The role of body segment movements on the control of centre of mass during balance corrections

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List of abbreviations:
COM: centre of mass; CNS: central nervous system; SCA: Spinal Cerebellar Ataxia; APA: anticipatory postural activity.

Human stance is an instable bipedal posture characterized by a high centre of mass (COM) located near the hips. The COM (projected onto ground level) needs to be held within the small area of support (defined by the two feet) to maintain equilibrium.
Elderly people and those with neurological deficits have problems with balance. About 30% of community-dwelling adults aged 65 and older fall at least once each year. Falls and fall-related injuries have been shown to be independent determinants of functional decline. Falls occur in different directions and at different speeds depending on the direction and intensity of the perturbation to balance.

The way the central nervous system (CNS) responds to an impending fall depends on many factors, direction and velocity of falling are the two most crucial. Thus, when human stance is perturbed, the CNS must utilize and integrate the available sensory and environmental information to select an appropriate response strategy, especially for fast backward falls.

In this thesis standing balance was perturbed using servo-controlled multi-directional rotations of the support surface. Balance perturbations consisted of combined pitch and roll rotations (7.5° and 60°/s) presented randomly in different directions. Thus, in a sideways rotation of the support surface to the right the subject's COM moved to the right side and needed to be corrected to avoid a fall. A visual feedback of COM position based on surface reaction forces was presented prior to stimulus onset in order to standardise stance position. Outcome measures were biomechanical responses (kinematics and kinetics) and surface EMG activity of several muscles.

The action of the CNS can be investigated by studying patient groups with clearly defined balance deficits. Thus, patients with spinal cerebellar ataxia (SCA) were the focus of the first study in this thesis. The goals of this study were to investigate the correlations between body segment movements and COM velocity during pathological balance corrections of SCA patients compared to controls, and to relate correlations indicating instability to EMG activity differences. Therefore, activation patterns of several leg and trunk muscles, kinematics and kinetics were compared between a group of SCA patients and age-matched controls. The results showed that, for lateral perturbations, peaks in COM lateral velocity were larger in SCA patients than controls. These peaks were correlated with increased ("hypermetric") trunk roll downhill and reduced uphill knee flexion velocity. Subsequent arm abduction partially corrected the lateral instability. Excessive posterior COM velocity coincided with marked trunk hypermetric flexion forwards. Early balance correcting responses in knee and paraspinal muscles have reduced amplitudes compared to normal responses, not increased response amplitudes as expected. Later responses were consistent with compensation mechanisms for the lateral instability created by the stiffened knee and pelvis.
It was concluded that truncal hypermetria coupled with insufficient uphill knee flexion are the primary causes of lateral instability in SCA patients. Holding the knees and pelvis more rigid possibly permits a reduction in the controlled degrees of freedom and concentration on arm abduction improves lateral instability. For backwards perturbations excessive posterior COM velocity coincided with marked trunk hypermetric flexion forwards. A further conclusion was that this flexion and the ensuing backwards shift of the pelvis results from rigidity which

jeopardizes posterior stability. Timing considerations and the lack of confirmatory changes in amplitudes of EMG activity suggest that both lateral and posterior instability in SCA is primarily a biomechanical response to pelvis and knee rigidity resulting from increased muscle background activity rather than changed evoked responses.

It has been shown that balance corrections depend on the impending fall direction. Thus direction is crucial in programming muscle activity to recover balance or damping a fall. Muscle activity is controlled by the CNS. Directional sensitivity of the sensory inputs and ensuing responses were shown by Allum et al. (2008) and Carpenter et al., 1999, 2001). The question is whether the CNS independently controls roll and pitch movements of the human body during balance corrections. To help provide an answer to this question, the balance of 16 young healthy subjects using multi-directional rotations of the support surface was perturbed. All rotations had pitch and roll components, for which either the roll or the pitch component were delayed by 150 ms or not at all.

Across all perturbation directions, delayed roll caused equally delayed shifts (150 ms) in peak lateral COM velocity. Across directions, delayed pitch did not cause equally delayed shifts in anterior-posterior COM velocity. After 300 ms however, the vector direction of COM velocity was similar to the directions seen in the no delay condition. Trunk, arm and knee joint rotations followed this roll compared to pitch pattern but were different from the no delay rotation synergies after 300 ms, suggesting inter-segmental compensation for the delay effects. Balance correcting responses of muscles demonstrated both roll and pitch directed components regardless of axial alignment. Muscles were categorised into three groups: pitch oriented, roll oriented and mixed. Lower leg muscles were pitch oriented, trunk muscles roll oriented, and knee and arm muscles mixed. The results of this study suggest that roll, but not pitch components, of balance correcting movement strategies and muscle synergies are separately programmed by the CNS. Reliance on differentially activated arm and knee muscles to correct roll perturbations reveals a dependence of the pitch response on that of roll, possibly due to biomechanical constraints, and accounts for the failure of delayed pitch to be transmitted equally in time across all limbs segments. Thus it appears the CNS preferentially programs the roll response of the body and then adjusts the pitch response accordingly.

During an impending fall some body segments may be preferentially used to recover balance. As shown in the study of SCA patients, the knees play a critical role for correcting fall in lateral directions - stiff knees impair balance recovery. Thus, training adequate knee flexion would help to recover balance.

To determine whether voluntary movements can be effectively incorporated into balance corrections two studies with voluntary body movements were performed. “Knee flexion” and “trunk bending” young healthy subjects had to execute unilateral knee flexion and lateral trunk bending, respectively, simultaneously with support surface tilts. Unilateral uphill knee flexion benefited balance recovery. Subjects rotated their pelvis uphill more than predicted. Downhill knee bending also reduced COM motion. This because of a greater than predicted simultaneous lateral shift of the pelvis uphill. Leg muscle activity of voluntary knee bending showed anticipatory postural activity (APA) with similar profiles to early balance correcting responses. EMG response amplitudes for combined voluntary and compensatory responses were generally not different from just compensatory responses and therefore smaller than predicted. These results suggest that because EMG patterns of APA of voluntary motion and early balance corrections have similar profiles, the CNS is able to incorporate voluntary activation associated

with unilateral knee flexion or lateral trunk bending into automatic postural responses. The effect on movement strategies appears to be non-linear.

In conclusion, these studies provide crucial insights into central programming of balance reactions useful for developing rehabilitation programs to improve balance.

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Zusammenfassung
(German Summary)

Zusammenfassung

Liste der Abkürzungen:
COM: Center of Mass (Körperschwerpunkt); CNS: Central nervous system (Zentralnervensystem); SCA: Spinale Cerebellum Ataxie (Degeneration des Kleinhirns).

Der aufrechte Stand auf zwei Beinen ist an sich eine instabile Körperhaltung. Der Körperschwerpunkt (centre of mass: COM) liegt relativ hoch über dem Boden (im Beckenbereich) und muss innerhalb einer Standfläche von wenigen cm^2 gehalten werden.
Ältere Leute und Patienten mit neurologischen Defiziten haben Probleme mit ihrem Gleichgewicht. Tatsächlich stürzen etwa 30% der selbständig lebenden Personen über 65 und älter mindestens einmal pro Jahr. Stürze und ihre Folgeverletzungen sind einschneidend für die Unabhängigkeit dieser Personen. Diese Stürze und ihre Folgen sind abhängig von der Geschwindigkeit, bzw. der Stärke, und der Richtung, aus welcher eine Störung des Gleichgewichts erfolgt.

Wie das Zentrale Nervensystem (CNS) auf einen drohenden Sturz reagiert, hängt von einigen Faktoren ab, Geschwindigkeit und Richtung als die zwei wichtigsten genannt. Wenn also unser aufrechter Stand gestört wird, muss das CNS an Hand aller vorhandenen Informationen von unseren Sinnesorganen eine angepasste Reaktion auszuwählen.

Für diese Arbeit wurde das Gleichgewicht mit Hilfe einer Rotations-Plattform gestört. Die Störungen bestanden aus kombinierten vor/rückwärts und seitwärts Kippungen (7.5° und 60°/s), welche in einer zufälligen Reihenfolge erfolgten. Kippte die Plattform nach rechts, schwankte auch das COM der Versuchsperson nach rechts zur Tal-Seite, was korrigiert werden musste, um einen Sturz zu verhindern. Um die Anfangsposition der Probanden zu kontrollieren, wurde ein visuelles Feedback genutzt, welches die COM-Position vor der Störung auf Grund von Bodenreaktionskräften anzeigte. Gemessen wurden biomechanische Daten der Körperbewegung (Kinematik) und der Bodenreaktionskraft (Kinetik) und die Aktivität verschiedener Muskeln.

Wir können unser CNS studieren, indem wir Patienten mit klar definierten Defiziten dieses Steuer-Systems untersuchen. Darum sind in der ersten Studie dieser Arbeit Patienten mit Cerebellum Ataxie (SCA) ausgesucht worden, mit dem Ziel, ihre pathologischen Gleichgewichtsreaktionen zu testen. Die Probenden hatten auf einer Plattform zu stehen, die plötzlich kippte. Die Bewegungen der einzelnen Körpersegmente sollten dann mit der Schwankung des COM korreliert werden und dies mit den Reaktionen gesunder Personen verglichen werden. Ausserdem sollte auch die Muskelaktivität weiteren Aufschluss über die Reaktionen der SCA Patienten geben. Also wurden Kinetische (Kraft), Kinematische (Bewegung) und Muskelaktivierungs-Daten von 18 SCA Patienten und 21 gesunden Personen gesammelt und verglichen. In den Resultaten zeigte sich, dass die Geschwindigkeit, mit der das COM abwärts schwankte, bei den Patienten deutlich höhere Werte erreichte als bei den gesunden Probanden. Diese Werte zeigten einen Zusammenhang (Korrelation) mit der übermässigen, abwärtsgerichteten Oberkörperbewegung der Patienten. Dieses Schwanken nach unten wurde dann teilweise mit einer zusätzlichen Armbewegung kompensiert. Bei einer übermässigen Rückwärtsschwankung des COM konnte eine verstärkte Oberkörperbeugung nach vorne beobachtet werden. Die Knie- und unteren Rückenmuskeln zeigten zudem in einer frühen Phase der Reaktion reduzierte Amplituden verglichen mit den Reaktionen der gesunden Personen. In einer späteren Phase waren dann aber entsprechende Kompensationsreaktionen zu sehen, die nötig wurden auf Grund der grösseren Instabilität.
Aus diesen Resultaten geht hervor, dass die Versteifung der Kniegelenke, was zu einer zu geringen Biegung des Berg-Knies führt, plus die übermässige Oberkörperbeugung nach vorne,

die Hauptfaktoren für die grössere Instabilität sind. Die Versteifung von Gelenken aber ermöglicht eine Reduktion der Freiheitsgrade und die Patienten können sich so mehr auf eine kompensatorische Armbewegung konzentrieren. Die versteiften Kniegelenke führen zu einer vermehrten Beugung des Oberkörpers nach vorne. Das Becken wird so nach hinten geschoben und die Stabilität ist auch hier gefährdet. Eine erhöhte Grundaktivität der Muskulatur lässt auf eine aktive Versteifung schliessen und die Instabilität ist demzufolge biomechanischer Natur.

