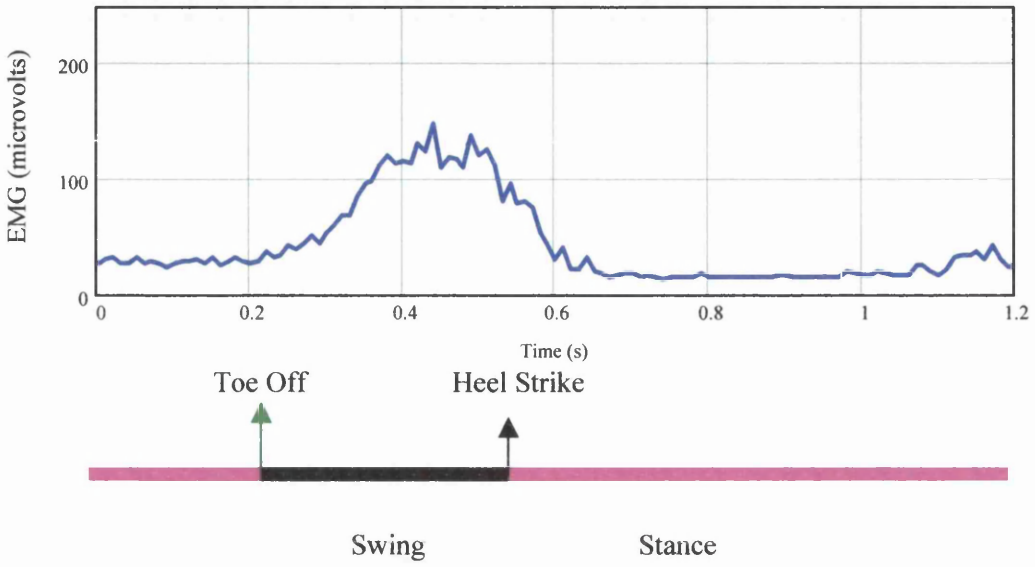



**Figure 3.4.10:** Muscular activity recorded from the **right medial gastrocnemius** (obstructed limb) following a 240 ms perturbation. The results are for a single individual (Subject 7) using **Recovery Strategy 2.** A) Normal walking, B) Perturbed stride, note the slightly shorter swing phase of the obstructed swing limb. * Indicates the time point from which the latency of the muscular response was measured, determined from force record.

A) Unperturbed Stride (mean of 84 strides)




B) Perturbed Stride (mean of 5 trips)

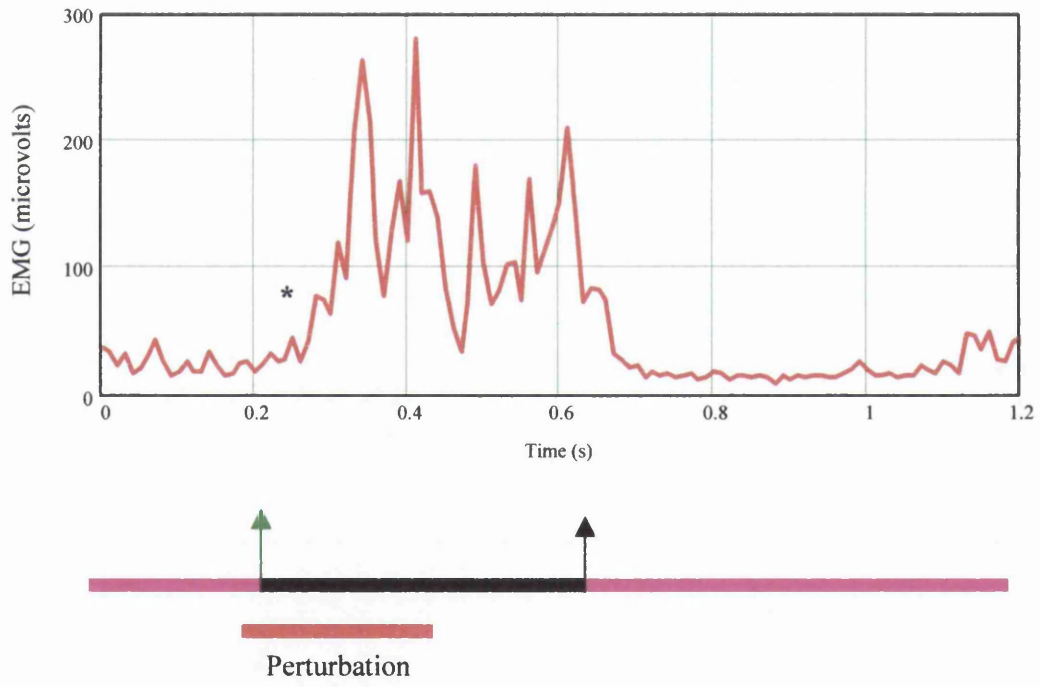



**Figure 3.4.11:** Muscular activity recorded from the **left medial gastrocnemius** (stance limb) following a 240 ms perturbation. The results are for a single individual (Subject 3) using **Recovery Strategy 1**. A) Normal walking, B) Perturbed stride, showing the longer swing phase of the obstructed limb. * Indicates the time from which the latency of the muscular response was measured, determined from force record.

*Summary of EMG responses*

For those subjects using **Recovery Strategy 1** the earliest responses (62 - 70 ms) were observed in the TA anterior of the right (obstructed) limb, the hamstrings of the left limb and the MGa bilaterally. Slightly later responses (97 - 117 ms) were observed in the quadriceps bilaterally and in the TA anterior of the left limb. In contrast to the activation observed in the muscles mentioned above, an inhibition of the normal activity was observed in the hamstrings of the right limb. A diagrammatic representation of the responses observed in subjects using **Recovery Strategy 1** is presented in Figure 3.4.12.

In subjects using **Recovery Strategy 2** the earliest responses were observed in the TA of the right limb (65 ± 5 ms) and the MGa of the left limb (60 ± 20 ms). In one of the subjects there was also a response with a shorter latency (40 ms) observed in the MGa of the right limb. Responses with latencies of around 100 ms were observed in the quadriceps and hamstrings of the right limb and the TA of the left limb in both subjects. Note that the response observed in the hamstrings in these subjects was an *activation* rather than an inhibition. Responses with latencies of approximately 100 ms were also recorded in the quadriceps of the left limb in one subject and the hamstrings of the left limb in the other subject.

A summary of the EMG responses recorded bilaterally for both Recovery Strategies following a 240 ms perturbation is presented in Table 3.4.3.

**Table 3.4.3:** Mean (± SEM) latency of the EMG responses recorded bilaterally after a 240 ms perturbation. Latency was measured from the time at which peak force was recorded at the ankle. * Inhibition

| | | STRATEGY 1 | | STRATEGY 2 | |
|---|---|---|---|---|---|
| Muscle | Limb | Latency (ms) (Mean ± SEM.) | N | Latency (ms) (Mean ± SEM.) | N |
| Quadriceps | Right | 108 ± 19 | 4 | 110 ± 10 | 2 |
| Quadriceps | Left | 117 ± 30 | 6 | 100 | 1 |
| Hamstrings | Right | *133 ± 13 | 7 | 100 ± 10 | 2 |
| Hamstrings | Left | 62 ± 6 | 4 | 90 | 1 |
| Tibialis Anterior | Right | 63 ± 5 | 6 | 65 ± 5 | 2 |
| Tibialis Anterior | Left | 97 ± 12 | 6 | 105 ± 5 | 2 |
| Medial Gastrocnemius | Right | 70 ± 10 | 3 | 40 | 1 |
| Medial Gastrocnemius | Left | 70 ± 15 | 4 | 60 ± 20 | 2 |

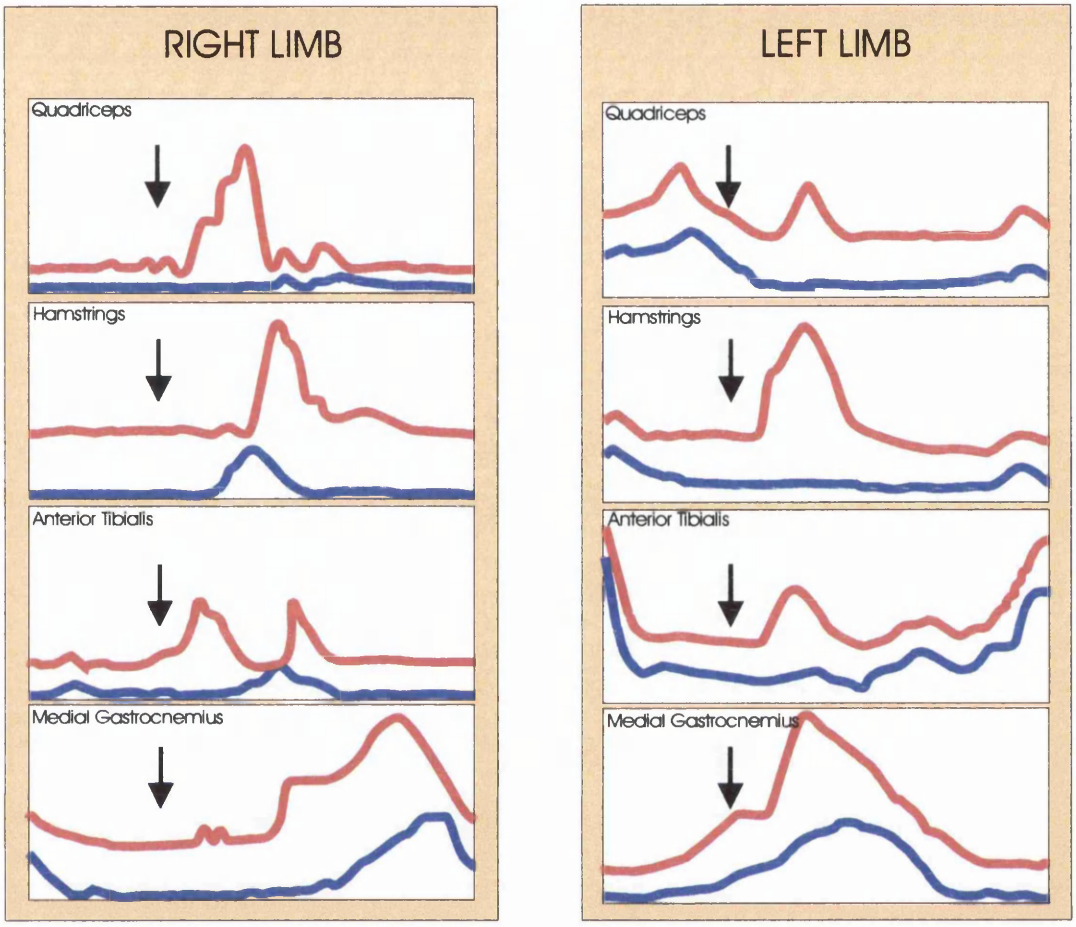



**Figure 3.4.12:** Summary diagram of the muscular responses observed bilaterally in **Recovery Strategy 1** subjects following a trip. The perturbation was applied to the right limb at toe off. The blue traces represent the activity in the muscle during normal walking and the red traces the pattern of activity observed in response to a trip. In the majority of the muscle groups the response was an activation of the muscle. In the hamstrings of the right (perturbed) limb there was an inhibition of the normal pattern of activity. The traces begin 50 ms after heel contact and the black arrow indicates the start of the perturbation.

# *Chapter 4: Discussion*

## 4.1 Postural stability during standing

The synchronous measurement of body movements and torque exerted on a force plate confirmed that the movements of the body during quiet stance could be likened to those of an inverted-pendulum as described theoretically by Gurfinkel (1973). The body can be represented by a point mass at a constant distance from a universal joint and it's position at any moment can be described by two angles $\theta_1$ and $\theta_2$ in the A/P and M/L axes respectively. (See figure 4.1.1 below).

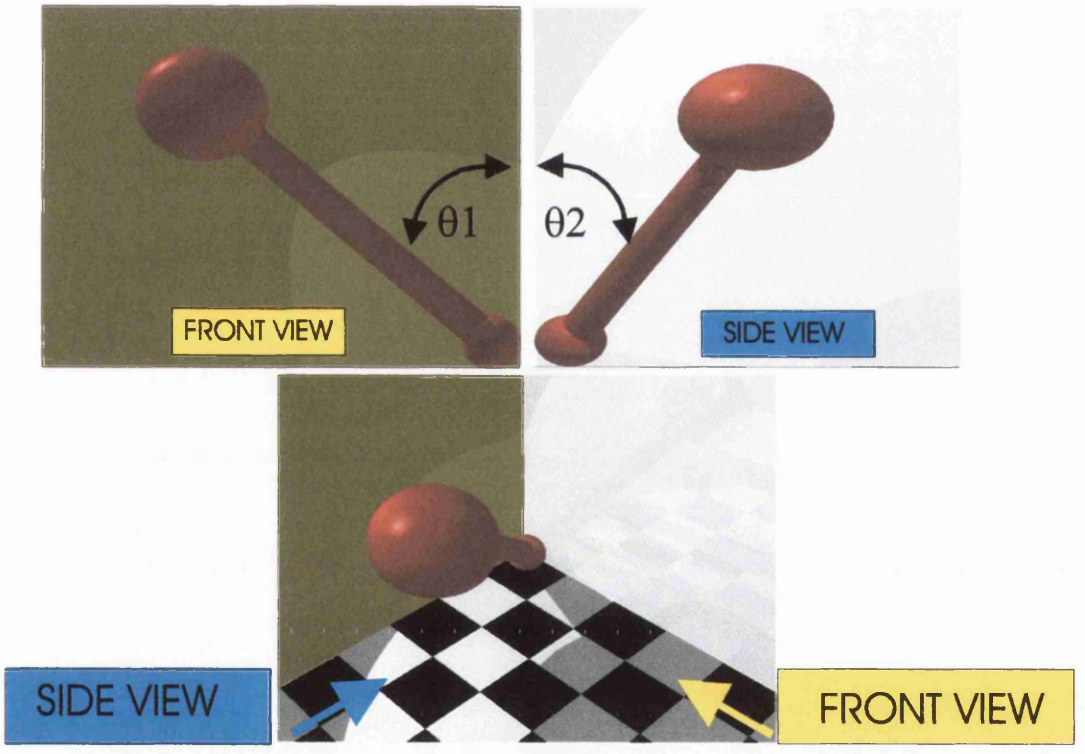



**Figure 4.1.1:** The inverted pendulum model of body sway during quiet stance. The body is represented by a point mass a constant distance from a universal joint and its position at any moment can be described by two angles $\theta_1$ and $\theta_2$ in the A/P and M/L axes respectively.

***The validity of the inverted-pendulum model of quiet stance for young subjects***

*Correlation of the marker movement*

The results of Chapter 3.1 showed that, for young subjects standing quietly with the eyes closed and their feet approximately 10 cm apart, there was a large positive correlation (above 0.9) between pelvis and shoulder marker movement in both the A/P and M/L axes. Large positive correlation was also found between the movement of other marker combinations such as pelvis-head and shoulder-knee. A positive correlation between pelvis and shoulder markers does not guarantee that the body is behaving as a single-link inverted pendulum. Bending at the pelvis may also produce a positive correlation if the pelvis and shoulders are both moving forward but the shoulders are moving faster (Stoffregen, Adolph, Thelen, Gorday & Sheng, 1997). Such a situation may arise just prior to a re-balancing step being taken to prevent a fall. A positive correlation may also arise if the body was behaving in a whip-like fashion with the shoulders following the movement of the pelvis with a time delay. However, for the young subjects measured in this experiment, a cross-correlation analysis showed that there was no phase lag between the body segments and all the segments were moving in the same direction at the same time.

Day *et al.* (1993) has suggested that the inverted pendulum model of body sway is incomplete. They found that in both the A/P and M/L axis, body markers situated furthest from the ground moved faster and further than the markers closer to the ground. However analysis of the amplitude of angular rotation between adjacent body segments revealed that in the A/P axis, the greatest angular fluctuations (standard deviation of the angle) occurred between the trunk and leg and not at the ankle. Similar results were observed in the M/L axis for stance widths of 16 and 24 cm. However, when the feet were closer together (< 8 cm apart) angular motion about the ankle dominated, i.e. the body behaved as an inverted pendulum. The mean stance width of the subjects described in Chapter 3.1 was 10 cm, close to the 8-cm separation where Day *et al.* (1993) observed similar angular fluctuations for the

trunk and ankle angle. A given rotation about the ankle will produce greater movement of the top of the body compared to that produced by the same angular rotation about the pelvis, as Day *et al.* (1993) themselves noted. Therefore even when subjects are permitted to adopt a 'comfortable stance width' the dominant motion of the body will be a rotation about the ankles in both the A/P and M/L axes.

*The relation of marker height to marker movement*

The marker movement data showed that the body segments furthest from the ground (such as the head) move the greatest distance from their mean position, as would be predicted from the inverted-pendulum model (Gurfinkel, 1973; Day *et al.* 1993). For each point in time, a linear regression line can approximate the relationship between the height of the markers on the body and the movement of the markers in the A/P and M/L axes. The slope of this regression line is an estimate of the angle of body sway. Therefore, a record of changes in the slope over the 60-second trial is effectively an inverted-pendulum model of the body's movements during this time period.

*The relation of COP movement to angular position of the model*

The COP movement record showed a large positive correlation with the model record in both A/P (0.95) and M/L (0.91) axes. The linear regression of COP movement variance upon model movement variance also showed a strong association between movements of the COP and movements of the model. In addition, the regression coefficient calculated for the linear regression of COP movement upon the model was shown to be a close approximation for the subjects' estimated COG height. Thus, a strong association exists between the position of the model and the movement of the COP, and the model is a close approximation of the position of the body's COG during quiet stance. Other investigators have used body segment parameters to estimate the position of the body's COG and have also found that a

close relationship exists between motions of the COP and the COG (Murray, Seirig & Sepic, 1975; Ruder, MacKinnon & Winter,. 1989; Benda *et al.* 1994; Hasan *et al.* 1996).

*Some of the high frequency components present in the COP record can be explained by angular acceleration of the model in the A/P and M/L axis*

It is well recognised that although a strong correlation exists between movement of the COP and movement of the COG the two are not identical (Gurfinkel, 1973; Murray *et al.* 1975; Ruder *et al.* 1989, Benda *et al.* 1994). The excursions of the COP are larger in amplitude and tend to oscillate either side of the COG position. Records of COP movement also contain higher frequency components not observed in records of COG movement. When the body is modelled as an inverted pendulum the product of body weight and the difference between the COP and COG is the controlling torque at the ankle (Ruder *et al.* 1989). This net torque is equal to the product of the body's moment of inertia and the angular acceleration of the body (Newton's second law). The record of angular position of the model was differentiated twice to produce a record of model angular acceleration. This angular acceleration record was correlated with the record of residual torque, obtained by multiplying the residual movement (COP - COG) by the subjects body weight (N). Although the correlation between the two records was low (0.31 (A/P) and 0.34 (M/L)) it was significantly different from zero. The rather modest correlation is not surprising given that the differentiation process used to obtain the angular acceleration tends to amplify the high frequency signal noise in the angular position data and the angular position of the model is only an approximation for the position of the body's COG. In addition, the mean spectral frequency of the COP record in the A/P and M/L axes was around 0.2 Hz. Gurfinkel (1973) estimated that at frequencies of 0.2 Hz the difference between the COP and COG movement was less than 10% and therefore inertial forces would only make a small contribution to the COP movement. If the residual torque itself is small in magnitude and the error associated with the acceleration record is relatively large, only a fairly modest

correlation between the two records should be expected. Winter *et al* (1997) correlated the residual movement (COP - COG) with the horizontal acceleration of the COG (based on body segment parameters) and found high correlation in the A/P direction (above 0.91) and in the M/L direction (above 0.82) for three different stance widths. The strength of the association is quite surprising considering the relatively small difference between movements of the COP and COG during quiet stance and the potentially large errors associated with estimating the position of an individual's COG from anthropometric parameters.