Gleichgewichtsreaktionen sind also abhängig von der Richtung eines drohenden Sturzes. Die Richtung ist grundlegend für die Programmierung einer angepassten Reaktion, den Sturz aufzuhalten oder abzufangen. Die Reaktion beruht auf Muskelaktivität, welche vom CNS gesteuert wird. Nun kommt die Frage auf, wie das CNS die Reaktionen auf alle möglichen Sturzrichtungen programmiert. Ist es möglich, dass die zwei Körperachsen, anterior-posterior und medial-lateral, unabhängig voneinander kontrolliert werden? Um dies zu beantworten sind 16 junge, gesunde Personen auf der kippbaren Plattform getestet worden. Dabei bestanden alle Kippungen aus einer seitlichen und einer vor-, bzw. rückwärts Komponente. Hier konnte dann entweder die seitliche oder die vor/rückwärts Komponente zeitlich verzögert (150 ms) oder simultan zueinander ausgelöst werden. In der seitlichen COM Schwankung zeigten sich gleichmässig verzögerte (150 ms) Geschwindigkeitsspitzen. Für die vor/rückwärts Richtung konnte keine solch gleichmässige Verzögerung festegestellt werden. Die einzelnen Köpersegmente zeigten dann auch entsprechende Kompensationsbewegungen. In der Muskelaktivität konnte ein klarer Richtungseffekt betreffend medial-lateral und vor/rückwärts beobachtet werden, unabhängig von der Ausrichtung des Muskels zu den Körperachsen. Demzufolge konnten die Muskeln in drei verschiedene Gruppen eingeteilt werden: Muskeln mit medial-lateraler, vor/rückwärts oder gemischter Orientierung. Dabei konnten die Muskeln des Rumpfes der ersten, die der Unterschenkel der zweiten, und die Knie- und Armmuskeln der dritten Gruppe zugeteilt werden.
Daraus geht hervor, dass die seitliche, aber nicht die vor/rückwärts Komponente der Gleichgewichtsreaktion separat vom CNS programmiert wird. Die Arm- und Kniemuskelaktivitäten lassen den Schluss zu, dass die vor/rückwärts von der seitlichen Komponente abhängig ist. Diese Abhängigkeit und der biomechanische Aufbau des menschlichen Körpers führen dazu, dass die Übertragung des vor/rückwärts Kippung nicht vollständig auf den ganzen Körper übertragen wird. Es scheint also, dass das CNS vorzugsweise zuerst die seitliche Komponente der Reaktion programmiert und dann die Reaktion für die vor/rückwärts Komponente entsprechend anpasst.

Die Richtung des drohenden Sturzes bedingt, welche Körpersegmente wie eingesetzt werden. Wie schon in der Patienten Studie gezeigt, spielen die Kniegelenke eine zentrale Rolle im aufrechten Stand – versteifte Kniegelenke verstärken die Körperschwankung. Also wäre eine Wieder- Integrierung der Kniebewegung wünschenswert.
Um herauszufinden, ob es möglich ist, eine willkürliche Bewegung und eine automatische Gleichgewichtsreaktion zu kombinieren, sind zwei Studien zu dieser Fragestellung gemacht worden. Junge, gesunde Probanden hatten die Aufgabe, gleichzeitig zur Kippung der Plattform eine vorgegebene Knie-, bzw. Oberkörperbiegung durchzuführen. Es zeigte sich, dass die zusätzliche Knieflexion eine klare Verminderung der COM Schwankung zur Folge hatte. Dies, wenn das Berg-Knie, aber auch wenn das Tal-Knie zusätzlich gebogen wurde. Beim letzteren wurde das Becken, und mit ihm eine grosse Körpermasse, entsprechend weiter zur Bergseite geschoben und der Körper somit stabilisiert. Die Muskelaktivitäten der Gleichgewichtsreaktion und einer willkürlichen Kniebewegung zeigten ähnliche Aktivierungsmuster. Weiter waren aber die kombinierten Muskelantworten nicht grösser als die der ‚normalen' Gleichgewichtsreaktion und somit weniger ausgeprägt als erwartet.

Wenn also die Aktivierungsmuster der zwei Bewegungen, die zu kombinieren sind, Ähnlichkeiten aufweisen, lässt sie das CNS zu einer Bewegung verschmelzen. Dies geschieht aber nicht linear, wie die Abweichung von den erwarteten Werten gezeigt hat.

Diese Studien haben Einblicke in die zentrale Steuerung von Gleichgewichtsreaktionen gegeben. Daraus lassen sich neue Ansätze für Rehabilitationsprogramme ableiten. So sollte aufrechtes Stehen nicht nur durch Stehhilfen stabilisiert, sondern auch aktiv trainiert werden, jedoch in einer sicheren Umgebung, um Stürze und Verletzungen zu vermeiden. Willkürliche Bewegungen, die automatischen Gleichgewichtsreaktionen ähneln, können unabhängig seitlich und vor/rückwärts trainiert werden, um dann später zu einem kombinierten Bewegungsmuster zu verschmelzen. Hier kann der Trainingsschwerpunkt auf verschiedene Körpersegmente gelegt werden, wobei die Arm, Knie und der Oberkörper einen positiven Effekt zeigen. So kann ein individuelles Programm zusammengestellt werden.

Introduction

Introduction

List of abbreviations:
COM: centre of mass; CNS: central nervous system; SCA: Spinal Cerebellar Ataxia; APA: anticipatory postural activity; EMG: Electromyograhpy; AP: anterior-posterior; LR: medial-lateral; Vert: vertical.
Roll: angle in the frontal (LR) plane; Pitch: Angle in the sagittal (AP) plane.

Posturography

Posturography is a method of measuring a subject's ability to control his balance specifically during upright stance. It covers the techniques including static (quiet stance) or dynamic posturography (perturbations to stance). Dynamic posturography uses a movable servo-controlled horizontal platform. A computer is used to control electric motors which can move the support surface in the horizontal direction (translation) and/or incline it thereby destabilising quiet stance. Early investigators, for example Allum (1979) and Nashner (1979) used dynamic posturography to quantify the neural mechanisms involved in the control of posture and balance by these sensory, motor and central processes. Thus, one version of dynamic posturography tests the efficacy of sensory contributions to balance control by servoing body sway to that of the support surface or the visual surround. Different protocols were used to investigate the complex interactions among these processes. Static posturography involves the subject standing on a fixed instrumented platform (force-plate) with embedded sensitive force detectors. Such sensors can detect tiny oscillations of the body.

The dynamic posturography platform of the Laboratory for experimental Neuro-Otology at the University Hospital Basel (Carpenter et al., 1999) was used for the studies described in this thesis. This platform can be rotated in two directions controlled with a computer controlling the velocity of the tilt motion and its amplitude in roll and pitch planes independent of each other. To standardize pre-stimulus subject position across trials, visual feedback of the subjects' own anterior-posterior (AP) and medio-lateral (LR) ankle torque was presented to the subject on a cross with light-emitting diodes at 5 m distance. Force sensors in the support surface measured ground reaction forces. Because the ankle joint centre location was fixed by strapping the foot in place on the platform, ankle torques could be calculated.

A video-based motion analysis system was used to collect full body kinematics using a three-dimensional optical tracking system with infrared-light-emitting diodes (IREDs). For calculating joint kinematics, sets of IREDs are placed on the skin at standard anatomical or bony landmarks, thereby defining body segments. Subjects wore tight fitting shorts and vests to reduce marker movements with respect to skin. Three Optotrak® cameras with known position and orientation were placed in front of the subject, thereby permitting location and measurement of the position of the IREDs in 3 dimensions.
Primary variables of interest were COM displacement and velocity, body segment displacements, joint flexions and muscle responses of the legs, arms and the trunk. Total body COM displacement was calculated separately for the AP, LR and vertical (Vert) directions using a 12 body segment adaptation (Visser et al., 2008) of a 14 segment model of the human body (Winter et al., 2003). In addition, the following angular displacements were calculated: knee angle (left and right), absolute trunk and pelvis angle (roll and pitch) and linear displacement, and upper arm abduction angle (left and right). Knee angles were calculated as the angle spanned

by the two unit vectors of the upper and lower legs. Absolute rotation angles of the planes defined by trunk and the platform support surface were defined using 3 or 4 markers on these segments. Rotations of the upper arm with respect to the trunk and the upper leg with respect to the pelvis were calculated as ball joint angles defined by two angels of spherical coordinates, e.g. for the upper arm by arm rotation and abduction.
To obtain information about muscle activation during balance corrections, electrical activity of single muscles was recorded with electromyography (EMG). The information extracted from EMG signals in this thesis is primarily amplitude (area under a response) and onset timing.

Body segment movement and postural instability

Falls become a problem when muscle and sensory pathophysiology begin interfering with the person's ability to compensate. Thus, age and severity of disability are contributors to fall risk (Tinetty et al., 1986). Falls create immense social problems because of their association with physical decline, negative impact on quality of life, and markedly reduced survival rate (Bloem et al., 2003, Vassallo et al., 2005). In addition, falls pose high costs to the public health service. For these reasons, falls and therefore balance and postural control is a crucial topic for research.

Maintenance of upright stance requires the COM of the body to be positioned over the base of support. The human body in the upright standing position is inherently instable due to high COM and short base of support when leaning backwards. Postural control is a complex process requiring integration of the sensory information and execution of appropriate postural responses. To maintain upright stance, the central neural system (CNS) must coordinate motion across many joints and muscles using sensory information provided by visual, somatosensory and vestibular systems.
To better understand postural control, responses following multidirectional perturbations to stance (dynamic posturography) have been investigated. Perturbations to upright stance cause shifts of the COM that can be corrected by movements of the arms, legs and/or trunk (Patla et al., 2002; Pozzo et al., 2001). Further postural instability may be caused by inappropriately scaled or timed muscle activity (Bloem et al., 2002; Carpenter et al., 2001; Diener et al., 1984; Horak and Diener, 1994) or by general stiffening of the body (Allum et al., 2002; Bloem et al., 2002; Oude Nijhuis et al., 2008) leading to destabilizing body segment movements. Thus, instability can be due to increased joint stiffness caused by prior muscle co-contraction leading to insufficient active joint flexion as seen for spinocerebellar ataxia (SCA) patients. For these patients, knee rigidity is associated with greater instability following support surface tilts (Oude-Nijhuis et al., 2008). In addition in these patients balance correcting response in the trunk are pathologically instable.

The knee joints are probably the most important joints for stable balance control (Allum et al., 2008; Oude-Nijhuis et al., 2008). As these two joints lie in the frontal plane and work in a "push-pull" manner in the sagittal plane they control both LR and AP movements of the human body. The question arises how knee flexions control LR and AP movements. If balance corrections are differently organised in the roll (medio-lateral) and pitch (anterior-posterior) directions, exploring these differences may provide more insights into mechanisms underlying falls.