An estimate for the body's moment of inertia was obtained by regression analysis of the residual torque and angular acceleration records. This estimate of the body's moment of inertia was shown to be comparable to one calculated from anthropometric parameters. When records of residual torque were overlaid with records obtained by multiplying the angular acceleration by the estimate of the body's moment of inertia it could be seen that many of the high frequency components present in the residual torque record were explained by angular acceleration of the model. The torque remaining once the inertial forces had been taken into account reflected some respiratory and cardiovascular influences. Records of torque about the ankle are sensitive to heart rate (Thomas & Whitney, 1959; Goldie, Bach & Evans, 1989) and a respiratory effect has been observed previously in the postural sway of subjects during quiet breathing (Hunter & Kearney, 1981; Bouisset & Duchêne, 1994).

*To what extent are the marker movements explained by the model?*

The movement variance of the 14 markers was compared to the movement variance of the model. The results of a linear regression analysis showed that most of the movements of the markers in the A/P and M/L axes could be explained by shifts in angular position of the inverted pendulum model. For several young subjects large marker movements were observed in the A/P axis and the results showed that these movements were due to large

movement of the model in the same direction. Thus, for young adults, the inverted pendulum model defined in Chapter 3.1 describes well the movements of the body during quiet stance.

***Movements responsible for increased COP excursions in the elderly***

*Elderly subjects make larger COP excursions in the M/L direction*

Elderly subjects were shown to have a significantly greater COP movement variance in the M/L axis than young subjects but no significant difference between the two groups was observed in the A/P axis. Other investigators have reported similar results. Hasselkus and Shambes (1975) reported that young women swayed proportionally more in the A/P plane compared to the M/L plane than elderly women. More recently, Maki *et al.* (1994) found changes in M/L COP measures in elderly subjects classified as 'fallers'. The fall status of 100 elderly volunteers was monitored for a year following a balance testing session. Six sets of balance measures were obtained for each subject including movement of the COP during quiet stance. The results showed that on average, fallers showed increased M/L COP excursions during quiet stance, eyes open, compared to non-fallers. The amplitude of M/L COP movement (RMS) was also shown to be the best predictor of individuals at risk of falling.

*Correlation of the marker movement*

There was a large positive correlation between the pelvis and shoulder marker movement in the elderly group for both the A/P and M/L axes. In the M/L axis the correlation for the elderly (0.96) was significantly greater ($p < 0.001$) than that observed for the young subjects (0.91). This was probably due to the large sway movements of some elderly in this axis producing very high correlation between the pelvis and shoulder. In a few individual trials, short episodes were observed where the pelvis and shoulders were negatively correlated suggesting the use of a hip strategy over this time period. These episodes were also observed

occasionally during individual trials of young subjects. However, for the majority of both young and elderly trials the results suggested that all the segments of the body moved in the same direction at the same time. Therefore movements of the body during quiet stance in elderly subjects could also be likened to that of an inverted pendulum rotating about the ankles.

*Correlation between the movement of the COP and the model*

A large positive correlation ($r > 0.9$) was found between the movement of the COP and the model for the elderly subjects in both the A/P and M/L axes. In the A/P axis, the correlation for the elderly ($r = 0.92$) was significantly less ($p < 0.05$) than that observed for the young subjects ($r = 0.95$). In the M/L axis, the reverse result was found, and higher correlation between the COP and the model was observed for the elderly ($r = 0.95$) compared to the young ($r = 0.91$, $p < 0.01$). These differences between the groups are probably due to a few young subjects making very large inverted-pendulum like movements in the A/P axis while several elderly subjects made relatively large inverted-pendulum-like rotations in the M/L axis. The linear regression of COP variance upon model variance (Figs. 3.1.8a and b) also illustrated this strong association between movement of the COP and the model in both A/P and M/L axes for the elderly. Additionally, for the majority of elderly subjects, the slope of the regression of COP movement upon the model was also a close approximation for the subject's estimated COG height. As the model reflects the movements of the body's COG and there is a strong association between the model record and the COP record, the large M/L COP excursions observed for some elderly subjects must be due to larger inverted-pendulum-like rotations about the ankles.

*To what extent does the model explain the marker movements?*

The movement variance of the 14 markers was compared to the movement variance of the model. The M/L marker and model variance was significantly greater in the elderly group

compared to the young group but there was no difference between the two groups in the A/P axis. The results of a linear regression analysis showed that most of the movements of the body in the A/P and M/L axes in the elderly could be explained by shifts in angular position of the inverted pendulum model. Therefore large movement of the markers in the M/L axis in the elderly is due to large angular movement of the model.

In summary, the results showed that the inverted pendulum model defined in Chapter 3.1 described the movements of the body during quiet stance for both young and elderly subjects thus confirming the original hypothesis that the increased COP excursions observed in some elderly would be due to larger inverted pendulum-like rotations of the body about the ankle. Therefore, the elderly subjects use the same 'ankle strategy' as the young subjects to maintain their balance during quiet stance but the sway movements of their body in the M/L axis are of much larger amplitude.

***Is the model still valid for the elderly considered 'balance impaired'?***

Initially the results of the elderly subjects were considered as a single group, but within the group there were some individuals who were considered to have reduced balance capabilities (as judged by their performance on a number of clinical balance tests) while others had balance capabilities equal to those of young subjects. To test whether the model was still valid for these 'balance impaired' individuals, a subgroup analysis was carried out on the marker and model variance data. Comparisons were made between 'balance impaired' elderly, elderly subjects with 'good balance capabilities' and young subjects. The variance of the marker and the model movement in the M/L axis was significantly greater for the balance-impaired elderly than that observed for the young subjects. Also the marker variance in the balance-impaired subjects was significantly greater than that observed for elderly subjects with good functional balance ability. No differences in marker or model movement variance were observed between the young subjects and the elderly with good

balance capabilities. As there was shown to be a strong association between the marker movement variance and the model movement variance, the increased movement variance observed in the M/L axis in the 'balance impaired' elderly is due to larger inverted pendulum rotations about the ankle in this direction. Therefore the model is valid not only for young subjects and elderly subjects with good balance ability but also for those elderly that appear to have reduced M/L postural control.

***Spectral analysis***

Postural stability during standing is dependent upon the integration of afferent information from the visual, vestibular and somatosensory systems. While there is some redundancy across systems, each works within a certain amplitude and frequency range. Extremely low frequencies of sway (< 0.1 Hz) are best stabilised by vision (Dichgans, Mauritz, Allum & Brandt, 1976; Lestienne, Soechting & Berthoz, 1977). Input from the soles of the feet is also important in stabilising posture at low frequencies in the absence of vision (Okubo, Watanabe & Baron, 1980; Diener, Dichgans, Bacher & Gompf, 1984a; Magnusson, Enbom, Johansson & Wiklund, 1990). The sensory organs of the vestibular system, the semicircular canals and the otolith organs, are thought be effective at sway frequencies above 0.1 Hz and below 0.5 Hz respectively (Nashner, Shupert, Horak & Black, 1989). Proprioception about the ankles has been shown to contribute to postural stability at frequencies above 1 Hz (Diener, Dichgans, Guschlbauer, & Mau, 1984b). In Chapter 3.1 a Fast Fourier Transform analysis (FFT) was used to estimate the frequency composition of the COP movement records and to calculate the power spectra (bandwidth 0.0167 - 4.99 Hz).

*Mean Spectral Frequency*

The mean spectral frequency of the M/L COP movement record was significantly different for young (0.25 Hz) and elderly subjects (0.20 Hz, $p < 0.05$) suggesting a greater concentration of power at low frequencies in the elderly group. No difference in mean

spectral frequency was observed between the young and elderly groups in the A/P direction. McClenaghan, Williams, Dickerson, Dowda, Thombs, & Eleazer (1996) investigated age-related changes in the power spectra of the ground reaction forces obtained from a force plate during quiet stance. They also reported that measures such as mean, median and dominant frequency were significantly lower in the M/L axis in elderly subjects compared to young subjects. Maki *et al.* (1994) compared the mean frequency of COP movements in a group of elderly fallers and non-fallers. In the M/L axis the mean frequency of the COP movement in fallers (0.41 Hz) was lower ($p = 0.04$) than that observed for non-fallers (0.46 Hz). No differences in mean spectral frequency were observed between young and elderly subjects (McClenaghan *et al.* 1996) or fallers and non-fallers (Maki *et al.* 1994) in the A/P direction. McClenaghan, Williams, Dickerson, Dowda, Thombs, & Eleazer (1995) suggested that a lack of difference between the spectral characteristics of older adults and young adults in the A/P axis may be due to the greater number of balance strategies available masking age differences. However, the results of the movement analysis described in Chapter 3.1 suggest that both young and elderly subjects make the low, frequency, inverted-pendulum-like rotations in the A/P axis during standing and the amplitude of the movements is similar in both groups.

*Power Spectra*

Averaging individual spectral signatures generated spectral envelopes for the young and elderly groups. The principal power was contained below 1 Hz for both the A/P and M/L axes, with greater power exhibited by the elderly subjects at frequencies below 0.25 Hz in the M/L axis. The amplitude of the COP movements were calculated for two discrete bandwidths, low frequency (0.0167 - 0.251 Hz) and high frequency (1.01 - 4.99 Hz). In the M/L axis, the amplitude of the COP movement in the low frequency bandwidth was significantly greater for the elderly but no significant difference in amplitude was observed between the young and elderly groups for the high frequency bandwidth. In the A/P axis, no

significant difference was observed between the two groups for either the low or high frequency bandwidths. These results confirmed that at the low frequencies associated with inverted-pendulum sway of the body (< 0.2 Hz), the elderly exhibit increased COP movement in the M/L direction compared to the young subjects.

*Subgroup analysis*

As the group of elderly was not homogeneous with regards to their balance ability, a subgroup analysis of the power spectra parameters was conducted. The results showed that 'balance impaired' elderly subjects who performed poorly on clinical balance tests, including the one-legged stance and tandem gait, exhibited significantly greater COP movement in the M/L axis at low frequencies than young subjects. Elderly subjects with good balance capabilities were indistinguishable from the young subjects. This suggests that the observed differences in the spectral characteristics of M/L COP movement are more related to balance ability than to age. These results are in agreement with those reported by Williams, McClenaghan & Dickerson (1997) comparing the spectral characteristics of postural sway in young adults and elderly individuals classified as 'high' or 'low' risk with regard to functional balance capacity. Their results indicated that 'high' risk elderly individuals were characterised by greater concentration of power at low frequencies in the M/L axis. 'Low' risk elderly were no different from young adults. In the A/P axis, differences in the mean and median frequency between the groups were not significant.

Maki *et al* (1994) suggested that the trend toward lower M/L mean frequency in elderly fallers may indicate a reduced 'stiffness' or postural 'reflex gain' in these subjects due to attenuation of plantar cutaneous (Okubo *et al.* 1980, Magnusson *et al.* 1990) and/or vestibular inputs (Mauritz, Dichgans & Hufschmidt, 1979). Both types of sensory input are capable of stabilising low frequency sway in the absence of vision. The significance of sensory input from the feet to lateral postural control has been demonstrated by Magnusson

*et al.* (1990). Anaesthesia of the feet was induced by immersion in ice-water for 20 minutes and the effects of the anaesthesia on movements of the COP during galvanic vestibular stimulation of the vestibular nerve (eyes closed) were measured in seven young subjects aged 26.4 ± 7.4 years (mean ± SD). The variance of the COP movements increased significantly in both M/L ($p < 0.01$) and A/P ($p < 0.05$) directions with a combined anaesthesia/galvanic stimulus and less adaptation to the galvanic stimulus was observed in the M/L direction when the feet were anaesthetised. In addition, the mean amplitude of the FFT spectrum was significantly higher ($p < 0.01$) in the M/L direction during combined anaesthesia/ stimulation conditions compared to stimulation alone.

It has also been suggested that the 'vestibular system, due to its bilateral organisation, is very well suited to control lateral stability of posture' (Mauritz *et al.* 1979). The clinical balance tests applied in Chapter 3.1 to separate the elderly subjects into either 'balance impaired' or 'good balance ability' subgroups, have been used previously to examine the integrity of the vestibular system (Fregly, Smith & Graybiel, 1973). Thus it is conceivable that impaired vestibular function could have contributed to the excessive lateral sway demonstrated by the 'balance impaired' elderly subjects.

The amplitude of the COP movement calculated for the 'balance-impaired' elderly in the high frequency band (1.0 - 4.99 Hz) was not statistically different from that calculated for young subjects or for the elderly with good balance. However, there was a trend towards larger movements in the 'BI' elderly in both the A/P and M/L axis compared to the young subjects and in the M/L axis compared to the GBA elderly. Somatosensory inputs associated with ankle rotation have been shown to contribute to postural stability at frequencies above 1 Hz (Diener *et al.* 1984b) and these inputs are important in controlling A/P stability (Nashner, 1981). An alteration in the sensitivity of the peripheral nervous system may be partly responsible for the increase in amplitude of the high frequency

components of the A/P COP movement in 'balance impaired' elderly. Similar high frequency instability has been noted, with visual deprivation, in the A/P power spectrum of subjects with peripheral neuropathies and of normal subjects deprived of some proprioceptive inputs through experimentally induced, temporary blocking of group I afferents (Mauritz & Dietz, 1980; Mauritz, Dietz & Haller, 1980; Diener *et al.* 1984b). Proprioceptors located in the muscles and joints of the hips may also play a role in controlling lateral sway as suggested by Day *et al.* (1993). Combined proprioceptive inputs from the ankles and hips will increase the sensitivity to lateral motion and provide more accurate information for central control of body movements. Hip fracture patients have been shown to have increased A/P and M/L COP movement and a larger sway area when standing blindfolded. With the eyes open only A/P sway was significantly different from the controls (Jarnlo & Thorngren, 1991). This suggests that vision is capable of providing the necessary sensory input to stabilise lateral sway but in the absence of vision the subject must rely on vestibular and somatosensory inputs, one or both of which may be impaired.

High frequency instability around 1 Hz and 3 Hz in the A/P axis was also observed in one elderly subject with severe balance impairment. Peripheral neuropathy is a rare but recognised side effect of Amiodarone, the medication prescribed for tachycardia for this subject and could be responsible for the increase in amplitude of the A/P COP movements around 1 Hz. in this subject. The distinct 3 Hz postural tremor also observed may have been due to an undiagnosed neurological condition. Patients with cerebellar anterior lobe atrophy show a spontaneous high frequency body tremor around 3 Hz (Diener *et al.* 1984a, Lucy & Hayes, 1985).

***Stabilogram-diffusion Analysis***

*Stabilogram-diffusion* plots were computed from the COP, the model and the residual (COP - COG) movement records. The results showed that the shape of the COP *stabilogram-diffusion* plot was very dependent on the relative contributions of the static and dynamic components of postural sway. The static component reflects movement of the COG and is therefore represented by the *stabilogram-diffusion* plot computed from the model. The dynamic components (inertial forces) present in the COP movement records are reflected by the *stabilogram-diffusion* plot of the residual record COP-COG. If the contribution from inertial forces was relatively small than the *stabilogram-diffusion plots* for the COP and model were very similar as were the values obtained for $D_{js}$, the slope of the short-term region of the plot. When the inertial forces were relatively large then the COP record had a much steeper short-term slope, and there was a more distinct transition between short and long-term regions of the plot.

In Chapter 3.1, no significant age-related changes were observed for values of the short-term slope obtained from the COP, model and residual plots in either the A/P or M/L direction. These results are in contrast to those reported by Collins, DeLuca, Burrows & Lipsitz (1995) when they used *stabilogram-diffusion* analysis to investigate age-related changes in COP trajectories. Collins *et al* (1995) found that the mean short term slope calculated for 25 elderly subjects (age: 71-80 years, mean 75 years) was substantially greater than that calculated for the young subjects (age: 19-30 years, mean 22 years) in both the A/P ($p < 0.005$) and M/L ($p < 0.05$) direction. When the 25 elderly subjects were classified as 'healthy elderly' or elderly 'at risk for falling' according to their health status the mean value calculated for A/P $D_{js}$ for 'at risk' elderly was significantly larger than that of both the young subjects ($p < 0.005$) and healthy elderly ($p < 0.05$). No significant difference was observed between 'at risk' elderly and the young adults or healthy elderly for the mean value of the $D_{js}$ in the M/L axis.

In Chapter 3.1 an observation was made that some of the elderly subjects that had large values for the A/P $D_{js}$ computed from the COP record also had increased values for the $D_{js}$ computed from residual record. This suggests that inertial forces make a substantial contribution to the large COP values obtained for these subjects. A few young subjects and elderly subjects with good balance capabilities also had large values for $D_{js}$ of the COP *stabilogram-diffusion* plot. However, the movements in the sagittal plane in the these subjects were of a low frequency and were due to large inverted pendulum rotations about the ankles which was reflected by the large value for $D_{js}$ calculated from the *stabilogram-diffusion* plot of the model.

Analysis of the COP, model and residual records suggest that the interpretation of the *stabilogram-diffusion* plots put forward by Collins and DeLuca (1993) is incorrect. They suggested that over short time periods the sway is uncontrolled and open-loop control schemes are in operation. Once a 'systematic threshold' is reached closed-loop or feedback mechanisms are called upon. The critical time point on a *stabilogram-diffusion* plot quantifies the time interval and COP displacement at which the control is switched from open-loop to closed-loop. The results of Collins *et al.* (1995) were interpreted as the elderly utilising open-loop control schemes for longer time intervals and correspondingly larger COP displacements during quiet stance. It may well be true that in the elderly there is a delay before feedback mechanisms are called into play but the parameters extracted from the *stabilogram-diffusion* plot do not provide this information. The results from 3.1 demonstrate that the short-term slope and critical time point are influenced by the contribution of inertial forces to the COP record while the long-term slope reflects the movement of the COG. In addition, as it is possible for two subjects to have large values for the short-term $D_{js}$ but for the nature of their postural sway to be very different the usefulness of *stabilogram-diffusion* analysis for investigating age-related changes in COP excursions must be questioned.

## 4.2 *Postural stability during walking*

The variability of the movements of the body from its mean path during walking has not previously been used to compare postural stability in young adults and older adults. Variability in spatial-temporal parameters of gait have been measured and were shown to be indicators of prospective (Lord *et al.* 1996; Maki, 1997) or retrospective falling (Hausdorff *et al.* 1997), neurological deficit (Blin *et al.* 1990), central cardiovascular disease and/or peripheral weakness (Hausdorff *et al.* 1994) in older adults. However, the spatial-temporal parameters of gait are probably not sensitive enough to identify subtle differences in stability that may exist in a population of fairly active, community dwelling elderly (Gabell & Nayak, 1984; Heitman *et al.* 1989).

The results of Chapter 3.2 demonstrated that the movements of the body from its mean path during walking can be described by an inverted-pendulum model similar to that defined for movements of the body away from its mean position during quiet stance. Although the magnitude of the movements during walking is much greater than for standing in both the A/P and M/L axes the variance of the inverted pendulum movements can be used as a quantitative measure to investigate the relationship between walking and standing balance. Previously, comparisons of balance during walking and standing in elderly subjects have typically used COP measures to quantify standing balance and qualitative or semi-qualitative measures such as the Tinetti Mobility Index (Tinetti, 1986) as an estimate of balance during walking (Imms & Edholm, 1981; Lichtenstein, Burger, Shields, & Shiavi, 1990; Topper & Holliday, 1993).