One hypothesis is that no differences exist between the roll and pitch commands issued by the CNS, rather a common movement strategy and muscle synergy is used regardless of perturbation direction (Henry et al., 1998a,b; Park et al., 2004; Jones et al., 2008). According to this viewpoint, differences in movement responses or joint torques with perturbation direction can be explained by a simple directional re-weighting of the muscle responses along the body according to the alignment of lines of muscle action with perturbation directions. It was suggested that this

re-weighting would take into account the inherent differences in skeletal geometry that lead to different initial responses of the body to the perturbation in the pitch and roll directions. On the other hand Allum et al. (2003) and Carpenter et al. (1999, 2001) suggested that there were too many factors to be taken into account for a single directionally re-weighted response synergy to work effectively. Some of the factors influencing differences in roll and pitch balance correcting strategies are the differences in the arrival of roll and pitch stimulus-related sensory information used to generate these strategies (Allum et al., 2008), the directional sensitivity of muscle responses (Carpenter et al., 1999) and the need for different knee flexing strategies in the response to roll and pitch tilts (Allum et al., 2008; Oude-Nijhuis et al., 2008). Thus another viewpoint that has been developed in this thesis is that the CNS controls roll and pitch joint torques separately. Winter et al. (1996) already suggested separate control of roll and pitch torques during quiet stance and others argued that this is the case for balance corrections (Allum et al., 2008; Carpenter et al., 2001; Matjacic et al., 2001; Ting et al., 2004; Torres-Oviedo et al., 2006). Matjacic et al. (2001) argued that control in the LR and AP directions is decoupled based on the observation that net joint torques in pitch only and the roll only directions were identical to those elicited for combined pitch and roll perturbations of the same magnitude. It could however be argued that this does not implicate different control in the two planes and may provide support for the viewpoint that a common torque strategy is utilized regardless of perturbation direction (Henry et al., 1998a, b). It is argued here that 3 different synergies required: One for lateral perturbations, similar for the left and right directions, yet opposite in polarity and two aligned in opposite directions in the pitch plane (that is for the toe-up and toe-down synergies; Allum et al., 2003, 2008). This concept that was tested here assumes different movement strategies for pitch and roll as concluded on the basis of previous studies on humans (Carpenter et al., 2001; Grüneberg et al., 2005; Matjacic et al., 2001; Winter et al., 1996).

Regardless of how balance corrections are corrected the key factor that ultimately determines whether or not a balance perturbation leads to a fall is the ability, or inability, to recover balance (Maki & McIlroy, 2006). Skilful motor performance produces the optimal response taking into account external gravitational forces or obstacles and internal constraints like the body physique itself (Massion, 1992). Earlier studies focussed investigating automatic balance corrections and then compared voluntary and automatic postural responses (Nashner and Cordo, 1981). Although these authors found a number of marked dissimilarities between those two types of responses, also similarities in response latencies were found when voluntary movements were well-practiced, executed in a predictable direction and performed under conditions of postural stability. These studies, however, were restricted to the sagittal plane. When laterally directed movements were studied, more dissimilarities were found between these two kinds of responses (Hughey and Fung, 2005). This effect appeared to be due to the different goals and biomechanical constraints of voluntary activation compared to automatic postural responses resulting from unexpected balance perturbations. The main difficulty of integrating the latter into balance corrections is that the muscle forces of anticipatory postural adjustments (APAs) of voluntary movements may, at the same time, provide sensory inputs that disturb the internal reference needed to plan balance corrections following perturbations to stance (Massion, 1992; Oude-Nijhuis et al., 2007). If a voluntary leg movement is to aid balance control it would seem important that the voluntary movement and the automatic balance correction have a similar muscle response synergy and movement strategy.

Overall, the question arises whether changed balance control in patients results from an alternative movement strategy being adopted, or a primary destabilisation mechanism such as muscle stiffness. It has to be clarified, which body segment motions lead to instability and which of them are due to instability in which plane (roll or pitch). Differentiating between instable

motion in the roll and pitch plane would further aid to improve our knowledge about programming of balance corrections. This information is important for developing alternative voluntary movement strategies aiding automatic postural responses. Finally these newly developed voluntary movement strategies need to be verified as beneficial rehabilitation techniques.

Research goals of the thesis

The knees play a critical role in maintaining balance. This is very apparent in SCA who provide a model of instable balance (Bakker et al., 2006). These patients tend to actively stiffen up their knees and, therefore they show clear difficulties in balance control in our perturbation study. The knees work as a kind of damping element between the support surface and the upper body. Thus, stiff knees transmit the support surface perturbation directly through to the trunk, which will be deflected out of the stable position. The upper body, which consists of pelvis, lower and upper trunk and the head, has a great influence on the COM due to its large mass. Thus, it was assumed that deficits in trunk, knee and arm movements of SCA patients to surface tilt and the resulting correlations with changes in COM velocities would provide insights into the pathophysiology of cerebellar balance disturbances. The main focus of my first study (Küng et al., 2009a) was on balance impairments and compensating strategies in these patients.

Knees are found to be a key-element in balance control. As the two knee joints lie in the frontal plane and working in the sagittal plane they control both lateral and frontal movements of the human body. But are these separately controlled? The goal of our second study (Küng et al., 2009b) was to provide supporting evidence for separate neural control of roll and pitch body motion during balance corrections. One hypothesis was that the biomechanical reactions of the human body in the roll and pitch planes are decoupled from one another and for this reason the CNS controls motion in these planes independently (Grüneberg et al., 2005). This control strategy was revealed using delays in the roll and pitch components of tilt stimuli.

A third study (Küng et al., 2009c) investigated incorporating voluntary knee bending into balance correcting responses with the goal of developing a possible compensatory strategy and developing training programs for patients with balance impairments. Thus, the interactions between balance corrections elicited by unexpected rotational perturbations of the support surface and synergies due to simultaneously executed voluntary unilateral knee flexion were examined. The question arose how voluntary unilateral knee flexion synergies alter the inter-segmental shaping of automatic balance corrections. It is presumed that additional knee flexion of the uphill knee would reduce the lateral shift of the COM and be well integrated into balance corrections but that flexion of the downhill knee would not. Thus an overall aim was to investigate whether the muscle synergies for voluntary knee movements and automatic balance corrections were similar in the leg muscles and whether these were well integrated when performed simultaneously.

Voluntary knee bending also yielded a relative large amount of lateral trunk bending opposite to the site of extra knee flexion. Hence, the effect of voluntary trunk roll to balance recovery has also been investigated to complete these series of papers on compensating voluntary strategies (Küng et al., 2009d). The purpose of this trunk study was to examine the effects of voluntary lateral trunk bending executed simultaneously with automatic balance recovery following a sudden unexpected rotational perturbation of the support surface. The question arises how voluntary lateral trunk bending alters the inter-segmental shaping of automatic balance corrections in comparison to knee bending.

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Postural Instability in Cerebellar Ataxia: Correlations of Knee, Arm and Trunk Movements to COM velocity

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Postural Instability in Cerebellar Ataxia: Correlations of Knee, Arm and Trunk Movements to COM velocity

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Abbreviations: AP, anterior–posterior; BF, biceps femoris; COM, center of mass; DM, deltoideus medius; GM, gluteus medius; ICARS, International Cooperative Ataxia Rating Scale; IRED, infrared-emitting diode; Lat, lateral direction; Para, paraspinalis; PL, peoneus longus; RF, rectus femoris; SARA, Scale for the Assessment and Rating of Ataxia; SCA, spinocerebellar ataxia; Sol, soleus; TA, tibialis anterior; VL, vestibular loss.

Abstract

The aim of this study was to investigate the correlations between body segment movements and centre of mass (COM) velocity during pathological balance corrections of spinocerebellar ataxia (SCA) patients compared to controls, and to relate correlations indicating instability to EMG activity differences.

Eighteen SCA patients and 21 age-matched controls were tested. Upright standing was perturbed using rotations of the support surface. We recorded body motion and surface EMG.

For lateral perturbations peaks in COM lateral velocity were larger in SCA patients than controls. These peaks were correlated with increased ("hypermetric") trunk roll downhill and reduced uphill knee flexion velocity. Subsequent arm abduction partially corrected the lateral instability. Excessive posterior COM velocity coincided with marked trunk hypermetric flexion forwards. Early balance correcting responses in knee and paraspinal muscles showed reduced amplitudes compared to normal responses. Later responses were consistent with compensation mechanisms for the lateral instability created by the stiffened knee and pelvis. We conclude that truncal hypermetria coupled with insufficient uphill knee flexion are the primary causes of lateral instability in SCA patients. Holding the knees and pelvis more rigid possibly permits a reduction in the controlled degrees of freedom and concentration on arm abduction to improve lateral instability. For backwards perturbations excessive posterior COM velocity coincided with marked trunk hypermetric flexion forwards. We concluded that this flexion and the ensuing backwards shift of the pelvis results from rigidity which jeopardizes posterior stability. Timing considerations and the lack of confirmatory changes in amplitudes of EMG activity suggest that both lateral and posterior instability in SCA is primarily a biomechanical response to pelvis and knee rigidity resulting from increased muscle background activity rather than changed evoked responses.

Key words: Cerebellar Ataxia, Centre of Mass Movements, Balance Corrections, EMG Activity.

Introduction

Human stance is an unstable bipedal posture, characterized by a high centre of mass (COM) that needs to be controlled exactly to maintain equilibrium. Perturbations of upright stance cause shifts of the COM that can be corrected by movements of the arms, legs or trunk (Patla et al., 2002; Pozzo et al., 2001). Postural instability may be caused by inappropriately scaled or timed muscle activity (Bloem et al., 2002; Carpenter et al., 2001, Diener et al 1984, Horak and Diener 1994) or by general stiffening of the body (Allum et al., 2002; Bloem et al., 2002; Oude Nijhuis et al., 2008) leading to destabilizing body segment movements.

Experimentally reduced inter-link movements or artificial rigidity applied to healthy controls can mimic some of the balance abnormalities seen in spinocerebellar ataxia (SCA) patients (Grüneberg et al., 2004; Oude Nijhuis et al., 2008). These abnormalities can be due to increased joint stiffness caused by prior muscle co-contraction or insufficient active joint flexion. For example, stiffening the hips and trunk of healthy subjects using a full-body rigid corset produces instability (Grüneberg et al., 2004) resembling the effects observed in Parkinson's disease or total leg proprioceptive loss patients (Adkin et al., 2005; Bloem et al., 2002). However, when only movement at the pelvis was blocked, trunk motion was hypermetric in both the anterior and lateral directions (Grüneberg et al., 2004). Blocking knee flexion with a casts causes a pitch-directed instability similar to that of SCA patients, but healthy controls quickly develop a compensatory strategy involving excessive arm movements to reduce lateral instability

(Oude-Nijhuis et al., 2008). Similar adaptive mechanisms have been observed in the elderly who have stiffer trunk motion than young (Allum et al., 2002). Thus, using artificially stiffened limbs in healthy subjects in an attempt to mimic patient responses may provide some, but limited, insights into their responses. The question arises whether changed balance control in patients results from an alternative movement strategy being adopted, or a primary destabilisation mechanism. Our aim was to clarify this issue by correlating COM and limb motion.

SCA patients are ideal for studying the influence of unstable body segment movements on COM shifts. Lateral and anterior-posterior instability are core features of deficient balance control in patients with autosomal dominant SCAs (Bakker et al., 2006; van de Warrenburg et al., 2005a). Vestibular loss (VL) patients, for example, generally show less pronounced instability to support surface tilt (Allum et al., 2008). Both, VL and SCA patients, however, have a clear lack of knee flexion and exaggerated arm movements in response to roll perturbations (Allum et al., 2008; Bakker et al., 2006), suggesting common mechanisms in patients with balance deficits. It is not known which of these deficient limbs movements creates instabilities. Thus, as a follow-up study to that of Bakker et al. (2006) we investigated whether lack of knee movement or large arm movements or truncal hypermetria are key elements of instability in SCA. We assumed that deficits in trunk, knee and arm movements to surface tilt and the resulting correlations with changes in COM velocities would provide insights into the pathophysiology of cerebellar balance disturbances. We further assumed that changes not explained by changes in balance correcting EMG activity would be the result of joint stiffness.