*Deviations from the mean path during walking*

Walking consists of an alternate receiving of the body weight first by one foot and leg, then by the other as each in turn is swung forward and placed on the ground again to form a new

base of support. This pattern of alteration is a fundamental part of walking and formed the basis of the body movement analysis. For each stride, the positions of the markers in the A/P and M/L axes relative to the ankle of the supporting foot were determined. The ankle marker with the lowest vertical height defined the supporting foot. This meant that for the first 50% of the stride the position of the markers were referenced to the left ankle and for the last 50% of the stride the markers were referenced to the right ankle. Individual strides were averaged together to determine a mean stride path for each marker. By subtracting the mean stride path of the markers from the actual excursion of the markers during each stride, deviations from the subjects normal walking pattern (the stride-to-stride variability of the marker movement) could be measured. Variability is often used as an index of the stability of the sensorimotor system the assumption being that enhanced variability is a reflection of reduced stability. For example, increased variability in movements of the COP during quiet stance was observed for patients with cerebellar pathology compared to normal subjects (Lucy & Hayes, 1985). In addition, Yack and Berger (1993) found that individuals with stability problems showed greater stride-to-stride variation in measures of trunk acceleration during walking.

*Correlation between movements of body segments during walking*

Analysis of the body segment position data showed that for young subjects there was a large positive correlation between movements of the pelvis from the mean path and movements of the head and shoulder from the mean path. Similar levels of correlation were observed for the elderly group and there was no significant difference between the two groups for either pelvis/shoulder correlation or pelvis/head correlation. The correlation between the pelvis and head in the M/L direction ($r$ = 0.79 (young), 0.77 (elderly)) was slightly lower than the correlation between the pelvis and shoulders ($r$ = 0.85 (young), 0.86 (elderly)). This may be due to attenuation of the M/L movements of the head relative to the pelvis in order to provide a stable visual and vestibular frame of reference. Cappozzo, Figura, Leo &

Marchetti (1978) determined that the motion of the head during walking is smoother than that of the pelvis and they related this to a need to protect the eyes and vestibular labyrinth from excessive mechanical stimulus. During quiet stance (Chapter 3.1) the magnitude of the M/L body sway during quiet stance is considerably less than that observed during walking and therefore attenuation of head movements is probably unnecessary.

*The relationship between marker deviations and the inverted pendulum model*

The inverted-pendulum model for walking was defined by the slope of the regression line fitted to the movements of the head, shoulder and pelvis markers (Top) from their mean stride path. The regression line was constrained through zero (the ankle of the supporting foot). Actual movements of the head from its mean path during walking were closely matched by the angular movements predicted by the inverted-pendulum model. In addition, actual movements of the Top markers showed large positive correlation with the deviation predicted by the inverted-pendulum model in both the A/P and M/L axes. The linear regression of marker movement variance against model variance (Figure 3.2.5) illustrated the validity of the model for both young and elderly subject groups. Increases in the marker movement variance were associated with increases in model variance. In other words, when a subject made large, additional sway movements during walking these movements were large inverted-pendulum-like sways of the body about the supporting foot.

*Are the deviations of the body during walking larger in the elderly compared to the young?*

In the A/P axis, there was no significant difference between the mean marker and model variance values for the elderly and young subject groups. However in the M/L axis, both mean marker and model variance were substantially larger in the elderly group compared to the young ($P < 0.001$). A similar result was obtained in Chapter 3.1 for the variance of the M/L marker and model movements from mean position during quiet stance. There is some evidence that elderly fallers have difficulty in controlling lateral stability during quiet and

perturbed stance (Maki *et al.* 1994; Maki *et al.* 1995). Older adults with stability problems would also experience difficulty controlling their lateral balance during an inherently unstable activity such as walking. Kaya *et al.* (1998) determined linear and angular momentum during walking in 10 elderly patients with bilateral vestibular hypofunction and ten age, height and weight matched controls. Their results showed that excessive lateral momentum was a clear marker of frontal plane instability during gait.

*Do the 'balance impaired elderly make large deviations of the body during walking?*

The mean marker and model variance values of the 'balance impaired' elderly were compared to those of young subjects and elderly subjects with good balance ability. Although there was a trend towards increased variance in the both elderly subgroups in the M/L axis compared to the young, the difference between the groups was not significant. The failure to find a difference between the subgroups may have been due partly to the low power of the tests. A repetition of the experiment using larger numbers of subjects may help to clarify this. It is also possible that because the walking trials were conducted with the eyes open, older subjects with impaired balance were able to compensate more easily for any vestibular or somatosensory deficits they had. It is well recognised that vision is an important stabilising input during both standing (Peterka & Black, 1990; Baloh *et al.* 1994) and walking (Patla, 1997).

*The relationship between movements of the body during walking and standing*

Inverted-pendulum-like movements of the body occur during walking and standing but the movements that occur during walking are much larger than those observed during quiet stance. A scatter plot of the variance of the marker movement during quiet stance and walking (Figure 3.2.8a and b) showed that that the variance of the body's movement from mean position during quiet stance was not a good predictor of the body's movement from mean stride path during walking. This was true for both the A/P and M/L axes and for both

the young and elderly subject groups. These results suggest that the association between measures of walking and standing balance is tenuous. If measures of postural stability during standing were used to estimate a subject's balance capabilities during walking and their predisposition to falling, a large proportion of the 'at-risk' population would be misclassified i.e. subjects that exhibit normal sway during standing but have poor balance during walking would not be identified. Bartlett, Maki, Fernie, & Holliday, (1986) estimated that the probability of incorrectly classifying a faller or non-faller based on sway velocity measured at the hip during standing was approximately 50%.

The information obtained about a person's balance from standing postural sway measures is not clearly understood. Increased postural sway has been associated with sensorimotor deficits (Ring *et al.* 1989; Duncan *et al.* 1992, Lord *et al.* 1992, Fife & Baloh, 1993) and in general, subjects with impaired balance have significantly greater sway during standing than subjects with normal balance (Diener *et al.* 1984a; Black *et al.* 1988; Baloh *et al.* 1995; Jarnlo & Thorngren, 1991). However normal sway values on stance tests are not uncommon in patients with gait imbalance. Some very unstable patients, such as those with Parkinson's disease can exhibit smaller than normal sway in stance (Horak *et al.,* 1992) and normal controls can have abnormal sway values, including loss of balance, on perturbed stance tests (Baloh *et al.,* 1995). In order to obtain a more accurate estimate of a subject's balance capabilities and risk of experiencing a fall quantitative measures of balance during walking should be obtained as well as measures of postural sway during standing.

## 4.3 Relationship between movements of COP and MVF/CSA

*Age-related changes in movements of COP and MVF/CSA*

In line with previous investigations (Bruce *et al.* 1989; Phillips *et al.* 1992) there was significant decline in the amount of muscle force produced per unit area for the adductor pollicis muscle in the elderly subjects when compared to the young subjects. The MVF produced by the elderly was 20.3 ± 2.6 % less (mean ± SEM) than that predicted from their CSA, using the regression line for the young subjects. In addition, the mean MVF/CSA ratio for the elderly group was significantly lower than the mean ratio calculated for the young group ($p < 0.05$). Measures of movement of the centre of pressure (COP) during quiet stance were significantly greater in the elderly subject group, particularly when proprioceptive input from the ankles and cutaneous inputs from the soles of the feet were reduced by standing on a foam pad. These findings are in accord with those of previous investigators (Sheldon 1963; Overstall. *et al.* 1977; Brocklehurst *et al.* 1982, Era and Heikkinen, 1985). The M/L measures were significantly greater under all test conditions in the elderly suggesting that the elderly have more difficulty controlling their balance in this direction. In the A/P direction the differences between the two groups only reached statistical significance when visual input was absent *and* somatosensory inputs were reduced by standing on the foam pad. Approximately one third of the elderly subjects were unable to perform the foam eyes closed test without losing their balance, so for this test condition the analysis was restricted to those with better balance capabilities. However, even these elderly subjects showed a significant increase in the variance of the COP movement compared to the young in both the M/L and A/P direction ($p < 0.001$).

*The relationship between movements of the COP and MVF/CSA*

Within the group of elderly subjects there was no significant correlation between MVF/CSA and movement of the COP for any of the four test conditions: eyes open, eyes closed, foam

eyes open and foam eyes closed. The scatter-plot of MVF/CSA versus M/L COP variance in the eyes closed condition (Figure 3.3.2b) illustrates that the elderly subjects can be classified into three main subgroups; those with MVF/CSA values and COP variance equal to young adults, those with reduced MVF/CSA but with COP variance within the limits of the young adults and those with similar MVF/CSA to young adults but with greater COP variance. Only one elderly subject exhibited both reduced MVF/CSA and increased COP variance for this test condition. This result suggests that these factors do not have a common underlying mechanism and in general afflict different individuals.

Previous investigators have found low but significant negative correlation between grip strength and sway with the eyes open and closed (Era and Heikkinen, 1985) and between quadriceps and ankle dorsiflexion strength and sway on a foam pad with the eyes closed (Lord *et al.* 1991). The force produced per cross sectional area of muscle (MVF/CSA) was not measured in either of the above mentioned studies, so it is possible that muscle atrophy may be responsible for the weak but significant correlation observed by these authors. If this is the case, then it is possible that the effects of atrophy could be reversed with exercise. Lord *et al.* (1994) measured quadriceps strength and body sway in 17 men and 27 women aged 50 to 75 years (mean age 62.4 years) before and after participating in a ten week exercise program (total of 19 one hour sessions) which included walking, muscle strength and flexibility exercises. On completion of the program, the subjects showed significantly improved performance in the tests of quadriceps strength, sway on a firm surface with the eyes closed and on a compliant surface with the eyes closed.

The muscle group measured, the difficulty of the postural sway test, and the characteristics of the subject group may also determine if a significant relationship is found between muscle strength and sway. Era and Heikkinen, (1985) found a significant correlation between grip strength and extent of postural sway ($r = -0.255$, $p < 0.01$) but no correlation

between sway and forces of the trunk and leg muscles. Lord *et al* (1991) found a low but significant negative correlation between quadriceps and ankle strength/height and sway when the subjects stood on a compliant surface either with the eyes open or closed ($r$ values between -0.23 and -0.25, $p < 0.05$). However no significant relationship was observed when the subjects stood on a firm surface. The subjects in the Lord *et al.* (1991) study were recruited from a hostel for the aged and were considerably older (mean age 82.7 years) and less functionally able (40% requiring personal care assistance on a daily basis and 22% used walking stick or frame) than the community-dwelling subjects examined in this thesis.

*Relationship between MVF/CSA and walking variance in the elderly*

No significant correlation between MVF/CSA and walking variance was found for the 15 elderly subjects who participated in both experiments. Other studies have examined the relationship between muscle strength and *walking speed* and found either a strong correlation (Fiatarone *et al.* 1990) or a weak but significant correlation between strength and walking speed (Bassey *et al.* 1988). Fiatarone *et al.* (1990) found a strong negative correlation ($r$ = -0.745, $P < 0.01$) between muscle strength of the quadriceps and the time taken to walk 6 m for a small group (N = 10) of extremely elderly (mean age 90 years, range 86 - 96 years), frail and institutionalised subjects. Bassey *et al.* (1988) found low but significant associations between triceps surae strength and chosen walking speed for elderly men (mean age 71 years, $r$ = 0.41, $P < 0.001$) and women (mean age 72 years, $r$ = 0.36, $P < 0.01$). Buchner *et al.* (1996) have suggested that the relationship between leg strength and gait speed in older adults is non-linear, based on measurements of isokinetic leg strength obtained for 4 muscle groups (knee extensor, knee flexor, ankle plantar flexor, ankle dorsiflexor) and gait speed measured over a 15.2 m course. The nature of the relationship is such that, in stronger subjects there is no association between strength and gait speed, while in weaker subjects, a significant association can be found. In Chapter 3.2 the mean MVF/CSA ratio calculated for the 15 elderly subjects was not significantly different from

that calculated for the young subjects. If a non-linear relationship exists between MVF/CSA and walking variance similar to that proposed by Buchner (1996) for leg strength and gait speed in the elderly, then it may be expected that for stronger elderly subjects there is no correlation between MVF/CSA and walking variance. If a larger study was conducted using both frail and strong elderly subjects it is possible that an association between MVF/CSA and walking variance might be observed.

*Correlation between COP variance and walking variance*

Significant relationships between postural sway parameters and spatial-temporal gait parameters have been observed previously by other investigators (Imms & Edholm 1981; Lord *et al.* 1996). For example, Imms and Edholm (1981) measured the amount of body sway in elderly subjects (aged 60 to 99 years) during quiet standing with the eyes open and correlated this measure with velocity of walking. A significant negative correlation between the two measures was found ($r$ = -0.599; $p$< 0.001). Approximately half of the 71 elderly subjects considered their outdoor activity to be unlimited and some of these subjects were capable of walking up to 9 km. The remainder had reduced mobility, ranging from experiencing difficulty negotiating stairs to being house-bound and relying on walking aids. Lord *et al.* (1996) found low but significant correlation between percentage of the stride in the stance phase and total length of sway path under four test conditions, EO, EC, FEO, FEC for a group of 183 community dwelling women aged between 22 - 99 years. The correlation coefficients ranged from 0.16 to 0.24, ($P$ < 0.01 (EO,EC,FEO) $P$ < 0.05 (FEC)). Sway on the foam with the eyes open and eyes closed was also associated with gait velocity and stride length ($r$-values ranging from = -0.24 to -0.17).

In this thesis the relationship between the variance of the COP movements and the variance of the body's movements in the A/P and M/L axes during walking were investigated. No significant correlation was observed between the walking variance and COP variance in the

A/P axis but in the M/L direction, a significant correlation was found between walking variance and COP variance for the EO ($r = 0.645$, $P < 0.01$) and FEC ($r = 0.818$, $p < 0.01$) test conditions. There was also a positive correlation between walking variance and M/L COP variance (EC), although this did not reach significance ($r = 0.465$, $p = 0.08$). In Chapter 3.2 it was shown that there was no significant relationship between walking variance and the variance of marker movement (and thus COP movement) during standing (EC) in either the A/P or M/L direction. It was therefore concluded (Chapter 4.2) that postural stability during standing was not a good predictor of postural stability during walking in older adults. Why the apparent discrepancy between the two sets of results? The data used for comparing movements of the body during standing and walking was collected during a single testing session, whereas the data used for comparing COP variance and walking variance was obtained on two separate occasions, approximately 8 months apart. There is a possibility that changes may have occurred in a subjects balance capabilities during this 8 month time period and two subjects were excluded from the analysis because of an apparent increase in COP variance from one test occasion to the next. Because of this potential for change in capabilities over time, data collected on the same test occasion must be a more accurate representation of the individual's stability. The number of subjects analysed in both sets of results were small, 21 elderly subjects in the walking versus standing marker variance analysis and 15 (EO) and 11 (FEC) subjects respectively in the walking variance versus M/L COP variance analysis. To determine whether the relationship observed between M/L COP measures and M/L walking variance is real a large-scale study with a greater number of subjects would need to be conducted. This would involve measuring COP movements during the four test conditions (EO, EC, FEO, FEC) and stability during walking on the same test occasion.

## 4.4 *Recovery responses to a trip*

A method of producing a repeatable and quantifiable perturbation to the swing limb during walking was developed and the kinematic and electromyographic responses to the perturbation were recorded in nine young adults using the Coda *mpx30*. One of the benefits of using the Coda *mpx30* system was that the perturbation could be triggered at a specified point in the gait cycle, (toe-off) by using the real-time position of the heel marker. Specific strides could also be targeted which allowed the trips to be applied randomly within the trial. This control over the application of the perturbation produced very consistent responses and enabled the averaging of an individual's results thus relatively small effects could be detected with certainty. The following discussion focuses on the recovery strategies observed and how the responses recorded bilaterally from lower limb muscle groups can be explained by the changes in kinematics following a perturbation.

*Movement Outcome*

The subjects in response to the perturbation adopted two different recovery strategies. The majority of subjects tended to lengthen swing phase of the obstructed limb so that heel contact occurred later in perturbed strides compared to normal walking (**Recovery Strategy 1**). In contrast, two of the subjects shortened the swing phase of the obstructed limb, which resulted in a more flexed knee and flattened heel contact on landing (**Recovery Strategy 2**). Previously, Dietz, Quintern, Boos & Berger (1986) found that when a mechanical disturbance was applied during the swing phase the duration of both ipsilateral swing and the contralateral stance were prolonged. A lowering strategy with a flat foot landing and shorter step length was observed by Eng, Winter & Patla (1994) in response to the foot contacting an obstacle during late swing. They suggested that this strategy was the safest to use when the perturbation occurs during late swing, a time at which the Centre of Mass is already anterior to the stance leg. They also suggested that the perceived threat of the task is

a likely factor contributing to the variability of the movement responses and highlights cognitive influences on the reflex responses. Fall prone subjects and Parkinson's Disease patients have shown a tendency to use a double step recovery strategy (as opposed to a single step strategy) more often than healthy subjects to clear an obstacle dropped 1 m in front of them on a treadmill (Duysens, Schillings & van Wesel, 1997). Factors such as a slow walking velocity and shorter stride as well as a fear of falling may have contributed to the preferential use of a double step recovery strategy by these subjects.

Why two of the nine young subjects in this study used an alternative strategy to the one used more generally is open to speculation. Both subjects had a shorter stride time than the subjects using **Recovery Strategy 1**. It is possible that if the perturbation of the swing phase is sufficiently long, than **Recovery Strategy 2** may be the preferred response for all subjects. The **Recovery Strategy 2** subjects were also more naïve to the experimental set-up than the other seven subjects who were students familiar with the Coda *mpx30* and the recording of EMG. It is possible that the novel environment may have made the two subjects more apprehensive and thus influenced the type of recovery strategy used. Whether the two subjects using **Recovery Strategy 2** are more at risk of experiencing a fall cannot be determined. The perturbation applied was quite small and all the subjects were able to regain their balance and re-establish their normal walking pattern relatively quickly.

***Can the responses observed in muscles of the obstructed limb be explained by changes in the kinematics following perturbation?***

*Quadriceps*

For the greater part of swing, knee movement occurs without active contraction of the quadriceps but is driven by passive forces. When the normal knee movement is obstructed, a corrective activation of the quadriceps occurs at a time when the muscle group is normally inactive. This activation enables the limb to overcome the resistance to forward movement

produced by the brake. Dietz, Quintern & Berger (1986) observed similar responses in rectus femoris following an 80 ms perturbation of the swing limb. The latency of the response was 65 ms from the onset of the perturbation. Garrett and Luckwill (1983) also applied a short resistance to the swing limb and found the latency of the response in the quadriceps to be 78.2 ± 26.4 ms (mean ± S.D.) from the onset of the perturbation. These latencies are somewhat shorter than those recorded in this experiment (108 ± 19 ms, mean ± S.E.M, measured from the peak force) but the timing suggests that in all three studies the responses involve polysynaptic pathways.

*Hamstrings*

During normal walking, the hamstrings (long head of BF) are active from late swing to just after heel strike. During late swing, eccentric or isometric contraction of the hamstrings decelerates the limb slowing down both hip flexion and knee extension. At heel strike the simultaneous activation of hamstrings and the quadriceps (Vasti) help to stabilise and position the knee and decelerate the body. Following the perturbation, an inhibition of activity in the right hamstrings was observed in subjects using **Recovery Strategy 1.** Obstruction of the right limb during the early swing phase delayed the 'switching on' of the hamstrings for 133 ± 13 ms (Mean ± S.E.M) and thus heel contact occurred later in perturbed strides compared to normal walking. Inhibitory responses have also been observed in the TA, rectus femoris and vastus lateralis of the obstructed limb following a perturbation applied during late swing Eng, *et al.* 1994).