Materials and Methods

Subjects

Eighteen patients with genetically proven autosomal dominant cerebellar ataxia (SCA) whose clinical presentation was dominated by CA, were recruited from outpatients of Radboud University Nijmegen Medical Centre (Table 1). Patients 10 to 18 in Table 1 were tested by Bakker et al. (2006) and the data of these patients were used in our correlation analysis. Twenty-one healthy subjects without neurologic or orthopaedic problems served as age-matched controls. Exclusion criteria were loss of independent ambulation, severe visual disturbances, and cognitive impairment. All patients were examined by a neurologist (BPCW) specialized in cerebellar ataxia who selected patients without prominent extracerebellar signs (such as spasticity or extrapyramidal features) that would affect balance. As there is a considerable range of additional, potentially relevant, pathologies in our patient group, those with extrapyramidal features are noted below and in Table 1.

All patients showed moderate to severe ataxia [patient 1 to 9: SARA (Scale for the Assessment and Rating of Ataxia), mean value of 9.9 ± 1.1; patient 10 to 18: ICARS (International Cooperative Ataxia Rating Scale), mean value of 25.8 ± 3.7] (references for SARA are: Schmitz-Hübsch et al., 2006a; Schmitz-Hübsch et al., 2006b; Schoch et al., 2007; Weyer et al., 2007; for ICARS: Trouillas et al., 1997). All patients had reduced balance confidence, as assessed by the Activities-specific Balance Confidence (ABC) scale (see Table 1). SCA 6 and SCA 14 patients listed in Table 1 are subtypes with mostly uncomplicated forms of spinocerebellar ataxia, although extrapyramidal features can be found in young-onset SCA 14 cases. This was indeed the case in patient 18 who had focal dystonia of the right hand. However, this symptom did not interfere with gait and balance. Although SCA 1 and 3 patients were selected specifically with no or minimal neuropathy, one subject had subtle neuronopathy (subject 6) and one had mild axonal neuropathy (subject 17). These symptoms are mostly subclinical features (van de Warrenburg et al., 2004). Three other subjects (SCA 2 or 6) had a neuropathy (7, 13, 16) on neurophysiological examination and subject 1 (SCA14) had a minor dorsal column disturbances. One SCA 2 patient had mild pontine atrophy on neuroimaging, but the clinical correlate was mainly saccadic slowing. Pyramidal tract signs were present in six patients. This mainly involved hyperreflexia and Babinski reflexes, but not a spastic muscle tone. Vestibulo-ocular reflexes were low or normal for ten patients but were not available for the remaining patients. The core clinical feature of these patients that interfered with gait and balance regulation was the spinocerebellar ataxia.

All subjects were also examined with the Tinetti Mobility Index [Tinetti et al., 1986; Trouillas et al., 1997]. Moreover, ataxia disease stage was determined (see Table 1) and a questionnaire was used to evaluate fall history. More patients than controls fell in the previous 3 months (10 patients versus one control). Fear of falling was also more common in patients (9 patients) than controls (1). All subjects gave witnessed informed and written consent to participate in the experiment according to the Declaration of Helsinki. The Institutional Review Board of the participating centres approved the study.

Protocol

Recording techniques were similar to those of our previous studies (Allum et al., 2008; Bakker et al., 2006). The subject's feet were lightly strapped into heel guides fixed to the surface of a movable platform. The heel guides were adjusted to ensure that the ankle joint axes were aligned with the pitch axis of the platform and prevented stepping reactions when stance perturbations occurred. The roll axis had the same height as the pitch axis and passed between the

Table 1. Patient data

Subject	Sex	SCA type	Age [yr]	Height [cm]	Weight [kg]	Body mass index [kg/m^2]	Duration of disease [yr]	Disease stage [a]	ABC scale [1] [%] [b]	Tinetti Mobility Index [c]	NSC/EMG [2]	Pyramidal tract signs	VOR [3]	Neuroimaging, atrophy of:
1	m	14	60	189	88	24.6	30	1	48	10	- (clin)	-	N.A.	N.A.
2	m	14	54	178	61	19.3	9	2	50	9	- (clin)	-	N.A.	Cerebellum
3	f	6	58	154	70	29.5	8	2	29	10	-	-	N.A.	Cerebellum (vermis)
4	m	1	41	178	92	29.0	5	2	58	8	-	+	N.A.	Cerebellum
5	m	14	54	183	83	24.8	2	2	38	4	- (clin)	-	N.A.	N.A.
6	f	1	51	162	74	28.2	15	1	73	4	subtle neuronopathy	+	N.A.	Cerebellum
7	f	6	51	168	69	24.4	9	2	24	7	minor axonal sensory neuropathy	+	Low	Cerebellum
8	m	6	63	176	78	25.2	3	1	70	5	- (clin)	-	N.A.	Cerebellum (vermis)
9	f	6	59	160	55	21.5	5	1	75	2	- (clin)	-	N.A.	Cerebellum
10	m	1	54	184	67	19.8	12	1	66	9	Normal	+	Normal	Cerebellum
11	m	6	48	179	85	26.5	12	2	38	15	Normal	-	Normal	N.A.
12	m	14	60	168	71	25.2	21	1	74	2	N.A.	-	Normal	N.A.
13	m	2	49	180	67	20.7	14	1	76	7	Neuropathy of upper extremities	+	Low	Cerebellum, pons (subtle)
14	m	14	55	178	75	23.7	26	1	72	3	Normal	-	Normal	Cerebellum (vermis)
15	f	3	32	181	73	22.3	13	2	69	6	N.A.	-	Normal	Cerebellum (vermis)
16	f	2	56	165	70	25.7	10	1	62	7	Neuropathy of upper and lower extremities	-	Normal	Cerebellum
17	m	3	41	185	79	23.1	3	1	57	5	Mild axonal neuropathy	-	Normal	Cerebellum (vermis)
18	m	14	44	172	72	24.3	23	2	68	7	N.A.	+	Normal	N.A.
Patients	Mean	-	**51.7**	**174.4**	**73.8**	**24.3**	**12.2**	-	**58.2**	**6.7**	-	-	-	-
	SEM		**1.9**	**2.3**	**2.2**	**0.7**	**1.9**		**3.9**	**0.8**				
Controls	Mean	-	**50.0**	**173.5**	**70.7**	**23.5**	-	-	**96.0**	**0.4**	-	-	-	-
	SEM		**1.9**	**1.7**	**1.7**	**0.5**			**0.9**	**0.2**				

Abreviations:
N.A. = no data available
[1] ABC = acitivites-specific balance confidence scale
[2] NCS/EMG = nerve conduction studies and electromyography
[3] VOR = vestibulo-ocular reflex
clin = no symptoms on clinical assessment

[a] 0 = normal, 1 = ataxia, but walking independently, 2 = permanently dependent on walking aids, 3 = permanently dependent on wheelchair, 4 = death

[b] no confidence = 0%, optimal confidence = 100%

[c] optimal score = 0, worst score = 28

feet. The stance width was standardized (14 cm) and two handrails were located 40 cm from the sides of the platform centre. Subjects were informed that they were allowed to grasp the handrails if needed. Two assistants were present to lend support in case of a near-fall or 'loss of balance'.

Responses to perturbations in 16 different directions with a constant velocity of 60 deg/s and a constant amplitude of 7.5 deg were investigated using a dual-axis rotating platform. Perturbation directions were defined as pure pitch forward (toes down or 0 deg), pure pitch backward (toes up or 180 deg), pure roll rightward (90 deg), and pure roll leftward (270 deg), and combinations of pitch and roll (directions 23, 45, 68, 113, 135, 158, 203, 225, 248, 293, 315 and 338 deg). One protocol (directions 0, 45, 90, 135, 180, 225, 270 and 315 deg) had been presented to half of the patients and controls in a previous study (Bakker et al., 2006). Data from the Bakker et al. (2006) study was used for the analysis of segment correlations to CoM velocity in the current study. To complete the analysis of this study a second protocol (directions 23, 68, 113, 158, 203, 248, 293 and 338 deg) were presented to the other half of patients and controls. Within a protocol, each perturbation direction was presented in random order eight times to a subject. To reduce the habituation effects, the first trial was excluded from further analysis (Keshner et al., 1987). To minimize fatigue, participants were given a 3-4 minute seated rest after every 32nd trial. Each trial was preceded by a random 5-15s interstimulus delay that was initiated automatically. During this time period, visual feedback of the subjects' own anterior-posterior (AP) and medio-lateral (LR) ankle torque was presented to the subject on a cross with light-emitting diodes at 5 m distance. This visual feedback was used to standardize pre-stimulus subject position across trials.

Data collection

Recordings of biomechanical and EMG data commenced 100 ms prior to perturbation onset and were collected for 1 s. To record EMG activity, pairs of silver-silver chloride electrodes were placed approximately 3 cm apart along the muscle bellies of left tibialis anterior (TA), left soleus (Sol), left peroneus longus (PL), left rectus femoris (RF), left biceps femoris (BF), left gluteus medius (GM), left medial deltoid (DM; pars acromalis) and bilaterally on paraspinals (Para) at the L1-L2 level of the spine. EMG recordings were band-pass analog filtered between 60 and 600 Hz, full-wave rectified, and low-pass filtered at 100 Hz prior to sampling at 1 kHz.

Full body kinematics were collected using a three-dimensional optical tracking system with 21 infrared-emitting diodes (IREDs) (Optotrak, Northern Digital). The Optotrak cameras were placed approximately 5 meters in front of the subject and sampled the IRED signals at 64 Hz. IREDs were placed bilaterally on the following anatomical landmarks: frontally at the

lateral malleolus; centre of patella; frontal greater trochanter; anterior superior iliac spine; radial styloid process; elbow axis; acromion; chin; angulus sterni; and 2 on a headband placed just above the ears. Three IREDs were placed at the front corners and the left side of the platform to define the pitch and roll movements of the platform. Subjects wore tight fitting shorts and vests to reduce marker movements with respect to skin.

Data analysis

Primary variables of interest were COM displacement, knee flexion, arm abduction, trunk pitch flexion, and muscle responses of the legs, arms and the trunk.

Following analogue to digital data conversion, biomechanical (potentiometer measures of platform rotation) and EMG signals were averaged offline across each perturbation direction. Zero latency was defined as the onset of platform rotation measured with the potentiometers. Subject averages were pooled to produce population averages for a single direction.

EMG analysis

Muscle background activity levels were evaluated by computing the absolute means of EMG signals over the pre-trigger interval of 90 ms ending 10 ms prior stimulus onset. EMG response amplitudes were analysed by computing the mean of muscle activity within a certain post-stimulus interval compared to the pre-stimulus background activity level of the muscle.

Kinematic analysis

IRED position data were digitally filtered at 16 Hz using a zero phase shift 4th order Butterworth filter. Total body COM displacement was calculated separately for the anterior-posterior (AP), lateral (Lat) and vertical (Vert) directions using a 12 body segment adaptation (Visser et al., 2008) of a 14 segment model of the human body (Winter et al., 2003). In addition, we calculated the following angular displacements: knee angle (left and right), absolute trunk angle (roll and pitch) and upper arm abduction angle (left and right). Knee angles were calculated as the angle spanned by the two unit vectors of the upper and lower legs. Absolute rotation angles of the planes defined by trunk and the platform support surface were defined using 3 or 4 markers on these segments. Rotations of the upper arm with respect to the trunk and the upper leg with respect to the pelvis were calculated as ball joint angles defined by two angels of spherical coordinates, e.g. for the upper arm by arm rotation and abduction.

Stimulus induced changes were calculated with respect to values averaged over a pre-trigger time inter-

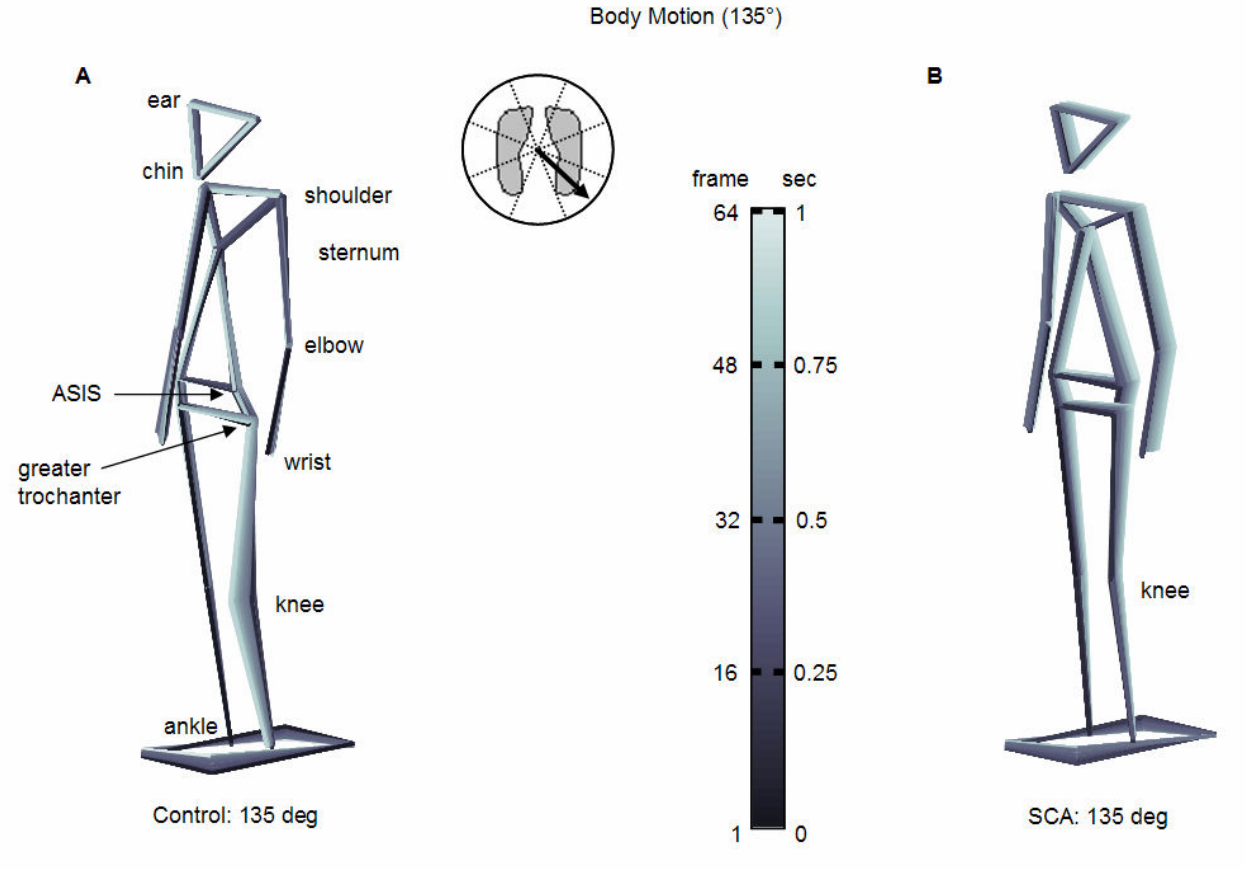

Figure 1: Stick figure representations of the movements of a healthy control (A) and a SCA patient (B) in response to a backward-right perturbation (direction 135 deg). 64 frames (16 ms per frame) of the recording are shown with platform movement starting at frame 6. The view is shown looking from front right. Note the differences in uphill (right) knee flexion and trunk motion.

val of 90 ms ending 10 ms prior stimulus onset. To investigate the influence of knee and arm movements on the COM velocity, we first calculated the uphill and downhill of knee flexion velocities and arm abduction velocities respectively, and then difference velocities between uphill and downhill segments. COM velocity peaks in LR and AP directions were first identified in the population averages.

Statistics

Our primary analysis concentrated on between-groups comparisons of SCA patients and controls using a repeated measures ANOVA model (group × direction) for both kinematic and EMG data. Significant main group effects were further explored with one-way ANOVAs. Results with $P < 0.05$ after Bonferroni corrections were considered significant. Calculations were done with SPSS® using a general linear model with repeated measurements and one-way ANOVA, respectively.

Results

In each perturbation direction the reaction of 9 patients was investigated pursuant to the two protocols described in methods. Both healthy subjects and SCA patients had COM displacements in the direction of platform tilt. Thus, for backward right perturbations, the total body COM was displaced backward and to the right. Figure 1 shows the body sway of a healthy control (A) and a SCA patient (B) for a platform backward right tilt. In the control, the uphill knee is flexed and the trunk and head rolled slightly more uphill, whereas SCA patient shows less uphill knee flexion, greater arm motion, and clear downhill movement of the trunk. Figure 2 illustrates examples of average sample population COM displacement and velocity traces for roll and pitch directed displacements. The variation for each of sample populations is also shown, documenting the similarity of responses within each population.

COM

The total body COM movement of SCA patients showed a greater displacement than controls in the direction of tilt in Lat as well as in AP directions (Fig. 2 A & B). The COM movement of controls plateaued around 600 ms. However, the COM of patients continued to move in the direction of perturbation. The findings were consistent across all patients and are similar to those reported by Bakker et al. (2006). A

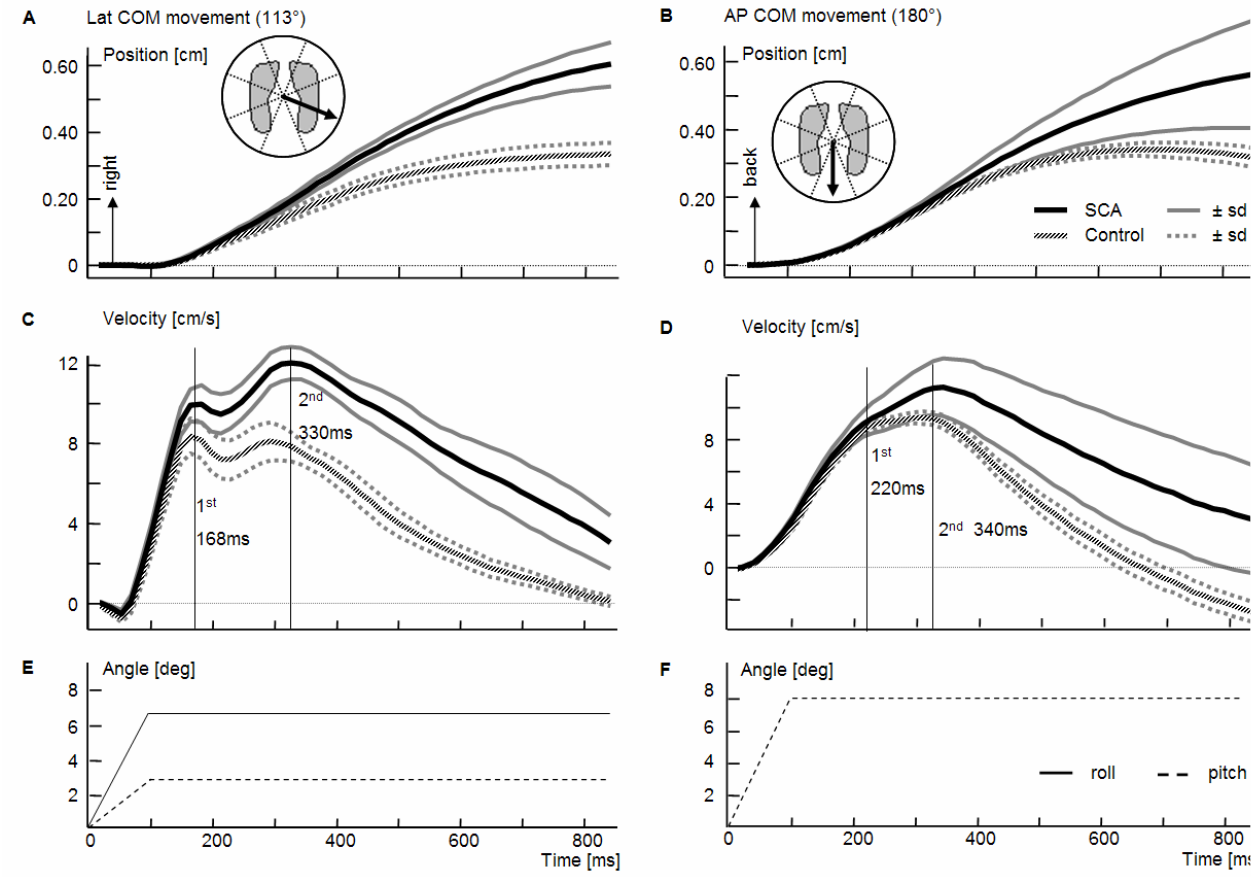

Figure 2: Mean population COM (centre of mass) motion in the medial-lateral (Lat; A, C) and anterior-posterior (AP; B, D) directions. Mean population traces of SCA patients and controls are shown for the displacement (upper traces) and the velocity (middle traces) of the COM. 0 ms marks the stimulus onset (see lower set of traces of platform angle in E & F). Vertical lines marked 1st and 2nd indicate the relative maximum values of COM velocity. Inserts in A and B indicate the direction of downward platform tilt for the traces.