No inhibition of activity in the hamstrings was observed for the two subjects using **Recovery Strategy 2,** rather there was increased activity recorded in the hamstrings which was probably responsible for the earlier heel contact observed in perturbed strides in these subjects. Step length can be reduced by increased eccentric hamstring muscle activity during late swing (Winter, 1992).

*Tibialis Anterior*

During initial swing, TA begins a concentric contraction to allow the foot to clear the treadmill as the limb is swung forward. During terminal swing and heel contact, TA continues its activity but switches to an isometric or eccentric contraction to control the foot as it is lowered to the treadmill. The perturbation was applied at toe off, when the right TA was already active, and in the concentric phase. The design of the foot harness and attachment was such that there was a tendency for the foot to be plantarflexed when the brake was applied. The resulting muscle stretch of the TA could potentially activate primary and secondary muscle spindle afferents. However responses in the TA of the perturbed limb never approached the short latencies which might indicate a monosynaptic reflex response. In all subjects an increase in the magnitude of the muscle activation was observed, occurring with a latency of around 65 ms. Plots of changes in ankle angle indicated that there was an increased dorsiflexion of the foot following the perturbation. This may explain the increased activity recorded in the TA of the obstructed limb. In subjects using **Recovery Strategy 2** there was an early and more flat-footed landing following the perturbation. In these subjects the duration of TA activation in perturbed strides was shorter than that recorded during normal walking and the peak of muscle activity responsible for controlled lowering of the foot at heel contact appeared to be absent.

*Medial Gastrocnemius*

The gastrocnemius is primarily active during the stance phase of gait. However, a small response with a latency of 70 ms was observed in the obstructed swing limb of 3 out of the 8 subjects using **Recovery Strategy 1.** It is unclear what the purpose of this response could be. It is possible that the response may have been part of a startle reaction. When the braking mechanism is activated the subject hears a relatively loud click. Startle responses are more frequently observed in flexors than in extensors but Rossignol (1975) recorded startle responses in gastrocnemius-soleus in response to auditory stimulation. Blindfolded

subjects sat with their leg supported and EMG recordings were made of the response to auditory stimulus in three conditions: with the ankle of the subject at rest, with the ankle in active flexion and with the ankle in active extension. A great deal of variation in the responsiveness to sound stimulation was found in the subjects with 5 out of 11 subjects not presenting any startle response in the leg at rest. Responses in gastrocnemius were more likely to occur during ongoing extensor activity or when the ankle was at rest but after a period of voluntary extensor activity. The latter condition may be similar to the situation in gastrocnemius at the beginning of the swing phase of gait. One of the subjects that used the double step strategy had a response that occurred at a relatively short latency (40 ms) in addition to an increased activation of the muscle at a longer latency. This early peak may correspond to the P1 response described in the cat (Forssberg, 1979). Responses consisting of two peaks with latencies of 47 and 73 ms have also been observed in humans in response to electrical stimulation during gait (Duysens and Tax, 1994).

*How do EMG responses in the stance (unobstructed limb) contribute to the recovery strategy?*

It is more difficult to interpret the EMG responses in the stance limb as no kinematic data was recorded from that side of the body. It is likely that some lateral rotation of the body occurred as a consequence of the perturbation and this movement may have been partly responsible for the muscle responses observed on the contralateral side. However, responses in the contralateral limb are also a necessary part of a generalised behavioural reaction that ensures stability and forward progression following a gait perturbation. Evidence of interlimb reflexes has previously been reported in response to skin stimulation during walking in cats and humans (Kugelberg, Eklund & Grimby, 1960; Duysens, Loeb & Weston, 1980; Tax, Duysens, Trippel & Dietz, 1990; Duysens, Tax, van der Doelen, Trippel, Dietz, 1991). For example, crossed flexor responses were described by Kugelberg, Ecklund & Grimby (1960), following high intensity stimulation of the sole of the foot.

In this study the responses observed in the contralateral limb appear to be aimed at stabilisation of the body. Typically there was an increased activation of both gastrocnemius and hamstrings on the stance limb. This would help to provide a stable base from which to recover from the perturbation. Gastrocnemius also provides propelling force in walking. Increased activation of this muscle during late stance would help maintain the forward progression of the body in spite of the perturbation. For subjects using **Recovery Strategy 1** small responses were also recorded in the quadriceps of the contralateral stance limb. Although the latency of the response in the quadriceps was longer than that observed for the hamstrings, the two muscles were active simultaneously. This co-contraction of the muscles may further increase the stability of the body. In one of the subjects using **Recovery Strategy 2** (Subject 7) no response was recorded in the contralateral hamstrings following the perturbation but a very strong activation of the quadriceps in the contralateral limb was recorded. The reverse was true for the other subject using **Recovery Strategy 2**. In Subject 9, *excitatory* responses were recorded from the hamstrings of both the obstructed limb and the contralateral stance limb but no response was recorded in the contralateral quadriceps. Although the movement response of these two subjects was very similar, different muscle activation patterns were observed. This adaptability suggests that the responses are not simple stereotyped reflex patterns but are probably dependent on supraspinal control as suggested previously by the abnormal function of polysynaptic spinal reflexes during perturbed human locomotion in subjects with lesions of the supraspinal motor centres (Berger *et al.* 1987, 1988).

Ghori and Luckwill (1989) observed reflex responses in vastus lateralis, rectus femoris and TA of the stance limb after a short perturbation applied in early swing. The latency of the responses were between 82 ± 14 ms (mean ± SD, vastus lateralis) and 86 ± 12 ms (TA) which is close to the latencies reported here for responses in the contralateral limb.

The present work demonstrates that complex patterns of muscle activation in the lower limbs on both sides of the body are generated in response to an unexpected gait perturbation. The recovery response must be initiated within a short time of the perturbation in order to prevent falling and to maintain the ongoing locomotion. The next stage of the research will be to study the recovery responses to a trip in healthy elderly subjects and in elderly subjects with balance impairments. With minor modifications this protocol would be suitable for testing elderly subjects. The aims of this future research would be: 1) to determine if the elderly use the same recovery strategies as young subjects, 2) to determine whether the latencies of the responses are similar and 3) to investigate whether there is a difference between the recovery strategies used by healthy elderly subjects and balance impaired elderly.

## 4.5 Summary of the main findings

- The movement of the body during quiet stance can be described as a simple inverted pendulum moving about the ankles in the anterior-posterior and medial-lateral directions.
- The inverted pendulum model of quiet stance is applicable to both young and elderly subjects.
- The greater COP movements observed in elderly subjects are due to increased inverted pendulum sway of the body in the medial-lateral direction compared to young subjects.
- An inverted pendulum model can also be used to describe movements of the body during walking.
- The elderly demonstrated greater sway in the medial-lateral direction when walking compared to young subjects.
- For both young and old subjects, no correlation was found between sway while walking and sway while standing with the eyes closed when the measurements were made on the same test occasion. However, for the elderly subjects a significant correlation was found between sway while walking and earlier measures of COP movements while standing. The static measurement which best-predicted sway during walking was medial-lateral movements of the COP when standing on a compliant surface with the eyes closed.
- There was no tendency for the elderly subjects who were weakest also to be the people with the greatest sway. This suggests that muscle weakness and increased postural sway are independent processes that each afflict a proportion of the elderly, and are thus probably caused by different physiological changes.
- Two different recovery responses were observed in young subjects in response to a perturbation of gait. The majority of subjects (7 out of 9) lengthened the swing phase of the perturbed leg, delaying heel strike. The alternative strategy involved a shorter swing phase in the perturbed leg with a flat-footed landing. The latency of the muscular responses indicates that polysynaptic pathways are involved.

# *References*

Alnaqeeb, M.A., Al Zaid, N.S. & Goldspink, G. (1984). Connective tissue changes and physical properties of developing and ageing skeletal muscle. *Journal of Anatomy* **139**, 677-689.

Aniansson, A., Grimby, G. & Rundgren, A. (1980). Isometric and isokinetic quadriceps muscle strength in 70-year-old men and women. *Scandinavian Journal of Rehabilitation Medicine* **12**, 161-168.

Aniansson, A., Sperling, L., Rundgren, A. & Lehnberg, E. (1983). Muscle function in 75-year-old men and women. *Scandinavian Journal of Rehabilitation Medicine* **9**, 92-102.

Archea, J.C. (1985). Environmental factors associated with stair accidents and falls in the elderly. *Clinics in Geriatric Medicine* **1**, 555-567.

Armstrong, A.L., Oborne, J., Coupland, C.A.A., Macpherson, M.B., Bassey, E.J. & Wallace, W.A. (1996). Effects of hormone replacement therapy on muscle performance and balance in post-menopausal women. *Clinical Science* **91**, 685-690.

Armstrong, D.M. (1988). The supraspinal control of mammalian locomotion. *Journal of Physiology* **405**, 1-37.

Arsenualt, A.B. (1986). Treadmill versus walkway locomotion in humans: an EMG study. *Ergonomics* **29**, 665-676.

Attebo, K., Mitchell, P. & Smith, W. (1996). Visual acuity and the causes of visual loss in Australia: The Blue Mountains Eye Study. *Ophthalmology* **103**, 357-364.

Austin, M.C. & Kalivas, P.W. (1991). Dopaminergic involvement in locomotion elicited from the ventral pallidum / substantia innominata. *Brain Research* **542**, 123-131.

Baloh, R.W., Fife, T.D., Zwerling, L., Socotch, T., Jacobsen, K., Bell, T. & Beykirch, K. (1994). Comparison of static and dynamic posturography in young and older normal people. *Journal of the American Geriatric Society* **42**, 405-412.

Baloh, R.W., Spain, S., Socotch, T.M., Jacobson, B.A. & Bell, T. (1995). Posturography and balance problems in older people. *Journal of the American Geriatric Society* **43**, 638-644.

Barrett-Connor, E. (1991). Postmenopausal estrogen and prevention bias. *Annals of Internal Medicine* **115**, 455-456.

Bartlett, S.A., Maki, B.E., Fernie, G.R. & Holliday, P.J. (1986). On the classification of a geriatric subject as a faller or nonfaller. *Medical and Biological Engineering and Computing* **24**, 219-222.

Bassey, E.J., Bendall, M.J. & Pearson, M. (1988). Muscle strength in the triceps surae and objectively measured customary walking activity in men and women over 65 years of age. *Clinical Science* **74**, 85-89.

Beatty, W.W. (1979). Gonadal hormones and sex differences in non-reproductive behaviors in rodents: organizational and activational influences. *Hormonal Behavior* **12**, 112-163.

Becker, J.B., Snyder, P.J., Westgate, S.A. & Jenuwine, M.J. (1987). The influence of estrous cycle and intrastriatal estradiol on sensorimotor performance in the female rat. *Pharmacology, Biochemistry and Behaviour* **27**, 53-59.

Belanger, M. & Patla, A.E. (1984). Corrective responses to perturbation applied during walking in humans. *Neuroscience Letters* **49**, 291-295.

Benda, B.J., Riley, P.O. & Krebs, D.E. (1994). Biomechanical relationship between centre of gravity and centre of pressure during standing. *IEEE Transactions on Rehabilitation Engineering* **2**, 3-9.

Bendz, H. & Feinberg, M. (1984). Aspirin increases serum lithium ion levels. *Archives of General Psychiatry* **41**, 311.

Berg, W.P., Alessio, H.M., Mills, E.M. & Tong, C. (1997). Circumstances and consequences of falls in independent community-dwelling older adults. *Age and Ageing* **26**, 261-268.

Berger, W., Dietz, V. & Quintern, J. (1984). Corrective reactions to stumbling in man: neuronal co-ordination of bilateral leg muscle activity during gait. *Journal of Physiology* **357**, 109-125.

Berger, W., Dietz, V. & Quintern, J. (1987). Afferent and efferent control of stance and gait: developmental aspects of compensatory mechanisms. *Electroencephalography and Clinical Neurophysiology* **61**, 244-252.

Berger, W., Horstmann, G. & Dietz, V. (1988). Spastic paresis: impaired spinal reflexes and intact motor programs. *Journal of Neurology, Neurosurgery and Psychiatry* **51**, 568-571.

Bergstrom, B. (1973). Morphology of the vestibular nerve. II. The number of myelinated vestibular nerve fibers in man at various ages. *Acta Otolaryngology. (Stockholm)* **76**, 173-179.

Binkhorst, R.A. & van't Hof, M.A. (1973). Force-velocity relationship and contraction time of the rat fast plantaris muscle due to compensated hypertrophy. *Pflügers Archives* **342**, 145-158.

Black, F.O., Shupert, C.L, Horak, F.B. & Nashner, L.M. (1988). Abnormal postural control associated with peripheral vestibular disorders. *Progress in Brain Research* **76**, 263-275.

Blake, A.J., Morgan, K., Bendall, M.J., Dallosso, H., Ebrahim, S.B.J., Arie, T.H.D., Fentem, P.H. & Bassey, E.J. (1988). Falls by elderly people at home: prevalence and associated factors. *Age and Ageing* **17**, 365-372.

Blanke, D.J. & Hageman, P.A. (1989). Comparison of gait of young men and elderly men. *Physical Therapy* **69**, 144-148.

Blin, O., Ferrandez, A.M. & Serratrice, G. (1990). Quantitative analysis of gait in Parkinson patients: Increased variability of stride length, *Journal of the Neurological Sciences* **98**, 91-97.

Bohannon, R.W., Larkin, P.A., Cook, A.C., Gear, J. & Singer, J. (1984). Decrease in timed balance test scores with ageing. *Physical Therapy* **64**, 1067-1070.

Bouisset, S. & Duchêne, J. (1994). Is body balance more perturbed by respiration in seating than in standing posture? *NeuroReport* **5**, 957-960.

Brandell, B.R. & Williams, K. (1974). An analysis of cinematographic and electromyographic recordings of human gait. In *Biomechanics IV,* ed. Nelson, R.C. & Morehouse, C.A., pp 91-97 Baltimore: University Park Press.

Briggs, R.C., Gossman, M.R., Birch, R., Drews, J.E. & Shaddeau, S.A. (1989). Balance performance among non-institutionalised elderly women. *Physical Therapy* **69**, 748-756.

Brocklehurst, J.C., Exton-Smith, A.N., Lempert-Barber, S.M., Hunt, L.P. & Palmer, N.K. (1978). Fractures of the femur in old age: a two-centre study of associated clinical factors and the cause of the fall. *Age and Ageing* **7**, 2-15.

Brocklehurst, J.C., Robertson, D. & James-Groom, P. (1982). Clinical correlates of sway in old age-sensory modalities. *Age and Ageing,* **11**, 1-10.

Brooks, S.V. & Faulkner, J.A. (1994). Skeletal muscle weakness in old age: underlying mechanisms. *Medicine & Science in Sports & Exercise* **26**, 432-439.

Brooks, V.B. and Thach, W.T. (1981). Cerebellar control of posture and movement. In *Handbook of Physiology. The Nervous System, Motor Control* ed. Brooks, V.B., pp. 877-946. Bethesda, MD: American Physiological Society.

Bruce, S.A., Newton, D. & Woledge, R.C. (1989). Effects of age on voluntary force and cross-sectional area of human adductor pollicis muscle. *Quarterly Journal of Experimental Physiology* **74**, 359-362.

Bruce, S.A., Phillips, S.K. & Woledge, R.C. (1986). Isometric force in maximum voluntary contractions related to an estimate of the cross-sectional area of adductor pollicis in the human subject. *Journal of Physiology* **372**, 31P.

Buchner, D.M., Larson, E.B., Wagner, E.H., Koepsell, T.D. & Lateur, B.J. (1996). Evidence for a non-linear relationship between leg strength and gait speed. *Age and Ageing* **25**, 386-391.

Caird, F. (1983). Ageing and accidents. *RoSPA Conference.*

Campbell, A.J., Borrie, M.J. & Spears, G.F. (1989). Risk factors for falls in a community-based prospective study of people 70 years and older. *Journal of Gerontology*, **44**, M112-117.

Campbell, A.J., Borrie, M.J., Spears, G.F., Jackson, S.L., Brown, J.S. & Fitzgerald, J.L. (1990). Circumstances and consequences of falls experienced by a community population 70 years and over during a prospective study. *Age and Ageing* **19**, 345-346.

Campbell, A.J., Reinken, J., Allan, B.C. & Martinez, G.S. (1981). Falls in old age: a study of frequency and other related factors. *Age and Ageing* **10**, 264-270.

Cappozzo, A., Figura, F., Leo, T. & Marchetti, M. (1978). Movements and mechanical energy changes in the upper part of the human body during walking. In: *Biomechanics VI-A,* ed. Asmussen, E. & K. Jorgensen, K., pp. 272-279. Baltimore : University Park Press.

Cauley, J.A., Seeley, D.G., Ensrud, K., Ettinger, B., Black, D. & Cummings, S.R. (1995). Estrogen replacement therapy and fractures in older women. *Annals of Internal Medicine* **122**, 9-16.

Chen, H.C., Ashton-Miller, J.A., Alexander, N.B. & Schultz, A.B. (1991). Stepping over obstacles: Gait patterns of healthy young and old adults. *Journal of Gerontology* **46**, M196-203.

Chen, H.C., Ashton-Miller, J.A., Alexander, N.B. & Schultz, A.B. (1994). Age effects on strategies used to avoid obstacles. *Gait and Posture* **2**, 139-146.

Chen, H.C., Schultz, A.B., Ashton-Miller, J.A., Giodani, B., Alexander, N.B. & Guire, K.E. (1996). Stepping over obstacles: dividing attention impairs performance of old more than young adults. *Journal of Gerontology* **51A**, M116-M122.

Collins, J.J. & De Luca, C.J. (1993). Open-loop and closed-loop control of posture: A random-walk analysis of centre-of-pressure trajectories. *Experimental Brain Research* **95**, 308-319.

Collins, J.J., De Luca, C.J., Burrows, A. & Lipsitz, L.A. (1995). Age-related changes in open-loop and closed-loop postural control mechanisms. *Experimental Brain Research* **104**, 480-492.

Crenna, P. & Frigo, C. (1984). Evidence of phase-dependent nociceptive reflexes during locomotion in man. *Experimental Neurology* **85**, 336-345.

Cummings, S.R., Kelsey, J.L., Nevitt, M.C. & O'Dowd, K.J. (1985). Epidemiology of osteoporosis and osteoporotic fractures, *Epidemiological Reviews* **7**, 178.

Day, B.L., Steiger, M.J., Thompson, P.D. & Marsden, C.D. (1993). Effect of vision and stance width on human body motion when standing: Implications for afferent control of lateral sway. *Journal of Physiology* **469**, 479-499.

Dempster, W.T. (1955). Space requirements of the seated operator. *Technical Report WADC-TR-55-159*, pp 55-159. Wright-Patterson Air Force Base, OHIO: Aerospace Medical Research Laboratory.

De Reuck, J., Crevits, L., De Coster, W., Sieben, G. & Vander-Eccken, H. (1980). Pathogenesis of Binswanger chronic progressive subcortical encephalopathy. *Neurology* 30, 920-928.

Dichgans, J., Mauritz, K.H., Allum, J.H.J. & Brandt, T. (1976). Postural sway in normals and ataxic patients: analysis of the stabilising and destabilising effects of vision. *Aggressologie* **17C**, 15-24.

Diener, H.C., Dichgans, J., Bacher, M. & Gompf, B. (1984a). Quantification of postural sway in normals and patients with cerebellar diseases. *Electroencephalography and Clinical Neurophysiology*, **57**, 134-142.