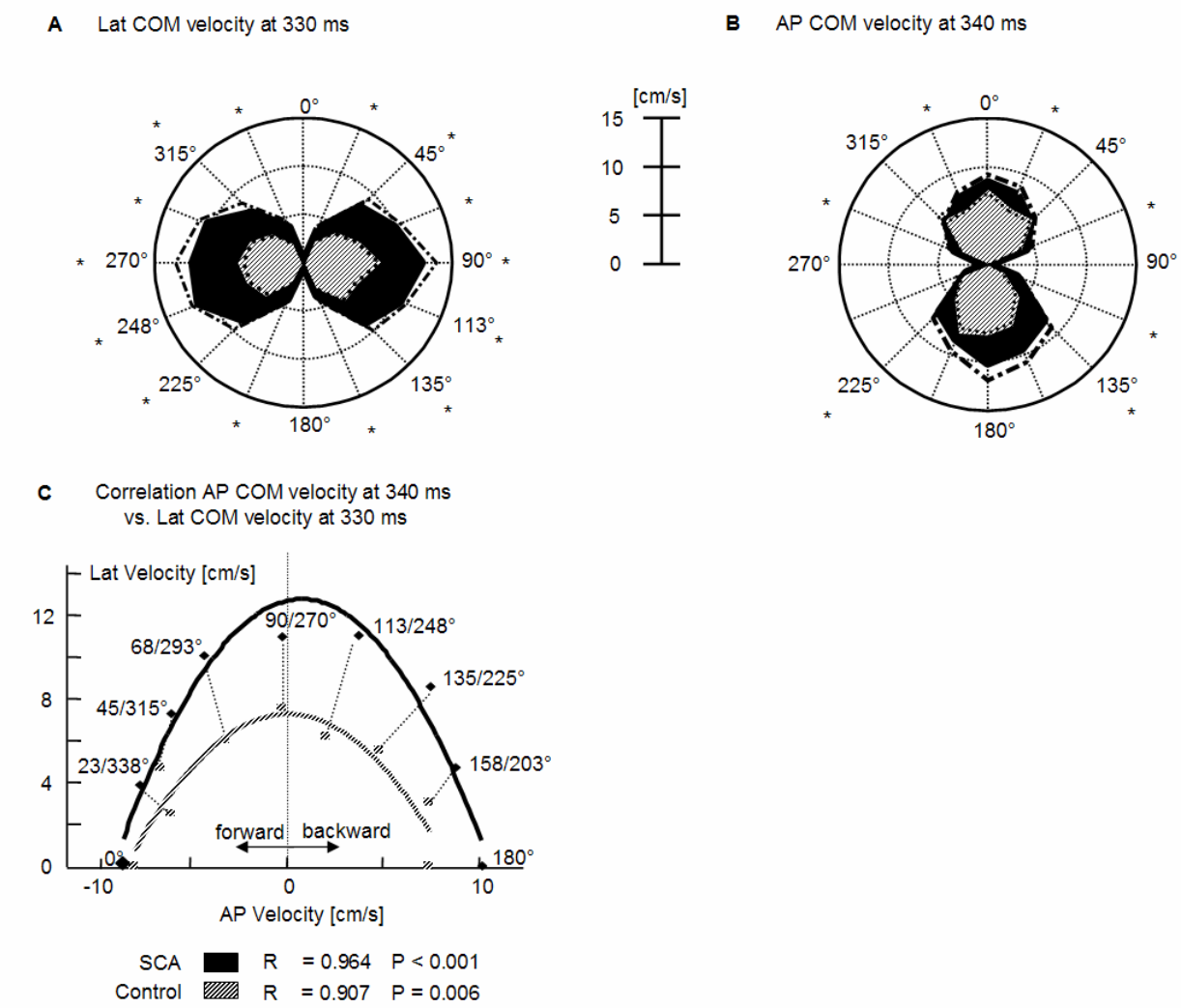

Figure 3: Mean population COM horizontal velocity. COM Lat (A: at 330 ms post stimulus) & AP (B: at 340 ms post stimulus) horizontal velocity for both SCA patients and controls are shown in polar plots for all perturbation directions. The direction of each radial line represents one of the 16 perturbation directions and the amplitude of COM velocity is plotted as the amplitude along the radial line. Asteriks (*) indicate $P < 0.05$ (significant post hoc comparisons of patients versus controls). Panel C illustrates the curvilinear correlations between Lat & AP COM velocities over all perturbation directions. Responses for identical directions of left and right roll are pooled.

clear group effect was found in the average COM displacement between 750 ms to 850 ms post stimulus in the Lat direction [$F(1,31) = 226.04$; $P < 0.001$]. Post hoc tests indicated that Lat COM displacement increased more in SCA patients with respect to controls with increased roll component of the stimulus. For AP displacements at 750 to 850 ms, statistical analysis indicated that the AP COM displacement was differently modulated by perturbation direction between controls and SCA patients [$F(1,8) = 5.03$; $P < 0.001$]. For backward tilts of the platform, patients moved their COM further in the direction of tilt than controls [$F(1,18) = 24.32$; $P < 0.001$]. In forward perturbation directions, patients showed a similar range of COM displacements as controls [$F(1,19) = 1.40$; $P = 0.252$].

As shown in figure 2C, Lat COM velocity traces peaked at two times. The amplitude of the first peak in COM velocity at 168 ms (± 2 ms) appeared to be slightly larger for the patients compared to controls. However, no significant differences were detected. The divergence between patient and control COM velocity traces subsequently increased with patients having a second peak at 330 ms (± 3 ms) post stimulus. At this time a clear group effect in the amplitude of COM velocity was present [$F(1,31) = 36.12$; $P < 0.001$]. This second peak could be clearly identified in patients, whereas in several controls this peak was not observed at all.

As the divergence between population COM velocity across perturbations was greatest at 330 ms, this velocity measure and a correlated measure of AP COM velocity at 340 ms (see below) were used as our primary outcome measures. AP COM velocity of controls plateaued at around 220 ms post stimulus (Fig. 2D), whereas the AP COM velocity of the SCA patients continued to increase and reached a maximal value at 340 ms (± 8 ms). At 220 ms post stimulus no significant differences in AP COM velocity emerged between the two groups [$F(1,18) = 0.12$; $P = 0.747$]. However, at 340 ms the AP COM velocity for the patients was clearly greater than for controls [$F(1,18)$

= 8.14; P = 0.011]. At this time point AP COM velocity of controls was decreasing again (Fig. 2D).

The differences in Lat and AP COM velocities between populations at 330 and 340 ms, respectively, are illustrated in the polar plots of figure 3A & B. Significant post-hoc group differences for each direction are marked with an asterisk (SCA > controls). Noticeably, Lat COM velocity was greater in SCA patients for all directions except those of pure pitch, whereas AP COM velocity differences were less pronounced and significant just in some of the pitch directions. Because the peaks at 330 and 340 ms in Lat, AP COM velocity respectively appear to be closely time correlated, we performed curvilinear regression analysis on the amplitudes. Figure 3C shows the correlations between Lat and AP COM velocities in the different directions. In both groups high regression values and significance was detected with separation in the regression lines being greatest for lateral directions and smallest for forward directions (figure 3C).

Correlations of body segment responses to Lat COM velocity for roll perturbations

Trunk

For perturbations with a roll component, the legs were initially displaced in the same direction as the platform tilt, while the upper body counter-rotated in the opposite direction. The initial counter-rotation of the trunk was present in both groups (Fig. 4A). However, at around 120 ms, patients trunk roll velocity peaked and after 150 ms reversed to the downhill (destabilizing) side, whereas controls continued to increase trunk roll velocity into the stabilizing direction. Therefore, at 150 ms a clear group difference in trunk roll velocity could be detected [F(1,34) = 9.80; P = 0.004]. Furthermore, at 230 ms (see vertical lines in Fig. 4A) the destabilizing trunk roll velocity peaked in patients, and controls showed a trunk roll velocity near zero. Again a group difference was evident [F(1,34) = 34.80; P < 0.001]. A comparison of trunk roll velocity at 230 ms and Lat COM velocity at 330 ms revealed a highly significant regression in patients and an insignificant zero slope in controls (Fig. 4B). Thus for patients, greater trunk roll velocity downhill (destabilizing) was associated with greater Lat COM downhill velocity.

Knees

The appropriate response to aid the initial trunk flexion uphill and prevent downhill trunk motion is to flex the uphill knee and extend the downhill knee 2006), SCA patients showed less knee flexion of the uphill knee and a small flexion in the downhill knee instead of extension (Figure 5D). In order to analyse the effect of differences in knee flexion on COM motion we calculated the difference between the knee flexion of the uphill and the downhill knee as shown in figure 5 A & B. The difference of the left and right

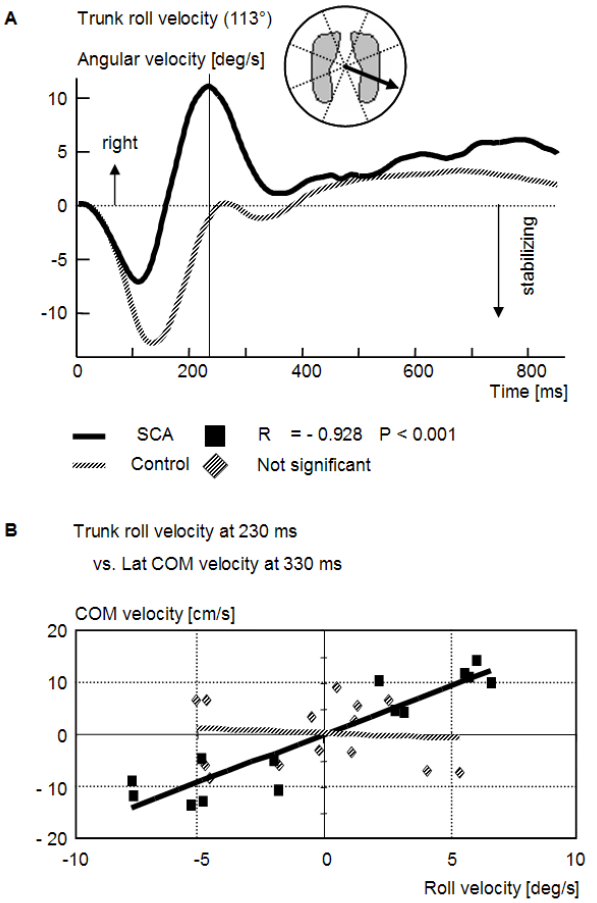

Figure 4: Trunk roll velocity. Mean population traces of SCA patients and controls for trunk roll angular velocities in response to a backward-right (A) perturbation is shown. The black vertical line at 230 ms marks the time of maximum destabilizing trunk roll velocity for the patients. Panel B illustrates the regressions between trunk roll velocities at 230 ms and the COM LR horizontal velocities at 330 ms over all roll perturbation directions.

knee angles provides information about the stabilizing effects of differential knee flexion due to insufficient uphill or downhill knee action. Controls showed a greater difference between left and right knee flexion between 300 ms and 700 ms (Fig. 5A), which was replicated in flexion velocity traces (Fig. 5B). The difference in flexion velocity peaked at 300 ms post stimulus in controls (Fig. 5B). The peak was less clear for SCA patients (see vertical line in Fig. 5B). A group x direction interaction [F(1,6) = 3.21; P = 0.005] indicated that at 300 ms (uphill–downhill) knee flexion velocity was differently modulated by perturbation direction in SCA patients with respect to controls with forward and roll directions showing the greatest post-hoc differences. These differences are illustrated by the regression analysis in figure 5C. Both patients and controls show significant correlations between differential knee flexion velocity at 300 ms and Lat COM velocity at 330 ms. The differences in the regression slopes indicate that controls achieved lower Lat COM velocity by utilising greater knee flexion.

Arms

As expected, roll perturbations initially caused an abduction in the uphill arm and an adduction of the downhill arm in both groups (Allum et al 2002). The overall effect of arm movement on total body COM is based on the difference in arm motion. Thus, to analyse the effect of arm motion on COM motion, we calculated the difference between the uphill and downhill arm abduction velocities. More abduction in the uphill arm with respect to the downhill arm is considered as a positive sign. Arm velocity difference showed a peak at ca. 150 ms (Fig. 6A). At this point in time controls had a greater velocity difference and therefore a more stabilising arm motion [F(1,34) = 4.40; P = 0.044] than SCA patients. Patients arm velocity reversed after 200 ms and again showed stabilizing arm motion which peaked at 330 ms (see vertical line at 330 ms in Fig. 6A). This action was contrary to that of controls who showed a tendency for destabilizing arm movements at this time point and yielded a group effect on velocity: F(1,34) = 10.74; P = 0.002 (Fig. 6B). Analysis of the uphill and downhill components of the difference in arm abduction velocities revealed that for most roll directions the uphill arm provided most stability in SCA patients (Fig. 6D). For pure roll perturbations the lower arm abduction also aided stabililty (Fig. 6D). A regression between arm abduction velocity difference at 330 ms and the peak in Lat COM velocity at 330 ms revealed a highly significant correlation in SCA patients but not in controls, indicating that patient arm motion at 330ms positively influences the peak of COM velocity at 330 ms (Fig. 6C).