Diener, H.C., Dichgans, J., Guschlbauer, B. & Mau, H. (1984b). The significance of proprioception on postural stabilisation as assessed by ischemia. *Brain Research* **296**, 103-109.

Dietz, V. & Berger, W. (1984). Interlimb coordination of posture in patients with spastic paresis. Impaired function of spinal reflexes. *Brain* **107**, 965-78.

Dietz, V., Quintern, J. & Berger, W. (1984). Corrective reactions to stumbling in man: functional significance of spinal and transcortical reflexes. *Neuroscience Letters* **44**, 131-135.

Dietz, V., Quintern, J. & Berger, W. (1985). Afferent control of human stance and gait: evidence for blocking of group I afferents during gait. *Experimental Brain Research* **61**, 153-163.

Dietz, V., Quintern, J. & Berger, W. (1986). Stumbling reactions in man: release of a ballistic movement pattern. *Brain Research* **362**, 355-357.

Dietz, V., Quintern, J., Boos, G. & Berger, W. (1986). Obstruction of the swing phase during gait: Phase dependent bilateral leg muscle co-ordination. *Brain Research* **384**, 166-169.

Dietz, V., Trippel, M. & Horstmann, G.A. (1991). Significance of proprioceptive and vestibulospinal reflexes in the control of stance and gait. In *Adaptability of Human Gait,* ed. Patla, A.E.., pp 37-52. New York: North Holland.

Do, M.C., Breniere, Y. & Brenguier, P. (1982). A biomechanical study of balance recovery during the fall forward. *Journal of Biomechanics,* **15**, 933-939.

Downton, J.H. & Andrews, K. (1990). Postural disturbance and psychological symptoms amongst elderly people living at home. *International Journal of Geriatric Psychiatry* 5, 93-98.

Drew, T. & Rossignol, S. (1987). A kinematic and electromyographic study of cutaneous reflexes evoked from the forelimb of unrestrained walking cats. *Journal of Neurophysiology* **57**, 1160-1184.

Duncan, G., Wilson, J.A., MacLennan, W.J. & Lewis S. (1992). Clinical correlates of sway in elderly people living at home. *Gerontology* **38**, 160-166.

Duysens, J., Loeb, G.E. & Weston, B.J. (1980). Crossed flexor reflex responses and their reversal in freely walking cats. *Brain Research* **197**, 538-542.

Duysens, J., Schillings, A.M. & van Wesel, B.M.H. (1997). The human solutions to perturbations during treadmill walking. *Proceedings of the Brain and Movement International Symposium, Moscow,* p 65.

Duysens, J. and Tax, T. (1994). Interlimb reflexes during gait in cat and human. In: *Interlimb Coordination: Neural, Dynamical and Cognitive Constraints*, ed. Swinnen, S., Heuer, H., Massion, J & Casaer, P. London: Academic Press Ltd.

Duysens, J. Tax, A.A., Trippel, M. & Dietz, V. (1993). Increased amplitude of cutaneous reflexes during human running as compared to standing. *Brain Research* **613**, 230-238.

Duysens, J., Trippel, M., Horstmann, G.A. & Dietz, V. (1990). Gating and reversal of reflexes in ankle muscles during human walking. *Experimental Brain Research* **82**, 351-358.

Elkind-Hirsh, K.E., Stoner, W.R., Stach, B.A. & Jerger, J.F. (1992) Estrogen influences auditory brainstem responses during the normal menstrual cycle. *Hearing Research* **60**, 143-148.

Elkind-Hirsch, K.E, Wallace, E., Stach, B.A. & Jerger, J.F. (1992). Cyclic steroid replacement alters auditory brainstem responses in young women with premature ovarian failure. *Hearing Research* **64**, 93-98.

Eng, J.J., Winter, D.A. & Patla, A.E. (1994). Strategies for recovery from a trip in early and late swing during human walking. *Experimental Brain Research* **102**, 339-349.

Era, P. & Heikkinen, E. (1985). Postural sway during standing and unexpected disturbance of balance in random samples of men of different ages. *Journal of Gerontology* **40**, 287-295.

Essen-Gustavsson, B. & Borges, O. (1986) Histochemical and metabolic characteristics of human skeletal muscle in relation to age. *Acta Physiologica Scandinavica* **126**, 107-114.

Fernie, G.R., Gryfe, C.I., Holliday, P.J. & Llewellyn A. (1982). The relationship of postural sway in standing to the incidence of falls in geriatric subjects. *Age and Ageing* **11**, 11-16.

Fiatarone, M.A., Marks, E.C., Ryan, N.D., Meredith, C.N., Lipsitz, L.A. & Evans, W.J. (1990). High-intensity strength training in nonagenarians: effects on skeletal muscle. *Journal of the American Medical Association* **263**, 3029-3034.

Fife, T.D. & Baloh, R.W. (1993). Disequilibrium of unknown cause in older people. *Annals of Neurology* **34**, 694-702.

Finley, F.R., Cody, K.A. & Finizie, R.V. (1969). Locomotion patterns in elderly women. *Archives of Physical Medicine and Rehabilitation* **50**, 140-146.

Fitzpatrick, R. & McCloskey, D.I. (1994). Proprioceptive, visual and vestibular thresholds for the perception of sway during standing in humans. *Journal of Physiology* **478**, 173-186.

Forssberg, H. (1979). Stumbling corrective reaction: a phase-dependent compensatory reaction during locomotion. *Journal of Neurophysiology* **42**, 936-953.

Forssberg, H., Grillner, S. & Rossignol, S. (1977). Phasic gain control of reflexes from the dorsum of the paw during spinal locomotion. *Brain Research* **132**, 121-139.

Fregly, A.R., Smith, M.J. & Graybiel, A. (1973). Revised normative standard of performance of men on a quantitative ataxia test battery. *Acta Otolaryngology (Stockholm)* **75**, 10-16.

Gabell, A. & Nayak, U.S.L. (1984). The effect of age on variability in gait. *Journal of Gerontology* **39**, 662-666.

Gabell, A., Simons, M.A. & Nayak, U.S.L. (1985). Falls in the healthy elderly: Predisposing causes. *Ergonomics* **28**, 965-975.

Garrett, M & Luckwill, R.G. (1983). Role of reflex responses of knee musculature during the swing phase of walking in man. *European Journal of Applied Physiology* **52**, 36-41.

Gauthier, L. & Rossignol, S. (1981). Contralateral responses to cutaneous stimulation during locomotion in high decerebrate cats. *Brain Research* **207**, 303-320.

Gehlsen, G.M. & Whaley, M.H. (1990). Falls in the elderly: Part I, gait. *Archives of Physical Medicine and Rehabilitation* **71**, 735-738.

Geursen, J.B., Altene, D., Massen, C.H. & Verduin, M. (1976). A model of standing man for the description of his dynamic behaviour. *Agressologie* **17**, 63-69.

Ghori, G.M.U. & Luckwill, R.G. (1989). Pattern of reflex responses in lower limb muscles to a resistance in walking man. *European Journal of Applied Physiology* **58**, 852-857.

Goldie, P.A., Bach, T.M. & Evans, O.M. (1989). Force platform measures for evaluating postural control: reliability and validity. *Archives of Physical Medicine and Rehabilitation* **70**, 510-517.

Grabiner, M.D., Koh, T.J., Lundin, T.M. & Jahnigen, D.W. (1993). Kinematics of recovery from a stumble. *Journal of Gerontology* **48**, M97-M102.

Granek, E., Baker, S.P., Abbey, H., Robinson, E., Myers, A.H., Samkoff, J.S. & Klein, L.E. (1987). Medications and diagnoses in relation to falls in a long term care facility. *Journal of the American Geriatric Society*, **35**, 503-511.

Greig, C.A., Young, A., Skelton, D.A., Pippet, E., Butler, F.M.M. & Mahmud, S.M. (1994). Exercise studies with elderly volunteers. *Age and Ageing* **23**, 185-189.

Grimby, G., Danneskjold-Samsøe, B., Hvid, K & Saltin B. (1982). Morphology and enzymatic capacity in arm and leg muscles in 78-81 year old men and women. *Acta Physiologica Scandinavica* **115**, 125-134.

Grimby, G. & Saltin, B. (1983). The ageing muscle. *Clinical Physiology* **3**, 209-218.

Gryfe, C.I., Amies, A. & Ashley, M.J. (1977). A longitudinal study of falls in an elderly population: I. Incidence and morbidity. *Age and Ageing* **6**, 201-210.

Guimaraes, R.M & Isaacs, B. (1980). Characteristics of the gait in old people who fall. *International Rehabilitation Medicine* **2**, 177-180.

Gurfinkel, E.V. (1973). Physical foundations of stabilography. *Agressologie* **14C**, 9-13.

Gurfinkel, V.S. & Osevets, M. (1972). Dynamics of the vertical posture in man. *Biophysics* **17**, 496-506.

Gutmann, E. & Hanzlikova, V. (1972). Basic mechanisms of ageing in the neuromuscular system. *Mechanisms of Aging and Development* **1**, 327-349.

Hageman, P.A. & Blanke, D.J. (1986). Comparison of gait of young women and elderly women. *Physical Therapy* **66**, 1382-1387.

Hampson, E. (1990). Estrogen-related variations in human spatial and articulatory-motor skills. *Psychoneuroendocrinology,* **15**, 97-111.

Hampson, E. & Kimura, D. (1988) Reciprocal effects of hormonal fluctuations on the human motor and perceptual-spatial skills. *Behavioral Neuroscience* **102**, 456-459.

Harrison, M.J. & Marshall, J. (1984). Hypoperfusion in the aetiology of subcortical arteriosclerotic encephalopathy (Binswanger type). *Journal of Neurology, Neurosurgery and Psychiatry* **47**, 754.

Hasan, S.S., Robin, D.W., Szurkus, D.C., Ashmead, D.H., Peterson, S.W. & Shiavi, R.G. (1996). Simultaneous measurement of body center of pressure and center of gravity during upright stance. Part I: Methods. *Gait and Posture* **4**, 1-10.

Hasselkus, B.R. & Shambes, G.M. (1975). Aging and postural sway in women. *Journal of Gerontology* **30**, 661-667.

Hausdorff, J.M., Edelberg, H.K., Mitchell, S.L., Goldberger, A.L. & Wei, J.Y. (1997). Increased gait unsteadiness in community dwelling elderly fallers. *Archives of Physical Medicine and Rehabilitation* **78**, 278-283.

Hausdorff, J.M., Forman, D.E., Ladin, Z, Goldberger, A.L., Rigney, D.R. & Wei, J.Y. (1994). Increased walking variability in elderly persons with congestive heart failure. *Journal of the American Geriatric Society* **42**, 1056-1061.

Heitman, D.K., Gossman, M.R., Shaddeau, S.A. & Jackson, J.R. (1989). Balance performance and step width in noninstitutionalized, elderly, female fallers and nonfallers. *Physical Therapy* **69**, 923-931.

Hoehn M.M., Yahr, M.D. (1967). Parkinsonism: Onset, progression and mortality. *Neurology* **17**, 427-442.

Horak, F.B. & Nashner, L.M. (1986). Central programming of postural movements: adaptation to altered support surface configurations. *Journal of Neurophysiology* **55**, 1369-1381.

Horak, F.B., Nashner, L.M. & Diener, H.C. (1990). Postural strategies associated with somatosensory and vestibular loss. *Experimental Brain Research* **82**, 167-177.

Horak, F.B., Nutt, J. & Nashner, L.M. (1992). Postural inflexibility in parkinsonian subjects. *Journal of the Neurological Sciences* **111**, 46-58.

Horak, F.B., Shupert, C.L & Mirka, A. (1989). Components of postural dyscontrol in the elderly: A review. *Neurobiology of Ageing* **10**, 727-738.

Horsman, A., Gallagher, J.C., Simpson, M. & Nordin, B.E.C. (1977). Prospective trial of oestrogen and calcium in postmenopausal women. *British Medical Journal* **2**, 789-792.

Horsman, A., Jones, M., Francis, R. & Nordin, C. (1983). The effect of oestrogen dose on postmenopausal bone loss. *New England Journal of Medicine* **309**, 1405-1407.

Hortobagyi, T., Zheng, D., Weidner, M., Lambert, N.J., Westbrook, S. & Houmard, J.A. (1995). The influence of aging on muscle strength and muscle fiber characteristics with special reference to eccentric strength. *Journal of Gerontology* **50**, B399-B406.

Hughes, M.A., Duncan, P.W., Rose, D.K., Chandler, J.M. & Studenski, S.A. (1996). The relationship of postural sway to sensorimotor function, functional performance and disability in the elderly. *Archives of Physical Medicine and Rehabilitation* **77**, 567-572.

Hunter, I.W. & Kearney, R.E. (1981). Respiratory components of human postural sway. *Neuroscience Letters* **25**, 155-159.

Huxley, A.F. (1980). Reflections on muscle. pp 41-43. Liverpool: Liverpool University Press.

Imms, F.J. & Edholm, O.G. (1981). Studies of gait and mobility in the elderly. *Age and Ageing* **10**, 147-156.

Ivers, R.Q., Cumming, R.G., Mitchell, P. & Attebo, K. (1998). Visual impairment and falls in older adults: The Blue Mountains Eye Study. *Journal of the American Geriatric Society* **46**, 58-64.

Jakobsson, F., Borg, K. & Edström, L. (1990). Fibre-type composition, structure and cytoskeletal protein location of fibers in anterior tibial muscle. *Acta Neuropathologica* **80**, 459-468.

Jarnlo, G. & Thorngren, K. (1991). Standing balance in hip fracture patients. *Acta Orthopaedic Scandinavica* **62**, 427-434.

Jensen, G.F., Christiansen, C. & Transbϕl, I. (1982). Fracture frequency and bone preservation in postmenopausal women treated with estrogen. *Obstetrics & Gynecology* **60**, 493-496.

Judge, J.O., Davis, R.B. & Õunpuu, S. (1996). Step length reductions in advanced age: the role of ankle and hip kinetics. *Journal of Gerontology*, **51A**, M303-M312.

Kallman, D.A., Plato, C.C. & Tobin, J.D. (1990). The role of muscle loss in the age-related decline of grip strength: cross-sectional and longitudinal perspectives. *Journal of Gerontology* **45**, M82-M88.

Kaneko, M., Morimoto,Y. Kimura, M., Fuchimoto, K. & Fuchimoto, T. (1991). A kinematic analysis of walking and physical fitness testing in elderly women. *Canadian Journal of Sports Science* **16**, 223-228.

Kapteyn, T.S. (1973). Afterthought about the physics and mechanics of the postural sway. *Agressologie,* **14C**, 27-35.

Karlsson, A. & Lanshammar, H. (1997). Analysis of postural sway strategies using an inverted pendulum model and force plate data. *Gait and Posture* **5**, 198-203.

Kase, C.S., Norrving, B., Levine, S.R., Babikian, V.L., Chodosh, E.H., Wolf, P.A. & Welch, K.M. (1993). Cerebellar infarction: Clinical and anatomic observations in 66 cases, *Stroke,* **24**, 76-83.

Kaya, B.K., Krebs, D.E. and Riley, P.O. (1998). Dynamic stability in elders: Momentum control in locomotor ADL. *Journal of Gerontology* **53A**, M126-M134.

Kiel, D.P., Felson, D. T., Anderson, J.J., Wilson, P.W.F. & Moskowitz, M.A. (1987). Hip fracture and the use of estrogens in postmenopausal women. The Framingham Study. *New England Journal of Medicine* **317**, 1169-1174.

King, D.L. & Zatsiorsky, V.M. (1997). Extracting gravity line displacement from stabilographic recordings. *Gait and Posture* **6**, 27-38.

Kirtley, C., Whittle, M.W. & Jefferson, R.J. (1985). Influence of walking speed on gait parameters. *Journal of Biomedical Engineering* **7**, 282-288.

Klein, B.E., Klein, R. & Linton, K.L. (1992). Prevalence of age related lens opacities in a population: The Beaver Dam Eye Study. *Ophthalmology* **99**, 546-552.

Klitgaard, H., Bergmann, O., Betto, R., Salviati, G., Schiaffino, S., Clausen, T. & Saltin, B. (1990a). Co-existence of myosin heavy chain I and Ia isoforms in human skeletal muscle with endurance training. *Pflügers Archives* **416**, 470-2.

Klitgaard, H., Mantoni, M., Schiaffino, S., Ausoni, S., Gorza, L., Laurent-Winter, C., Schnohr, P. & Saltin, B. (1990b). Function, morphology and protein expression of ageing skeletal muscle: a cross-sectional study of elderly men with different training backgrounds. *Acta Physiologica Scandinavica* **140**, 41-54.

Klitgaard, H., Zhou, M., Schiaffino, S., Betto, R., Salviati, G. & Saltin, B. (1990c). Ageing alters the myosin heavy chain composition of single fibres from human skeletal muscle, *Acta Physiologica Scandinavica* **140**, 55-62.

Kornzweig, A.L. (1977). Visual loss in the elderly. *Hospital Practice* **12**, 51-59.

Kugelberg, E., Eklund, K. & Grimby, L. (1960). An electromyographic study of the nociceptive reflexes of the lower limb. Mechanism of the plantar responses. *Brain* **83**, 394-410.

Labadie, E., Awerbuch, G., Hamilton, R. & Rapesak, S. (1989). Falling and postural deficits due to acute unilateral basal ganglia lesions. *Archives of Neurology* **261**, 492-496.

Larson, E.B., Kukull, W.A., Buchner, D, Reifler, B.U. (1987). Adverse drug reactions associated with global cognitive impairment in elderly persons. *Annals of Internal Medicine*, **107**, 169-173.

Larsson, L. (1983). Histochemical characteristics of human skeletal muscle during aging. *Acta Physiologica Scandinavica* **117**, 469-471.

Larsson, L., Grimby, G. & Karlsson, J. (1979). Muscle strength and speed of movement in relation to age and muscle morphology. *Journal of Applied Physiology* **46**, 451-456.

Lestienne, F., Soechting, J. & Berthoz, A. (1977). Postural readjustment induced by linear motion of visual scenes. *Experimental Brain Research* **28**, 363-384.

Lexell, J., Taylor, C.C. & Sjostrom, M. (1988). What is the cause of ageing atrophy? Total number, size and proportion of different fiber types studied in whole vastus lateralis muscle from 15 to 83 year old men. *Journal of the Neurological Sciences* **84**, 275-294.

Lichtenstein, M.J., Shields, S.L., Shiavi, R. & Burger, M.C. (1988). Clinical determinants of biomechanics platform measures of balance in aged women. *Journal of the American Geriatric Society* **36**, 996-1002.

Lichtenstein, M.J., Burger, M.C., Shields, S.L. and Shiavi, R.G., 1990, Comparison of biomechanics platform measures of balance and videotaped measures of gait with a clinical mobility scale in elderly women. *Journal of Gerontology* **45**, M49-M54.

Lilley, J.M., Arie, T. & Chilvers, C.E.D. (1995). Accidents involving older people: a review of the literature. *Age and Ageing* 24, 346-365.

Lindsay, R., Hart, D.M., Aitken, J.M., MacDonald, E.B., Anderson, J & Clarke, A.C. (1976). Long-term prevention of post-menopausal osteoporosis by oestrogen: evidence for an increased bone mass after delayed onset of oestrogen treatment. *The Lancet* **1**, 1038-41.

Lipsitz, L. Jonsson, P., Kelly, M. & Koestner, J. (1991). Causes and correlates of recurrent falls in ambulatory frail elderly. *Journal of Gerontology* **46**, M114-M122.

Lombardi, V. & Piazzesi, G. (1990). The contractile response during steady lengthening of stimulated frog muscle fibres. *Journal of Physiology* **431**, 141-171.

Lord, S.R. & Castell, S. (1994). Physical activity program for older persons: Effect on balance, strength, neuromuscular control and reaction time. *Archives of Physical Medicine and Rehabilitation* **75**, 648-652.

Lord, S.R., Clark, R.D. & Webster, I.W. (1991). Postural stability and associated physiological factors in a population of aged persons. *Journal of Gerontology* **46**, M69-76.

Lord, S.R., Lloyd, D.G. & Li, S.K. (1996). Sensorimotor function, gait patterns and falls in community-dwelling women. *Age and Ageing* **25**, 292-299.

Lord, S.R., McClean, D. & Stathers, G. (1992). Physiological factors associated with injurious falls in older people living in the community. *Gerontology*, **38**, 338-396.

Lucy, S.D. & Hayes, K.C. (1985). Postural sway profiles: Normal subjects and subjects with cerebellar ataxia. *Physiotherapy Canada* **37**, 140-148.

Lundberg, A. (1979). Multisensory control of spinal reflex pathways. *Progress in Brain Research* **50**, 11-28.

Luukinen, H., Koski, K., Hiltunen, L. & Kivelä, S-L. (1994). Incidence rate of falls in an aged population in northern Finland. *Journal of Clinical Epidemiology* **47**, 843-850.

Luukinen, H., Koski, K., Laippala, P. & Kivelä, S. (1997). Factors predicting fractures during falling impacts among home-dwelling older adults. *Journal of the American Geriatric Society* **45**, 1302-1309.

MacKinnon, C.D. & Winter, D.A. (1993). Control of whole body balance in the frontal plane during human walking. *Journal of Biomechanics* **26**, 633-644.

MacLennan, W.J., Timothy, J.I & Hall, M.R.P. (1980). Vibration sense, proprioception and ankle reflexes in old age. *Journal of Clinical and Experimental Gerontology* **2**, 159-171.

Magnusson, M., Enbom, H., Johansson, R. & Wiklund J. (1990). Significance of pressor input from the human feet in lateral postural control. *Acta Otolaryngology* **110**, 321-327.

Maki, B.E. (1995). Direction and vision-dependence of postural responses in elderly 'fallers' and 'non-fallers'. *Facts and Research in Gerontology* **9**, 83-96.

Maki, B.E. (1997). Gait changes in older adults: Predictors of falls or indicators of fear? *Journal of the American Geriatric Society* **45**, 313-320.

Maki, B.E., Holliday, P.J. & Topper, A.K. (1994). A prospective study of postural balance and risk of falling in an ambulatory and independent elderly population. *Journal of Gerontology* **49**, M72-M84.

Manchester, D., Woollacott, M., Zederbauer-Hylton, N. & Marin, O. (1989). Visual, vestibular and somatosensory contributions to balance control in the older adult *Journal of Gerontology* **44**, M118-127.

Martin, W.E., Loewenson, R.B., Resch, J.A., Baker, A.B. (1973). Parkinson's disease. Clinical analysis of 100 patients. *Neurology* **23**, 783-790.

Masdeu, J., Alampur, U., Cavaliere, R. & Tavoulareas, G. (1994). Astasia and gait failure with damage of the pontomesencephalic locomotor region. *Annals of Neurology* **35**, 619-621.

Masdeu, J. & Gorelick, P. (1988). Inability to stand after unilateral thalamic lesions. *Annals of Neurology* **23**, 596-603.

Masdeu, J.C., Wolfson, L., Lantos, G., Tobin, J.N., Grober, E., Whipple, R. & Amerman, P. (1989). Brain white-matter changes in the elderly prone to falling. *Archives of Neurology* **46**, 1292-1296.

Mathias, S., Nayak, U.S.L. & Isaacs, B. (1986). Balance in elderly patients: The 'Get-up-and-go-test'. *Archives of Physical Medicine and Rehabilitation* **67**, 387-389.

Matsuoka, I., Takagi, A. & Masashi, S. (1986). Intoxication and postural control, In *Disorders of Posture and Gait*, ed. Bles, W. & Brandt, T., pp 269-285. Holland: Elsevier.

Maughan, R.J., Watson, J.S. & Weir, J. (1983). Strength and cross-sectional area of human skeletal muscle. *Journal of Physiology* **338**, 37-49.

Mauritz, K.H., Dichgans, J. & Hufschmidt, A. (1979). Quantitative analysis of stance in late cortical cerebellar atrophy of the anterior lobe and other forms of cerebellar ataxia. *Brain*, **102**, 461-486.

Mauritz, K.H. & Dietz, V. (1980). Characteristics of postural instability induced by ischemic blocking of leg afferents. *Experimental Brain Research* **38**, 117-119.

Mauritz, K.H., Dietz, V. & Haller, M. (1980). Balancing as a clinical test in the differential diagnosis of sensory motor disorders. *Journal of Neurology, Neurosurgery and Psychiatry* **43**, 407-412.

McClenaghan, B.A., Williams, H.G., Dickerson, J, Dowda, M, Thombs, L. & Eleazer, P. (1995). Spectral characteristics of ageing postural control. *Gait and Posture* **3**, 123-131.

McClenaghan, B.A., Williams, H.G., Dickerson, J., Dowda, M., Thombs, L. & Eleazer, P. (1996). Spectral characteristics of aging postural control, *Gait and Posture* **4**, 112-121.

McCormick, D.A. & Thompson, R.F. (1984). Cerebellum: essential involvement in the classically conditioned eyelid response. *Science* **223**, 296-299.

McGoldrick, T., Phillips, S.K., Bruce, S.A. & Woledge, R.C. (1998). Changes in isometric force of mouse soleus muscle during the oestrous cycle. *Pflügers Archives - European Journal of Physiology* **437**, 70-73.

Mills, M.E. & McIntyre, D. (1994). A simple and inexpensive device for use in the measurement of postural sway. *Journal of Physiology* **475.P**, 6P.

Mitchell, P., Smith, W., Attebo, K. & Wang, J.J. (1995). Prevalence of age-related maculopathy in Australia: The Blue Mountains Eye Study. *Ophthalmology* **103**, 357-364.

Moghissi, K.S., Syner, F.N. & Evans, T.N. (1972). A composite picture of the menstrual cycle. *American Journal of Obstetrics and Gynaecology* **114**, 405-418.

Mori, S. (1987). Integration of posture and locomotion in acute decerebrate cats and in awake cats. *Progress in Neurobiology* **28**, 161-195.

Morin, C., Katz, R., Mazieres, L. & Pierrot-Deseilligny, E. (1982). Comparison of soleus H-reflex facilitation at the onset of soleus contractions produced voluntarily and during the stance phase of human gait. *Neuroscience Letters* **33,** 47-53.

Murray, M.P., Clarkson, B.H. (1966). The vertical pathways of the foot during level walking. I: Range of variability in normal men. *Journal of the American Physical Therapy Association* **46**, 585-589.

Murray, M.P., Kory, R.C. & Clarkson, B.H. (1969). Walking patterns in healthy old men. *Journal of Gerontology* **24**, 169-178.

Murray, M.P., Sepic, S.B. & Barnard, E.J. (1967). Patterns of sagittal rotation of the upper limbs in walking. *Journal of the American Physical Therapy Association* **47**, 272-284.

Murray, M.P., Siereg, A.A. & Sepic, S.B. (1975). Normal postural stability and steadiness: quantitative assessment. *Journal of Bone and Joint Surgery* **57A**, 510-516.

Naessen, T., Lindmark, B. & Larsen, H. (1997). Better postural balance in elderly women receiving estrogens. *American Journal of Obstetrics and Gynaecology* **177**, 412-416.

Naessen, T., Persson, I., Adami, H.O., Bergstrom, R. & Bergkvist, L. (1990). Hormone replacement therapy and the risk for first hip fracture: a prospective, population-based cohort study. *Annals of Internal Medicine* **113**, 95-103.

Nashner, L.M. (1980). Balance adjustments of humans perturbed while walking. *Journal of Neurophysiology* **44**, 650-664.

Nashner, L.M. (1981). Analysis of stance posture in humans. In *Handbook of Behavioural Neurobiology, Motor Co-ordination,* ed. Towe, A.L. & Luschei, E.S., pp 527-565. New York: Plenum Press.

Nashner, L.M., Black, F.O. & Wall, C. (1982). Adaptation to altered support and visual conditions during stance: Patients with vestibular deficits. *Journal of Neuroscience* **2**, 536-544.

Nashner, L.M. & McCollum, G. (1985). The organization of human postural movements: A formal basis and experimental synthesis. *The Behavioral and Brain Sciences* **8**, 135-172.

Nashner, L.M., Shupert, C.L., Horak, F.B. & Black, F.O. (1989). Organization of posture controls: an analysis of sensory and mechanical constraints. *Progress in Brain Research,* **80**, 411-423.

Nevitt, M.C., Cummings, S.R., Hudes, E.S. (1991). Risk factors for injurious falls: A prospective study. *Journal of Gerontology* **46**, M164-170.

Nevitt, M.C., Cummings, S.R., Kidd, S. & Black, D. (1989). Risk factors for recurrent nonsyncopal falls: a prospective study. *Journal of the American Medical Association,* **261**, 2663-2668.

Oberg, T., Karsznia, A. & Oberg, K. (1994). Joint angle parameters in gait: Reference data for normal subjects, 10-79 years of age. *Journal of Rehabilitation, Research & Development* **31**, 199-213.

Okubo, J., Watanabe, I., & Baron, J.B. (1980). Study on the influences of the plantar mechanoreceptor on body sways. *Agressologie* **21**, 61-69.

Overstall, P.W., Exton-Smith, A.N., Imms, F.J. & Johnson, A.L. (1977). Falls in the elderly related to postural imbalance. *British Medical Journal* **1**, 261-264.

Owen, D.H. (1985). Maintaining posture and avoiding tripping. Optical information for detecting and controlling orientation and locomotion. *Clinics in Geriatric Medicine* **1**, 581-599.

Pack, D.R., Wolfson, L.I., Amerman, P, Whipple, R. & Kaplan, J.G. (1985). Peripheral nerve abnormalities and falling in the elderly. *Neurology* **35**, 79.

Pate, E. & Cooke, R. (1989). A model of crossbridge action: the effects of ATP, ADP and Pi. *Journal of Muscle Research and Cell Motility* **10**, 181-196.

Patla, A.E. (1997). Understanding the roles of vision in the control of human locomotion. *Gait and Posture* **5**, 54-69.

Patla, A.E., Prentice, S.D., Robinson, C. & Neufeld, J. (1991). Visual control of locomotion: Strategies for changing direction and for going over obstacles. *Journal of Experimental Psychology: Human Perception and Performance* **17**, 603-634.

Paulus, W., Straube, A. & Brandt, T. (1987). Visual postural performance after loss of somatosensory and vestibular function. *Journal of Neurology, Neurosurgery and Psychiatry* **50**, 1542-1545.

Perez, J., Zucchi, I. & Maggi, A. (1986). Sexual dimorphism in the response of the GABA-ergic system to estrogen administration. *Journal of Neurochemistry* **47**, 1798-1803.

Peterka, R.J. & Black, F.O. (1990). Age-related changes in human posture control: Sensory organization tests. *Journal of Vestibular Research* **1**, 73-85.

Phillips, S.K., Bruce, S.A., Newton, D. & Woledge, R.C. (1992). The weakness of old age is not due to failure of muscle activation. *Journal of Gerontology* **47**, M45-M49.

Phillips, S.K., Bruce, S.A. & Woledge, R.C. (1991). The muscle weakness due to age is absent during stretching. *Journal of Physiology* **437**, 63-70.

Phillips, S.K., Gopinathan, J., Meehan, K., Bruce, S.A. & Woledge, R.C. (1993a). Muscle strength changes during the menstrual cycle in human adductor pollicis. *Journal of Physiology* **473**, 125P.

Phillips, S.K., Rook, K., Siddle, N, Bruce, S.A. & Woledge R.C. (1993). Muscle weakness in women occurs at an earlier age than in men, but strength is preserved by hormone replacement therapy. *Clinical Science* **84**, 95-98.

Phillips, S.K., Rowbury, J.L., Bruce, S.A. & Woledge, R.C. (1993c). Muscle force generation and age: the role of sex hormones. In. *Sensorimotor impairment in the elderly,* ed. Stelmach, G.E. & Homberg, V., pp 129-141. Netherlands: Kluwer.

Phillips, S.K., Woledge, R.C., Bruce, S.A., Young, A., Levy, D., Yeo, A. & Martin, F.C. (1998). A study of force and cross-sectional area of adductor pollicis muscle in female hip fracture patients. *Journal of the American Geriatric Society* **46**, 999-1002.

Podsiadlo, D. & Richardson, S. (1991). The timed 'Up and Go': A test of basic functional mobility for frail elderly persons. *Journal of the American Geriatric Society* **39**, 142-148.

Poulin, M.J., Vandervoort, A.A., Paterson, D.H, Kramer, J.F. & Cunningham, D.A. (1992). Eccentric and concentric torques of knee and elbow extension in young and older men. *Canadian Journal of Sport Science* **17**, 3-7.

Prudham, D. & Grimley Evans, J. (1981). Factors associated with falls in old age. *British Medical Journal* **4**, 1685-1690.

Quoniam, C., Hay, L., Roll, JP. & Harlay, F. (1995). Age effects on reflex and postural responses to propriomuscular inputs generated by tendon vibration. *Journal of Gerontology* **50A**, B155-B165.

Ray, W.A., Griffin, M.R., Schaffner, W., Baugh, D.K. & Melton, I.J. (1987). Psychotropic drug use and the risk of hip fracture. *New England Journal of Medicine*, **316**, 363-369.

Riley, P.O., Mann, R.W. & Hodge, W.A. (1990). Modelling of the biomechanics of posture and balance. *Journal of Biomechanics* **23**, 503-506.

Ring, C., Nayak, U.S.L. & Isaacs, B. (1989). The effect of visual deprivation and proprioceptive change on postural sway in healthy adults. *Journal of the American Geriatric Society* **37**, 745-749.

Robbins, A.S., Rubenstein, L.Z., Josephson, K.R., Schulman, B.L., Osterweil, D. & Fine, G. (1989). Predictors of falls among elderly people. Results of two population-based studies. *Archives of Internal Medicine*, **149**, 1628-1633.

Robbins, S., Waked, E., McClaren, J. & Krouglicof, N. (1997). Foot position awareness in younger and older men: The influence of footwear sole properties. *Journal of the American Geriatrics Society* **45**, 61-66.

Roll, J.P., Hay, L., Quoniam, C. & Roll, R. (1992). Muscle proprioception: A powerful sensory input for postural adaptation in man. In: *Falls, Balance and Gait disorders in the elderly.* ed. *Vellas, B. et al.* Paris: Elsevier.

Romberg, M.H. (1853). Manual of Nervous diseases of Man. Sydenham Society, London, 395-482.

Rosenhall, U. & Rubin, W. (1975). Degenerative changes in the human vestibular sensory epithelia. *Acta Otolaryngology (Stockholm)* **79**, 67-80.

Rossignol, S. (1975). Startle responses recorded in the leg of man. *Electroencephalography and Clinical Neurophysiology* **39**, 389-397.

Round, J.M, Jones, D.A., Chapman, S.J., Edwards, R.H.T., Ward, P.S., & Fodder, D.L. (1984). The anatomy and fibre type composition of the human adductor pollicis in relation to its contractile properties. *Journal of the Neurological Sciences* **66**, 263-272.

Rubenstein, L.Z., Robbins, A.S., Shulman, B.L., Rosado, J., Osterweil, D. & Josephson, K.R. (1988). Falls and instability in the elderly. *Journal of the American Geriatrics Society* **36**, 266-278.

Ruder, G.K., MacKinnon, C.D. & Winter, D.A. (1989). C of P vs C of G changes during quiet stance and voluntary sway in the frontal and sagittal planes. In: *Proceedings of the nineteenth Conference of the International Society of Biomechanics,* Los Angeles.

Rutherford, O.M. & Jones, D.A. (1992). The relationship of muscle and bone loss and activity levels with age in women. *Age and Ageing* **21**, 286-293.

Sabin, T.D. (1997). Peripheral neuropathy: disorders of proprioception. In: *Gait Disorders of Aging, Falls and Therapeutic Strategies,* ed. Masdeu, J.C., Sudarsky, L. and Wolfson, L. Philadelphia: Lippincott-Raven.

Saunders, J.B., Inman, V.T. & Eberhart, H.D. (1953). The major determinants in normal and pathological gait. *Journal of Bone and Joint Surgery* **35A**, 543-558.

Scheibel, A.B. (1985). Falls, motor dysfunction and correlative neurohistologic changes in the elderly. *Clinics in Geriatric Medicine* **1**, 671-676.

Sekuler, R, Hutman, L.P. & Owsley, C.J. (1980). Human ageing and spatial vision. *Science*, **209**, 1255-6.

Sheldon, J.H. (1960). On the natural history of falls in old age. *British Medical Journal*, **2**, 1685-1690.

Sheldon, J.H. (1963). The effect of age on the control of sway. *Gerontology Clinics* **5**, 129-138.

Shimba, T. (1984). An estimation of centre of gravity from force platform data. *Journal of Biomechanics* **17**, 53-60.

Sixt, E. & Landahl, S. (1987). Postural disturbances in a 75-year-old population. I. Prevalence and functional consequences. *Age and Ageing* **16**, 393-398.

Smallegan, M. (1983). How families decide on nursing-home admission. *Geriatr. Consult.* **2**, 21-24.

Smith, S.S. (1989). Estrogen administration increases neuronal responses to excitatory amino acids as a long-term effect. *Brain Research* **503**, 354-357.

Smith, J.W. (1957). The forces operating at the human ankle joint during standing. *Journal of Anatomy* **91**, 545-564.

Soames, R.W. & Atha, J. (1980). The validity of physique-based inverted pendulum models of postural sway behaviour. *Annals of Human Biology* **7**, 145-153.

Sorock, G.S. & Shimkin, E.E. (1988). Benzodiazepine sedatives and the risk of falling in a community-dwelling elderly cohort. *Archives of Internal Medicine*, **148**, 2441-2444.

Stoffregen, T.A., Adolph, K., Thelen, E., Gorday, K.M. & Sheng, Y. (1997). Toddlers' postural adaptations to different support surfaces. *Motor Control* **1**, 119-137.

Thomas, D.P. & Whitney, R.J. (1959). Postural movements during normal standing in man. *Journal of Anatomy* **93**, 524-539.

Thompson, P.D. & Marsden, C.D. (1987). Gait disorder of subcortical arteriosclerotic encephalopathy: Binswangers disease. *Movement Disorders* **2**, 1-8.

Tinetti, M.E. (1986). Performance-oriented assessment of mobility problems in elderly patients. *Journal of the American Geriatric Society* **34**, 119-126.

Tinetti, M.E. (1987). Factors associated with serious injury during falls by ambulatory nursing home residents. *Journal of the American Geriatric Society* **35**, 644-648.

Tinetti, M.E. Baker, D.I., McAvay, G., Claus, E.B., Garrett, P., Gottschalk, M., Koch, M.L., Trainor, K. & Horwitz, R.I. (1994). A multifactorial intervention to reduce the risk of falling among elderly people living in the community. *New England Journal of Medicine* **331**, 821-827.

Tinetti, M.E. & Speechley, M. (1989). Prevention of falls among the elderly. *New England Journal of Medicine* **320**, 1055-1059.