Given that trunk velocity at 230 ms, the difference in arm abduction at 330 ms were all correlated with changes in Lat COM velocity at 330 ms, we performed a multivariate stepwise regression analysis on these variables for SCA patients. We determined knee flexion velocity at 300 ms, and the difference in that reduced differential knee velocity had a greater negative influence on Lat COM velocity than trunk velocity at 230 ms and confirmed the positive influence of differential arm abduction at 330 ms.

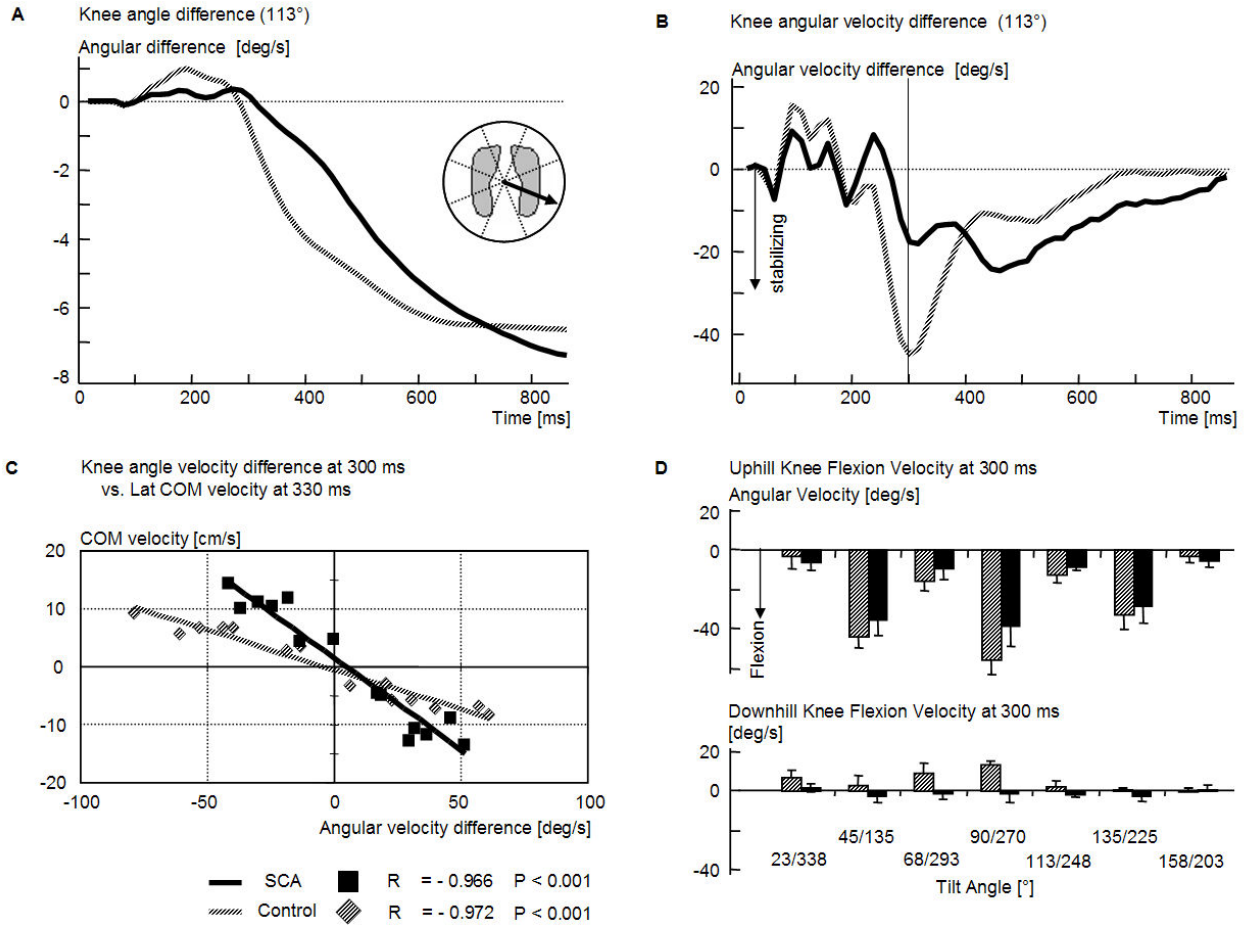

Figure 5: Mean population differences in left and right knee flexion. Mean population traces are shown for the difference of left and right knee angle (A) and knee angle velocity (B) to a 113° perturbation (right and slightly backward). The black vertical line in B marks the maximum flexion velocity at 300 ms found in healthy controls. Panel C illustrates the regressions between knee angle velocity differences at 300 ms and the COM Lat horizontal velocities at 330 ms over all roll perturbation directions. D Uphill and downhill knee velocities at 300 ms used to yield the regression shown in C

Correlations of body segment responses to AP COM motion for pitch perturbations

Trunk

Trunk motion of SCA patients was clearly different compared to controls (Fig. 7A & B). Controls extended the trunk slightly for forward perturbations (7A) and flexed the trunk for backwards perturbations (7B). Regardless of perturbation direction, the initial (over the first 200 ms) trunk movements of SCA patients were similar to controls. However, SCA patients flexed the trunk forward after 300 ms for all perturbation directions (compare Fig. 7A & B). Thus patients showed more trunk forward pitch between 500 – 600 ms than controls across perturbation directions [$F(1,19) = 86.80$; $P < 0.001$] (Fig. 7C). With forward trunk flexion, both SCA patients and controls moved their pelvis backwards resulting in a high correlation between AP COM velocity at 340 ms and a peak in pelvis AP velocity at 270 ms ($R^2 = 0.97$, not shown). SCA patients showed, however, greater backward movement of the pelvis [$F(1,19) = 44.31$; $P < 0.001$] over the interval 500 – 600 ms with greatest post-hoc differences for backward directions (Fig. 7D).

Knees

Given the relationship between Lat and AP COM velocity as depicted in figure 3C and the influence of differential knee flexion (as illustrated in Fig. 5C) on Lat COM velocity, we expected that summed left and right knee flexion velocity would be positively related to the peak in AP velocity at 340 ms, and that this relation would be different for patients and controls. The summed knee flexion velocity (divided by 2) between 200 ms – 300 ms post stimulus showed a group difference [$F(1,19) = 5.24$; $P = 0.034$]. Furthermore, compared to controls, patients showed less mean knee flexion velocity at its peak of 220 ms (see vertical line in Fig. 8A) in all directions [$F(1,19) = 6.85$; $P = 0.017$]. Knee flexion velocity at this time was highly correlated to AP COM velocity at 340 ms

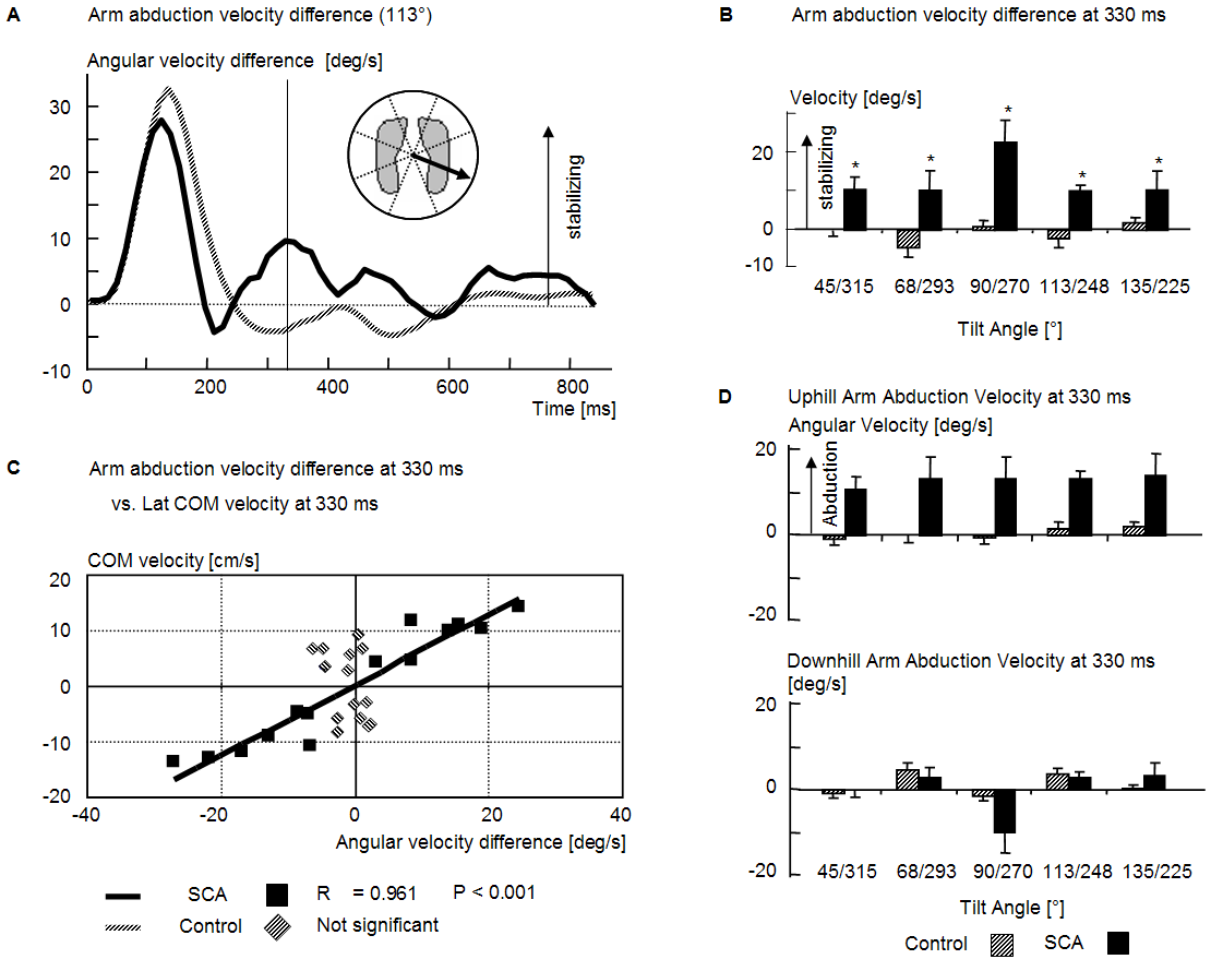

Figure 6: Mean population differences in left and right arm abduction. Mean population traces are shown for the velocity difference of left and right arm abduction to a backward-right perturbation, direction 113° (A). The black vertical line marks the second maximum present at 330 ms in the SCA patient data. Average velocity differences at this time (mean and SEM) for all roll perturbation directions are shown in B. Data with equal but opposite directions of roll are pooled. * indicates $P < 0.05$ post hoc comparison of patients versus controls are significant. Panel C illustrates the regressions between arm angle velocity differences at 330 ms and the COM LR horizontal velocities at 330 ms over all roll perturbation directions. D uphill and downhill mean arm abduction velocities (and SEM) used to compute results shown in B and C.