Tinetti, M.E., Speechley, M. & Ginter, S.F. (1988). Risk factors for falls among elderly persons living in the community. *New England Journal of Medicine* **319**, 1701-1707.

Tinetti, M.E., Williams, T.F. & Mayewski, R. (1986). Fall risk index for elderly patients based on the number of chronic disabilities. *American Journal of Medicine* **80**, 429-434.

Topper, A.K. & Holliday, P.J. (1993). Are activity based assessments of balance and gait in the elderly predictive of risk of falling and/or type of fall? *Journal of the American Geriatric Society* **41**, 480-487.

Vandervoort, A.A., & McComas, A.J. (1986). Contractile changes in opposing muscles of the human ankle joint with aging. *Journal of Applied Physiology* **61**, 361-367.

Vandervoort, A.A., Kramer, J.F. & Wharram, E.R. (1990). Eccentric knee strength of elderly females. *Journal of Gerontology* **45**, B125-128.

Walter, J.G. (1955). The effect of salicylates on the inner ear. *Annals of Otorhinolaryngology* **64**, 617-622.

Whipple, R.H., Wolfson, L.I & Amerman, P.M. (1987). The relationship of knee and ankle weakness to falls in nursing home residents: an isokinetic study. *Journal of the American Geriatric Society* **35**, 13-20.

Whitehead, M.I. & Godfree, V. (1994). Hormone Replacement Therapy: Your questions answered. Edinburgh: Churchill Livingstone.

Wild, D., Nayak, U.S.L., Isaacs, B. (1981). Prognosis of falls in old people at home. *Journal of Epidemiology and Community Health* **35**, 200-204.

Williams, H.G., McClenaghan, B.A. & Dickerson, J. (1997). Spectral characteristics of postural control in elderly individuals. *Archives of Physical Medicine and Rehabilitation* **78**, 737-744.

Winter, D.A. (1991). *The Biomechanics and Motor Control of Human Gait: Normal, Elderly and Pathological*, 2nd edition. Waterloo: University of Waterloo Press.

Winter, D.A. (1992). Foot trajectory in human gait: A precise and multifactorial motor control task. *Physical Therapy* **72**, 45-56.

Winter, D.A. (1995). A.B.C. (Anatomy, Biomechanics and Control) of balance during standing and walking, Waterloo: Waterloo Biomechanics.

Winter, D.A., Patla, A.E., Frank, J.S. & Walt, S.E. (1990). Biomechanical walking pattern changes in the fit and healthy elderly. *Physical Therapy* **70**, 340-347.

Winter, D.A., Prince, F., Stergiou, P. and Powell, C. (1993a). Medial-lateral and anterior-posterior motor responses associated with centre of pressure changes in quiet standing, *Neuroscience Research Communications* **12**, 141-148.

Winter, D.A., MacKinnon, C.D., Ruder, G.K. and Wieman, C. (1993b). An integrated EMG/biomechanical model of upper body balance and posture during human gait, *Progress in Brain Research* **97**, 359-367.

Winter, D.A., Prince, F. and Patla, A. (1997). Validity of the inverted pendulum model of balance in quiet standing *Gait and Posture* **5,** 153.

Wolfson, LI., Whipple, R., Amerman,P. and Tobin, J.N. (1990). Gait assessment in the elderly: A gait abnormality rating scale and its relation to falls. *Journal of Gerontology,* **45,** M12-19.

Wolfson, L., Whipple, R., Derby, C.A., Amerman, P., Murphy, T., Tobin, J.N. & Nashner, L. (1992). A dynamic posturography study of balance in healthy elderly. *Neurology* **42,** 2069-2075.

Woollacott, M.H., Shumway-Cook, A. and Nashner, L.M. (1986). Aging and posture control: changes in sensory organization and muscular coordination, *International Journal of Aging and Human Development* **23**, 97-114.

Yack, H.J. and Berger, R.C. (1993). Dynamic stability in the elderly: Identifying a possible measure. *Journal of Gerontology* **48**, M225-M230

Yang, J.F. and Stein, R.B. (1990). Phase-dependent reflex reversal in human leg muscles during walking. *Journal of Neurophysiology* **63**, 1109-1117.

Yoshimoto, Y. (1978). A case of epithelium intoxication with vertical beat nystagmus. *Practical Otolaryngology (Kyoto)* **71**, 435-440.

Young, A., Stokes, M. and Crowe, M. (1985). The size and strength of the quadriceps muscles of old and young men. *Clinical Physiology* **5**, 145-154.

Zatsiorsky, V. and Seluyanov, V. (1983). The mass and inertia characteristics of the main segments of the human body, In: *Biomechanics VIII-B,* ed. H. Matsui, H. & K. Kobayashi, K., pp 1152-1159. Champaign, Illinois: Human Kinetics.

# *Appendix A*

**Table A1:** A summary of individual performances on the balance tests, rank for each test, the mean balance score (mean of rank on the three tests) and overall rank of the GBA, BI, and SBI elderly subgroups. For the tandem gait test, performances are graded by the experimenter with either a '2', able to walk 10 steps heel-to-toe in a confident manner, '1', able to walk between 2 and 10 steps heel-to-toe before taking a side step or '0', unable to walk more than 2 steps heel-to-toe before taking a side step. The one-legged stance (OLS) and foam eyes closed balance test scores are the time in seconds the subject was able to maintain their balance before returning the suspended foot to the floor (OLS) or opening their eyes and requiring support (FEC).

| Subject | Sex | Class | Test Score | | | Rank | | | | **Overall** |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | Tandem | OLS | FEC | Tandem | OLS | FEC | Mean | **Rank** |
| 1 | Male | GBA | 2 | 30 | 90 | 1 | 1 | 1 | 1 | **1** |
| 2 | Male | GBA | 2 | 30 | 90 | 1 | 1 | 1 | 1 | **2** |
| 3 | Female | GBA | 2 | 30 | 90 | 1 | 1 | 1 | 1 | **3** |
| 4 | Female | GBA | 2 | 30 | 90 | 1 | 1 | 1 | 1 | **4** |
| 5 | Female | GBA | 2 | 30 | 90 | 1 | 1 | 1 | 1 | **5** |
| 6 | Male | GBA | 2 | 30 | 90 | 1 | 1 | 1 | 1 | **6** |
| 7 | Female | BI | 1 | 30 | 90 | 10 | 1 | 1 | 4 | **7** |
| 8 | Male | BI | 2 | 20 | 90 | 1 | 11 | 1 | 4.3 | **8** |
| 9 | Female | BI | 2 | 15 | 90 | 1 | 14 | 1 | 5.3 | **9** |
| 10 | Male | BI | 2 | 30 | 0 | 1 | 1 | 14 | 5.3 | **10** |
| 11 | Male | BI | 1 | 29 | 90 | 10 | 9 | 1 | 6.7 | **11** |
| 12 | Female | BI | 1 | 20 | 90 | 10 | 11 | 1 | 7.3 | **12** |
| 13 | Male | BI | 1 | 23 | 60 | 10 | 10 | 12 | 10.7 | **13** |
| 14 | Female | BI | 1 | 16 | 15 | 10 | 13 | 13 | 12 | **14** |
| 15 | Male | BI | 1 | 10 | 0 | 10 | 15 | 14 | 13 | **15** |
| 16 | Male | BI | 1 | 5 | 0 | 10 | 16 | 14 | 13.3 | **16** |
| 17 | Male | SBI | 1 | 0 | 0 | 10 | 17 | 14 | 13.7 | **17** |
| 18 | Male | SBI | 0 | 0 | 0 | 18 | 17 | 14 | 16.3 | **18** |

**Table A2:** Summary of the correlation coefficients calculated for pelvis-knee, pelvis-head, shoulder-head, shoulder-knee and head-knee movements during quiet stance, eyes closed trials, for the young (n = 20) and elderly (n = 22). Mean ± S.E.M. calculated from transformed data. Also shown are the results of a two-tailed *t*-test (d.f. = 40).

| SEGMENT | AXIS | GEOMETRIC MEAN (± S.E.M.) | | *P* |
|---|---|---|---|---|
| | | Young | Elderly | Young = Elderly |
| Pelvis-Knee | A/P | 0.86 (0.83, 0.88) | 0.84 (0.81, 0.87) | 0.638 |
| | M/L | 0.97 (0.97, 0.98) | 0.97 (0.97, 0.98) | 0.604 |
| Pelvis-Head | A/P | 0.85 (0.83, 0.88) | 0.85 (0.83, 0.87) | 0.966 |
| | M/L | 0.81 (0.79, 0.83) | 0.91 (0.90, 0.93) | <0.001# |
| Shoulder-Head | A/P | 0.96 (0.96, 0.97) | 0.97 (0.97, 0.98) | 0.178 |
| | M/L | 0.95 (0.94, 0.96) | 0.97 (0.97, 0.98) | 0.004** |
| Shoulder-Knee | A/P | 0.88 (0.86, 0.90) | 0.81 (0.77, 0.84) | 0.086 |
| | M/L | 0.89 (0.87, 0.91) | 0.94 (0.93, 0.95) | 0.031* |
| Head-Knee | A/P | 0.84 (0.82, 0.86) | 0.75 (0.71, 0.78) | 0.026* |
| | M/L | 0.79 (0.76, 0.82) | 0.89 (0.87, 0.90) | 0.003** |

*, ** , # denotes significant difference, P < 0.05, 0.01, 0.001 respectively

**Table A3:** Inertial characteristics of elderly subjects in the frontal axis

| Subject No. | Sex | Height (m) | Weight (kg) | Estimated Segment Moments of Inertia (kg $m^2$) | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | head/neck | upper arm | forearm | hand | thigh | lower leg | lower torso | mid torso | upper torso | **Total** |
| 1 | M | 1.653 | 66.8 | 11.21 | 4.93 | 1.79 | 0.38 | 8.15 | 0.38 | 4.78 | 11.36 | 15.81 | **58.80** |
| 2 | M | 1.707 | 72.8 | 12.40 | 5.73 | 2.05 | 0.43 | 9.57 | 0.45 | 5.71 | 13.17 | 18.04 | **67.54** |
| 3 | M | 1.796 | 82.0 | 14.51 | 7.16 | 2.50 | 0.53 | 12.09 | 0.56 | 7.38 | 16.33 | 21.93 | **83.01** |
| 4 | M | 1.760 | 95.0 | 14.39 | 8.11 | 2.76 | 0.55 | 13.45 | 0.61 | 7.93 | 19.38 | 25.49 | **92.67** |
| 5 | M | 1.626 | 63.5 | 10.63 | 4.52 | 1.66 | 0.35 | 7.44 | 0.34 | 4.33 | 10.44 | 14.67 | **54.38** |
| 6 | F | 1.677 | 60.6 | 11.37 | 4.53 | 1.68 | 0.37 | 7.60 | 0.36 | 4.58 | 10.05 | 14.30 | **54.85** |
| 7 | M | 1.768 | 78.2 | 13.78 | 6.61 | 2.33 | 0.49 | 11.13 | 0.52 | 6.76 | 15.06 | 20.38 | **77.06** |
| 8 | M | 1.625 | 74.7 | 11.05 | 5.41 | 1.91 | 0.39 | 8.83 | 0.40 | 5.04 | 12.94 | 17.64 | **63.61** |
| 9 | M | 1.815 | 72.9 | 14.46 | 6.40 | 2.29 | 0.50 | 10.97 | 0.53 | 6.89 | 13.98 | 19.19 | **75.20** |
| 10 | F | 1.495 | 55.7 | 8.38 | 3.38 | 1.27 | 0.26 | 5.34 | 0.23 | 2.89 | 8.13 | 11.70 | **41.59** |
| 11 | F | 1.610 | 58.0 | 10.17 | 4.02 | 1.51 | 0.32 | 6.61 | 0.31 | 3.85 | 9.15 | 13.11 | **49.05** |
| 12 | F | 1.574 | 62.5 | 9.77 | 4.20 | 1.54 | 0.32 | 6.81 | 0.31 | 3.84 | 9.92 | 13.97 | **50.68** |
| 13 | F | 1.612 | 70.0 | 10.66 | 4.96 | 1.78 | 0.36 | 8.10 | 0.37 | 4.62 | 11.79 | 16.25 | **58.90** |
| 14 | F | 1.612 | 59.0 | 10.24 | 4.11 | 1.53 | 0.33 | 6.75 | 0.31 | 3.93 | 9.38 | 13.39 | **49.97** |
| 15 | M | 1.771 | 70.9 | 13.51 | 5.94 | 2.14 | 0.46 | 10.11 | 0.48 | 6.24 | 13.16 | 18.14 | **70.18** |
| 16 | F | 1.540 | 55.5 | 9.02 | 3.55 | 1.34 | 0.28 | 5.70 | 0.26 | 3.18 | 8.30 | 11.98 | **43.60** |
| 17 | M | 1.662 | 68.6 | 11.44 | 5.12 | 1.85 | 0.39 | 8.48 | 0.39 | 4.99 | 11.84 | 16.39 | **60.90** |
| 18 | M | 1.622 | 67.5 | 10.72 | 4.82 | 1.74 | 0.36 | 7.90 | 0.36 | 4.55 | 11.31 | 15.70 | **57.48** |
| 19 | M | 1.668 | 68.0 | 11.52 | 5.11 | 1.85 | 0.39 | 8.47 | 0.39 | 5.00 | 11.74 | 16.29 | **60.76** |
| 20 | F | 1.596 | 56.0 | 9.87 | 3.81 | 1.44 | 0.31 | 6.24 | 0.29 | 3.62 | 8.65 | 12.50 | **46.73** |
| 21 | M | 1.805 | 89.5 | 15.05 | 7.96 | 2.74 | 0.57 | 13.36 | 0.62 | 8.08 | 18.48 | 24.48 | **91.33** |
| 22 | F | 1.558 | 48.5 | 9.03 | 3.11 | 1.22 | 0.27 | 5.05 | 0.24 | 2.90 | 6.95 | 10.41 | **39.16** |

**Table A4:** Inertial characteristics of elderly subjects in the transverse axis

| Subject No. | Sex | Height (m) | Weight (kg) | Estimated Segment Moments of Inertia (kg $m^2$) | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | head/neck | upper arm | forearm | hand | thigh | lower leg | lower torso | mid torso | upper torso | **Total** |
| 1 | M | 1.653 | 66.8 | 11.21 | 4.93 | 1.79 | 0.37 | 8.15 | 0.37 | 4.77 | 11.32 | 15.72 | **58.65** |
| 2 | M | 1.707 | 72.8 | 12.40 | 5.73 | 2.04 | 0.43 | 9.57 | 0.44 | 5.70 | 13.12 | 17.94 | **67.37** |
| 3 | M | 1.796 | 82.0 | 14.52 | 7.16 | 2.50 | 0.53 | 12.10 | 0.56 | 7.37 | 16.28 | 21.81 | **82.81** |
| 4 | M | 1.760 | 95.0 | 14.39 | 8.11 | 2.76 | 0.55 | 13.45 | 0.60 | 7.91 | 19.31 | 25.35 | **92.44** |
| 5 | M | 1.626 | 63.5 | 10.64 | 4.52 | 1.66 | 0.35 | 7.44 | 0.34 | 4.32 | 10.40 | 14.58 | **54.24** |
| 6 | F | 1.677 | 60.6 | 11.37 | 4.53 | 1.68 | 0.37 | 7.60 | 0.36 | 4.57 | 10.02 | 14.22 | **54.72** |
| 7 | M | 1.768 | 78.2 | 13.79 | 6.60 | 2.33 | 0.49 | 11.13 | 0.52 | 6.75 | 15.01 | 20.26 | **76.88** |
| 8 | M | 1.625 | 74.7 | 11.05 | 5.40 | 1.91 | 0.39 | 8.83 | 0.40 | 5.03 | 12.89 | 17.54 | **63.44** |
| 9 | M | 1.815 | 72.9 | 14.46 | 6.39 | 2.29 | 0.50 | 10.97 | 0.52 | 6.87 | 13.94 | 19.08 | **75.03** |
| 10 | F | 1.495 | 55.7 | 8.38 | 3.38 | 1.27 | 0.26 | 5.34 | 0.23 | 2.88 | 8.09 | 11.63 | **41.47** |
| 11 | F | 1.610 | 58.0 | 10.17 | 4.02 | 1.51 | 0.32 | 6.61 | 0.30 | 3.85 | 9.11 | 13.04 | **48.93** |
| 12 | F | 1.574 | 62.5 | 9.78 | 4.20 | 1.54 | 0.32 | 6.81 | 0.30 | 3.84 | 9.88 | 13.88 | **50.55** |
| 13 | F | 1.612 | 70.0 | 10.66 | 4.96 | 1.78 | 0.36 | 8.10 | 0.36 | 4.62 | 11.74 | 16.16 | **58.74** |
| 14 | F | 1.612 | 59.0 | 10.24 | 4.11 | 1.53 | 0.33 | 6.75 | 0.31 | 3.92 | 9.34 | 13.31 | **49.84** |
| 15 | M | 1.771 | 70.9 | 13.51 | 5.94 | 2.14 | 0.46 | 10.11 | 0.48 | 6.23 | 13.12 | 18.04 | **70.02** |
| 16 | F | 1.540 | 55.5 | 9.02 | 3.54 | 1.34 | 0.28 | 5.69 | 0.25 | 3.18 | 8.27 | 11.91 | **43.49** |
| 17 | M | 1.662 | 68.6 | 11.44 | 5.12 | 1.85 | 0.39 | 8.48 | 0.39 | 4.98 | 11.80 | 16.30 | **60.75** |
| 18 | M | 1.622 | 67.5 | 10.73 | 4.82 | 1.74 | 0.36 | 7.90 | 0.36 | 4.55 | 11.27 | 15.61 | **57.33** |
| 19 | M | 1.668 | 68.0 | 11.52 | 5.10 | 1.85 | 0.39 | 8.47 | 0.39 | 4.99 | 11.70 | 16.19 | **60.61** |
| 20 | F | 1.596 | 56.0 | 9.88 | 3.81 | 1.44 | 0.31 | 6.24 | 0.29 | 3.61 | 8.62 | 12.43 | **46.62** |
| 21 | M | 1.805 | 89.5 | 15.06 | 7.95 | 2.73 | 0.56 | 13.37 | 0.61 | 8.06 | 18.41 | 24.35 | **91.11** |
| 22 | F | 1.558 | 48.5 | 9.03 | 3.11 | 1.22 | 0.26 | 5.04 | 0.23 | 2.89 | 6.92 | 10.35 | **39.07** |

**Table A5:** Inertial characteristics of young subjects in the frontal axis

| Subject No. | Sex | Height (m) | Weight (kg) | Estimated Segment Moments of Inertia (kg $m^2$) | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | head/neck | upper arm | forearm | hand | thigh | lower leg | lower torso | mid torso | upper torso | **Total** |
| 1 | F | 1.750 | 58.0 | 12.53 | 4.64 | 1.75 | 0.40 | 8.00 | 0.40 | 5.07 | 9.70 | 14.02 | **56.50** |
| 2 | F | 1.750 | 59.0 | 12.58 | 4.73 | 1.78 | 0.40 | 8.14 | 0.40 | 5.14 | 9.95 | 14.32 | **57.45** |
| 3 | F | 1.600 | 56.7 | 9.96 | 3.88 | 1.46 | 0.31 | 6.36 | 0.30 | 3.69 | 8.82 | 12.70 | **47.49** |
| 4 | M | 1.875 | 87.0 | 16.41 | 8.26 | 2.86 | 0.61 | 14.10 | 0.66 | 8.80 | 18.55 | 24.67 | **94.91** |
| 5 | M | 1.800 | 70.0 | 14.03 | 6.03 | 2.18 | 0.48 | 10.34 | 0.50 | 6.49 | 13.10 | 18.12 | **71.27** |
| 6 | M | 1.796 | 75.7 | 14.22 | 6.55 | 2.33 | 0.50 | 11.15 | 0.53 | 6.89 | 14.62 | 19.91 | **76.70** |
| 7 | F | 1.602 | 63.0 | 10.23 | 4.37 | 1.60 | 0.33 | 7.14 | 0.33 | 4.10 | 10.20 | 14.34 | **52.65** |
| 8 | M | 1.762 | 76.0 | 13.57 | 6.36 | 2.25 | 0.48 | 10.73 | 0.50 | 6.53 | 14.43 | 19.62 | **74.49** |
| 9 | F | 1.610 | 62.5 | 10.34 | 4.37 | 1.61 | 0.34 | 7.16 | 0.33 | 4.13 | 10.13 | 14.28 | **52.69** |
| 10 | F | 1.620 | 45.0 | 9.83 | 3.05 | 1.23 | 0.28 | 5.10 | 0.25 | 3.11 | 6.32 | 9.79 | **38.95** |
| 11 | F | 1.704 | 66.5 | 12.08 | 5.17 | 1.88 | 0.40 | 8.68 | 0.41 | 5.24 | 11.61 | 16.20 | **61.68** |
| 12 | M | 1.794 | 84.5 | 14.59 | 7.39 | 2.57 | 0.54 | 12.44 | 0.58 | 7.55 | 16.99 | 22.70 | **85.34** |
| 13 | F | 1.679 | 57.5 | 11.27 | 4.28 | 1.61 | 0.36 | 7.21 | 0.35 | 4.39 | 9.32 | 13.45 | **52.24** |
| 14 | F | 1.678 | 70.5 | 11.79 | 5.37 | 1.93 | 0.40 | 8.92 | 0.41 | 5.27 | 12.41 | 17.09 | **63.58** |
| 15 | F | 1.600 | 48.0 | 9.64 | 3.21 | 1.27 | 0.28 | 5.31 | 0.26 | 3.15 | 6.94 | 10.48 | **40.55** |
| 16 | F | 1.659 | 53.0 | 10.76 | 3.82 | 1.47 | 0.33 | 6.44 | 0.31 | 3.92 | 8.20 | 12.09 | **47.34** |
| 17 | M | 1.761 | 61.0 | 12.87 | 4.97 | 1.85 | 0.42 | 8.55 | 0.42 | 5.40 | 10.51 | 15.00 | **59.98** |
| 18 | M | 1.731 | 73.0 | 12.85 | 5.90 | 2.10 | 0.45 | 9.90 | 0.46 | 5.97 | 13.41 | 18.37 | **69.43** |
| 19 | M | 1.760 | 98.0 | 14.53 | 8.39 | 2.84 | 0.57 | 13.89 | 0.62 | 8.15 | 20.17 | 26.42 | **95.58** |
| 20 | M | 1.782 | 66.0 | 13.49 | 5.54 | 2.03 | 0.45 | 9.52 | 0.46 | 5.99 | 11.92 | 16.70 | **66.11** |

**Table A6:** Inertial characteristics of young subjects in the transverse axis

| Subject No. | Sex | Height (m) | Weight (kg) | Estimated Segment Moments of Inertia (kg $m^2$) | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | head/neck | upper arm | forearm | hand | thigh | lower leg | lower torso | mid torso | upper torso | **Total** |
| 1 | F | 1.750 | 58.0 | 12.54 | 4.64 | 3.40 | 0.40 | 8.00 | 0.39 | 5.05 | 9.67 | 13.95 | **58.03** |
| 2 | F | 1.750 | 59.0 | 12.58 | 4.73 | 3.40 | 0.40 | 8.14 | 0.40 | 5.13 | 9.93 | 14.25 | **58.95** |
| 3 | F | 1.600 | 56.7 | 9.96 | 3.88 | 2.49 | 0.31 | 6.36 | 0.29 | 3.69 | 8.79 | 12.63 | **48.40** |
| 4 | M | 1.875 | 87.0 | 16.42 | 8.25 | 4.32 | 0.61 | 14.10 | 0.66 | 8.78 | 18.49 | 24.54 | **96.17** |
| 5 | M | 1.800 | 70.0 | 14.03 | 6.02 | 3.75 | 0.48 | 10.34 | 0.50 | 6.48 | 13.06 | 18.03 | **72.69** |
| 6 | M | 1.796 | 75.7 | 14.22 | 6.55 | 3.72 | 0.50 | 11.15 | 0.53 | 6.88 | 14.58 | 19.80 | **77.92** |
| 7 | F | 1.602 | 63.0 | 10.23 | 4.37 | 2.50 | 0.33 | 7.14 | 0.32 | 4.09 | 10.16 | 14.26 | **53.40** |
| 8 | M | 1.762 | 76.0 | 13.57 | 6.36 | 3.48 | 0.48 | 10.73 | 0.50 | 6.52 | 14.38 | 19.52 | **75.54** |
| 9 | F | 1.610 | 62.5 | 10.34 | 4.37 | 2.54 | 0.34 | 7.16 | 0.33 | 4.13 | 10.09 | 14.20 | **53.49** |
| 10 | F | 1.620 | 45.0 | 9.83 | 3.04 | 2.59 | 0.28 | 5.10 | 0.25 | 3.10 | 6.30 | 9.74 | **40.23** |
| 11 | F | 1.704 | 66.5 | 12.08 | 5.17 | 3.10 | 0.40 | 8.68 | 0.41 | 5.23 | 11.57 | 16.11 | **62.75** |
| 12 | M | 1.794 | 84.5 | 14.59 | 7.38 | 3.71 | 0.53 | 12.44 | 0.57 | 7.54 | 16.93 | 22.58 | **86.29** |
| 13 | F | 1.679 | 57.5 | 11.28 | 4.28 | 2.94 | 0.36 | 7.21 | 0.34 | 4.38 | 9.29 | 13.37 | **53.45** |
| 14 | F | 1.678 | 70.5 | 11.79 | 5.37 | 2.94 | 0.40 | 8.92 | 0.41 | 5.26 | 12.36 | 16.99 | **64.43** |
| 15 | F | 1.600 | 48.0 | 9.64 | 3.21 | 2.49 | 0.28 | 5.31 | 0.25 | 3.15 | 6.92 | 10.43 | **41.47** |
| 16 | F | 1.659 | 53.0 | 10.76 | 3.82 | 2.82 | 0.33 | 6.43 | 0.31 | 3.91 | 8.18 | 12.02 | **48.58** |
| 17 | M | 1.761 | 61.0 | 12.87 | 4.96 | 3.47 | 0.42 | 8.55 | 0.42 | 5.38 | 10.48 | 14.92 | **61.47** |
| 18 | M | 1.731 | 73.0 | 12.85 | 5.90 | 3.27 | 0.44 | 9.90 | 0.46 | 5.96 | 13.37 | 18.27 | **70.43** |
| 19 | M | 1.760 | 98.0 | 14.53 | 8.39 | 3.47 | 0.57 | 13.89 | 0.62 | 8.14 | 20.09 | 26.28 | **95.97** |
| 20 | M | 1.782 | 66.0 | 13.49 | 5.54 | 3.62 | 0.45 | 9.52 | 0.46 | 5.97 | 11.89 | 16.61 | **67.56** |

# *Appendix B*

**Table B1:** Summary of EMG responses to gait perturbations recorded from the right quadriceps (N = 7). A,B,C represent perturbations of 120, 180 and 240 ms duration respectively.

| SUBJECT NUMBER | LATENCY (ms) | | | DURATION (ms) | | |
|---|---|---|---|---|---|---|
| | A | B | C | A | B | C |
| Strategy 1 | **ACTIVATION (N = 4)** | | | | | |
| 1 | 110 | 100 | 90 | 110 | 170 | 190 |
| 3 | 60 | 80 | 70 | 190 | 180 | 210 |
| 5 | 120 | 110 | 110 | 200 | 140 | 190 |
| 8 | 130 | 120 | 160 | 160 | 180 | 160 |
| Mean ± SEM | **105 ± 15** | **113 ± 17** | **108 ± 19** | **165 ± 20** | **168 ± 9** | **180 ± 17** |
| 6 | * | * | * | * | * | * |
| Strategy 2 | **ACTIVATION (N = 2)** | | | | | |
| 7 | 140 | 110 | 90 | 100 | 240 | 150 |
| 9 | 120 | 100 | 130 | 150 | 140 | 130 |
| Mean ± SEM | **120 ± 20** | **105 ± 5** | **110 ± 20** | **125 ± 25** | **190 ± 50** | **140 ± 10** |

* No obvious change in EMG activity above normal

N.B. Due to equipment malfunction no recordings were obtained from Subjects 2 and 4.

**Table B2:** Summary of EMG responses to gait perturbations recorded from the left quadriceps (N = 6). A,B,C represent perturbations of 120, 180 and 240 ms duration respectively.

| SUBJECT NUMBER | LATENCY (ms) | | | DURATION (ms) | | |
|---|---|---|---|---|---|---|
| | A | B | C | A | B | C |
| Strategy 1 | **ACTIVATION (N = 3)** | | | | | |
| 2 | 110 | 140 | 130 | 120 | 220 | 170 |
| 4 | 80 | 60 | 60 | 150 | 210 | 190 |
| 8 | 120 | 180 | 160 | 130 | 110 | 100 |
| Mean ± SEM | **103 ± 12** | **127 ± 35** | **117 ± 30** | **133 ± 9** | **180 ± 35** | **153 ± 27** |
| 1 | * | * | * | * | * | * |
| Strategy 2 | **ACTIVATION (N = 1)** | | | | | |
| 7 | 100 | 80 | 100 | 200 | 190 | 140 |
| 9 | * | * | * | * | * | * |

* No change in EMG above normal activity. No records obtained for Subjects 3, 5 & 6.

**Table B3:** EMG responses to gait perturbations recorded from the right hamstrings (N = 9). A,B,C represent perturbations of 120, 180 and 240 ms duration respectively.

| SUBJECT NUMBER | LATENCY (ms) | | | DURATION (ms) | | |
|---|---|---|---|---|---|---|
| | A | B | B | A | B | C |
| Strategy 1 | **INHIBITION (N = 7)** | | | | | |
| 1 | 120 | 120 | 120 | 150 | 170 | 150 |
| 2 | 210 | 180 | 110 | 110 | 140 | 210 |
| 3 | 130 | 180 | 140 | 130 | 120 | 130 |
| 4 | 160 | 140 | 150 | 240 | 80 | 100 |
| 5 | 140 | 140 | 200 | 150 | 110 | 70 |
| 6 | 100 | 110 | 110 | 130 | 140 | 150 |
| 8 | 150 | 150 | 100 | 150 | 180 | 220 |
| Mean ± SEM | **144 ± 13** | **146 ± 10** | **133 ± 13** | **151 ± 16** | **134 ± 13** | **147 ± 21** |
| Strategy 2 | **ACTIVATION (N = 2)** | | | | | |
| 7 | 120 | 130 | 100 | 50 | 40 | 60 |
| 9 | 170 | 120 | 110 | 80 | 40 | 60 |
| Mean ± SEM | **145 ± 25** | **125 ± 5** | **105 ± 5** | **65 ± 15** | **40** | **60** |

**Table B4:** EMG responses to gait perturbations recorded from the left hamstrings (N = 6). A,B,C represent perturbations of 120, 180 and 240 ms duration respectively.

| SUBJECT NUMBER | LATENCY (ms) | | | DURATION (ms) | | |
|---|---|---|---|---|---|---|
| | A | B | C | A | B | C |
| Strategy 1 | **ACTIVATION (N = 4)** | | | | | |
| 3 | 60 | 50 | 60 | 200 | 190 | 300 |
| 4 | 40 | 50 | 60 | 150 | 170 | 260 |
| 6 | 40 | 50 | 50 | 200 | 210 | 220 |
| 8 | 60 | 70 | 80 | 230 | 320 | 320 |
| Mean ± SEM | **50 ± 6** | **55 ± 5** | **63 ± 6** | **195 ± 17** | **223 ± 34** | **275 ± 22** |
| Strategy 2 | **ACTIVATION (N = 1)** | | | | | |
| 7 | * | * | * | * | * | * |
| 9 | 70 | 40 | 90 | 160 | 220 | 170 |

* No reflex change above normal activity. No records obtained from Subjects 1, 2 & 5.

**Table B5:** EMG responses to gait perturbations recorded from the right tibialis anterior (N = 8). A,B,C represent perturbations of 120, 180 and 240 ms duration respectively.

| SUBJECT NUMBER | LATENCY (ms) | | | DURATION (ms) | | |
|---|---|---|---|---|---|---|
| | A | B | C | A | B | C |
| Strategy 1 | **ACTIVATION (N = 6)** | | | | | |
| 1 | 70 | 70 | 70 | 120 | 170 | 170 |
| 3 | 50 | 70 | 50 | 130 | 150 | 200 |
| 4 | 50 | 50 | 60 | 110 | 150 | 160 |
| 5 | 70 | 60 | 70 | 110 | 150 | 200 |
| 6 | * | 50 | 50 | * | 190 | 190 |
| 8 | 80 | 60 | 80 | 100 | 170 | 160 |
| Mean ± SEM | **64 ± 6** | **60 ± 4** | **63 ± 5** | **114 ± 5** | **163 ± 7** | **180 ± 8** |
| Strategy 2 | **ACTIVATION (N = 2)** | | | | | |
| 7 | 70 | 70 | 60 | 170 | 200 | 160 |
| 9 | 60 | 60 | 70 | 120 | 170 | 160 |
| Mean ± SEM | **65 ± 5** | **65 ± 5** | **65 ± 5** | **145 ± 25** | **185 ± 15** | **160** |

* No change in EMG above normal activity. No record was obtained for Subject 2.

**Table B6:** EMG responses to gait perturbations recorded from the left tibialis anterior (N = 9). A,B,C represent perturbations of 120, 180 and 240 ms duration respectively.

| SUBJECT NUMBER | LATENCY (ms) | | |
|---|---|---|---|
| | A | B | C |
| Strategy 1 | **ACTIVATION (N = 6)** | | |
| 2 | * | * | 70 |
| 3 | 70 | 60 | 70 |
| 4 | 90 | 80 | 100 |
| 5 | 110 | 110 | 150 |
| 6 | 70 | 110 | 110 |
| 8 | 70 | 70 | 80 |
| Mean ± SEM | **82 ± 8** | **86 ± 10** | **97 ± 13** |
| 1 | * | * | * |
| Strategy 2 | **ACTIVATION (N = 2)** | | |
| 7 | 100 | 100 | 100 |
| 9 | 110 | 110 | 110 |
| Mean ± SEM | **105 ± 5** | **105 ± 5** | **105 ± 5** |

* No obvious reflex change above normal activity

**Table B7:** EMG responses to gait perturbations recorded from the right medial gastrocnemius (N = 8). A,B,C represent perturbations of 120, 180 and 240 ms duration respectively.

| SUBJECT NUMBER | LATENCY (ms) | | | DURATION (ms) | | |
|---|---|---|---|---|---|---|
| | A | B | C | A | B | C |
| Strategy 1 | **ACTIVATION (N = 3)** | | | | | |
| 2 | 100 | * | 80 | 90 | * | 110 |
| 3 | 70 | 60 | 50 | 80 | 90 | 110 |
| 5 | 90 | 70 | 80 | 80 | 130 | 80 |
| Mean ± SEM | **87 ± 9** | **65 ± 5** | **70 ± 10** | **83 ± 3** | **110 ± 20** | **100 ±10** |
| 4 | * | * | * | * | * | * |
| 6 | * | * | * | * | * | * |
| 8 | * | * | * | * | * | * |
| Strategy 2 | **ACTIVATION (N = 1)** | | | | | |
| 7 | 40 | 40 | 40 | 40 | 40 | 30 |
| 9 | * | * | * | * | * | * |

* No obvious reflex change above normal activity.

N.B. No records were obtained for Subject 1.

**Table B8:** EMG responses to gait perturbations recorded from the left medial gastrocnemius (N = 7). A,B,C represent perturbations of 120, 180 and 240 ms duration respectively.

| SUBJECT NUMBER | LATENCY (ms) | | |
|---|---|---|---|
| | A | B | C |
| Strategy 1 | **ACTIVATION (N = 6)** | | |
| 2 | 60 | 80 | 80 |
| 3 | 60 | 40 | 30 |
| 4 | 60 | 60 | 70 |
| 8 | 90 | 60 | 100 |
| Mean ± SEM | **68 ± 8** | **60 ± 8** | **70 ± 15** |
| 1 | * | * | * |
| 6 | * | * | * |
| Strategy 2 | **ACTIVATION (N = 2)** | | |
| 7 | 40 | 40 | 40 |
| 9 | 50 | 60 | 80 |
| Mean ± SEM | **45 ± 5** | **50 ± 10** | **60 ± 20** |

* No reflex change above normal activity. No records were obtained for Subject 5.

# *Appendix C*

## HEALTH HISTORY QUESTIONNAIRE

***(all information is strictly confidential)***

**Name:**.....

**Address:**.....

.....

.....

.....

**Age:**.....**DOB:**.....

**Telephone Number:**.....

***Part I***

***If the answer is YES to any of the following questions, please give some details including dates where possible.***

***Male volunteers are requested to ignore questions 24 - 26.***

---

**1. Have you any history of heart trouble? (such as heart attack, angina, valve disease, palpitations, pains in the chest, dizzy spells).**

---

**2. Have you any history of problems with blood vessels? (such as thrombosis, embolus, claudications, aneurysm, dizzy spells).**

---

**3. Have you any history of chest problems (bronchitis, asthma, or wheezy chest).**

---

**4. Have you ever smoked? (if YES, please state if you are a current or ex-smoker and how much).**

---

**5. Do you suffer from diabetes? (if YES, please state if insulin dependent).**

---

**6. Have you any history of major illness now or in the last 20 years? (such as rheumatoid arthritis, blood disorders, cancer).**

---

**7. Do you suffer from osteoarthritis? (if YES state joints affected & indicate mild, moderate or severe & any medication regularly taken).**

---

**8. Have you had falls in the past 12 months? (If YES, please state number of falls in the last year).**

---

**9. Have you ever broken or fractured any bones? (Please indicate when).**

---

**10. Do you have problems with your bones? (e.g., osteoporosis, loss of height).**

---

**11. Have you any history of back problems?**

---

**12. Have you had any surgery on your joints? (please specify).**

---

**13. Do you have any history of Central Nervous System Disease (such as Parkinson, Alzheimer, Epilepsy).**

---

**14. Do you have any history of emotional or psychiatric problems? (please describe).**

---

**15. Do you suffer from high blood pressure?**

---

**16. Have you any acute illness in the last 6 months? (such as influenza or bronchitis).**

**17. Have you been in the hospital in the last 5 years & if so, for how long?**

**18. Please state any medication regularly taken for any condition.**

**19. Have you ever had any history of dizziness or balance-system dysfunction?**

**20. Have you had any ear infection in the last twelve months?**

**21. Do you have any physical disabilities?**

**22. Do you have any visual or hearing problems? (please specify).**

**23. Is there any other illness or condition that affects your general health or interferes with your mobility?**

**24. What was your age when you had the menopause? (i.e., when did your periods stop?).**

---

**25. Have you had a hysterectomy or ovariectomy (i.e., removal of womb or ovaries?).**

---

**26. Have you ever been prescribed hormone replacement therapy?**

---

**27. Do you take any exercise? (If YES, please state its type, frequency and duration per session).**

---

**28. Approximately how tall are you?**

---

**29. Approximately how much do you weigh?**

---