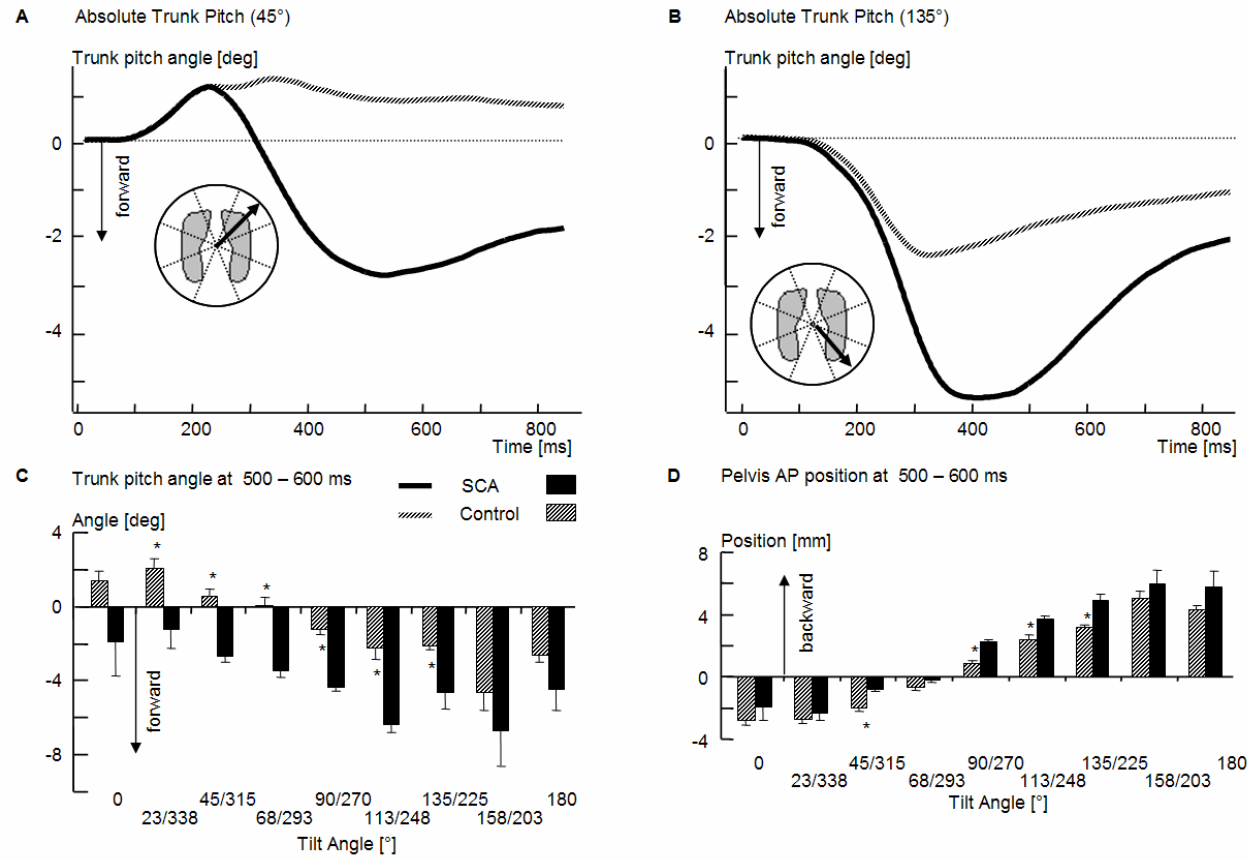

Figure 7: Trunk pitch angle and pelvis horizontal position. Mean population traces are shown of absolute trunk pitch in response to a forward-right, direction 45 deg, (A) and a backward-right, direction 135 (B) perturbation. Mean trunk pitch angles (C) and mean pelvis anterior-posterior position at 500 ms – 600 ms post stimulus onset (mean and SEM) for all perturbation directions are shown (C and D).

(Fig. 8B). Thus, in both populations, increased knee flexion was associated with increased AP COM velocities. However the difference in slopes of the regressions [P<0.001] indicated that a given AP COM velocity was achieved by greater knee flexion velocities in controls than in SCA (see Fig. 8C).

We investigated with multi-variate regression analysis the relative influence of trunk and knee flexion on AP COM velocity. Trunk flexion velocity peaking at 290 ms had a greater negative influence on AP COM velocity.

EMG responses

It is well known that the amplitude of balance correcting and preceding stretch reflex EMG responses increases with the level of background EMG activity (Allum 1983, Diener et al 1984, Weiss et al 1988, Sinkjaer et al 1988). Thus the question arose whether or not changes in background activity influenced SCA balance correcting responses, as baseline activity was higher in patients than controls (see Fig. 9). Figure 10 A & B show examples of the typical response pattern observed in paraspinal muscles following a left tilt of the support-surface. In patients, activation of the right (uphill) muscle (with respect to background activity prior to stimulus onset) was less than controls thus not aiding to maintain the trunk uphill as effectively. However, activity in the downhill muscle, which would have caused downhill tilt of the trunk, was also less. This effect was observed across all backward directions for the individual muscles (10 C & D). The sum of the left and right paraspinal activation was also less in SCA patients during the 100 – 200 ms balance correcting period than in controls [group effect: $F(1,15) = 4.75$; $P = 0.046$]. Remarkably, no group effect was observed for early reflex responses measured between 30 and 100 ms. Early balance correcting responses were also decreased for left gluteus medius muscle in the patients [$F(1,16) = 9.845$; $P = 0.006$].

The typical pattern noted in lower leg muscles of SCA patients in earlier studies (Diener et al 1984, Horak and Diener 1994, Bakker et al 2006), i.e. a reduced early balance correcting response followed by higher levels of later (post 400 ms) activity, was also observed in the rectus femoris (RF) or quadriceps muscle in SCA patients in this study. EMG activity of the upper leg muscle RF showed higher baseline activity than controls (Fig. 11A & B) but similar balance correcting responses (Fig. 11C). Later stabilizing responses in the RF of the downhill leg were much greater in patients (Fig. 11D) [direction x group effect: $F(1,16) = 3.81$; $P = 0.016$].

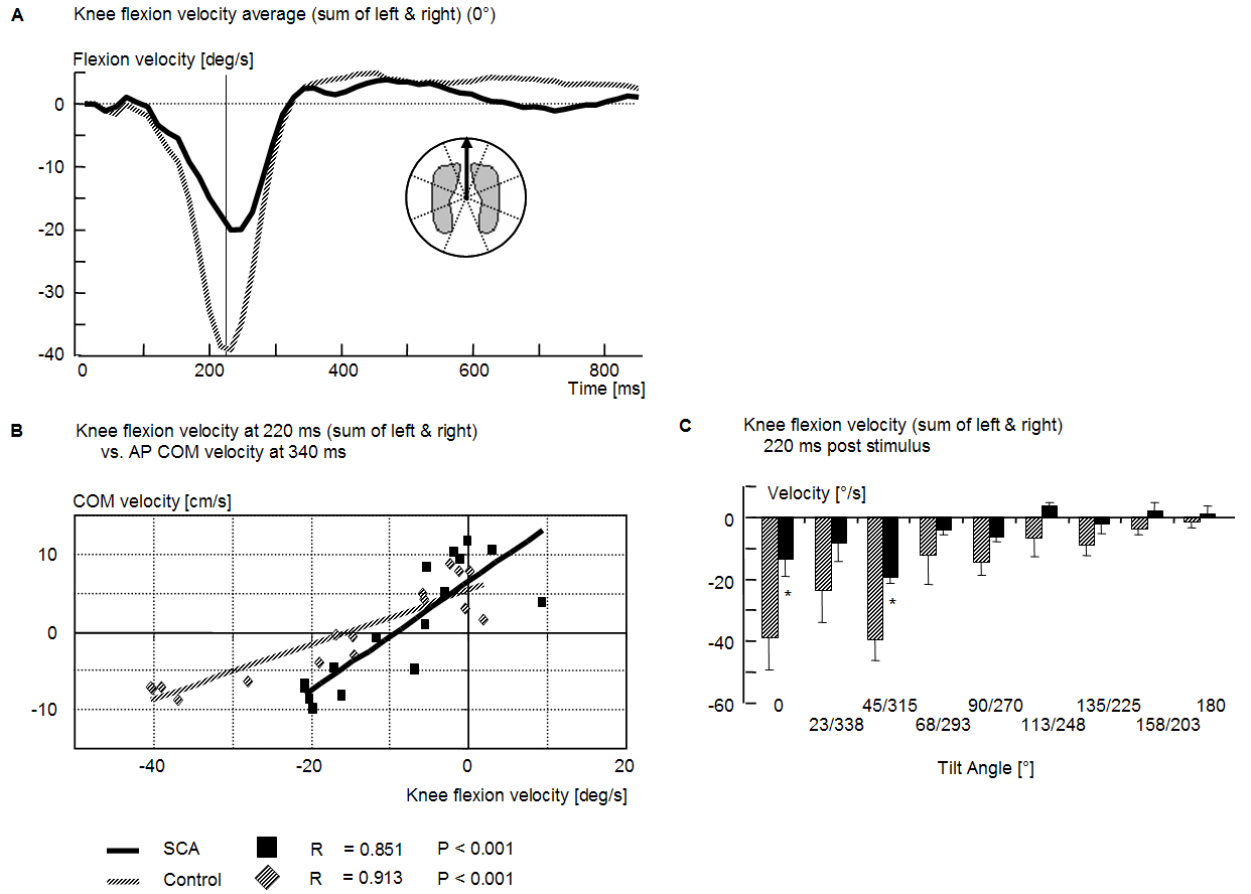

Figure 8: Mean population sum of left and right knee flexion velocity. Population traces of both groups are shown for the sum of left and right knee angular velocity for pure pitch forward, direction 0 deg (A). The black vertical line marks the peak flexion velocity for SCA patients and controls. Panel B illustrates the regression between the knee angular velocity sum at 220 ms and the AP COM at 340 ms for all perturbation directions. Average angular velocities at 220 ms (mean + SEM) for all perturbation directions (C).

In the biceps femoris (BF) or hamstrings, no significant differences in EMG responses amplitudes were observed. Patients also showed increased late 400 ms – 700 ms balance stabilising responses in the following muscles:tibialis anterior (TA) [$F(1,16) = 12.554$; $P = 0.003$], Soleus [$F(1,16) = 4.671$; $P = 0.046$], and Deltoid [$F(1,16) = 13.659$; $P = 0.002$].

Discussion

This study investigated body segment movements critically related to instability of SCA patients, and whether active muscle mechanisms underlie such movements. The primary findings were that initial trunk motion downhill and insufficient uphill knee flexion were directly correlated with lateral instability observed in COM velocity profiles. Reduced knee flexion had the greatest influence on COM instability. Although decreased knee flexion in SCA patients has been described before (Bakker et al., 2006), this study provides the first direct correlates of knee flexion to the COM lateral instability. Interestingly, the large arm movements in SCA patients appear, based on correlation analysis, to stabilise rather than destabilise COM motion, as originally assumed (Bakker et al., 2006). Recordings of knee and trunk muscle activity yielded no active correlates to reduced knee flexion and trunk hypermetria. Instead, we noted generally lower amplitudes of balance correcting responses in these muscles despite higher muscle background activity. Thus our general conclusion is that most of the *lateral* trunk hypermetria in SCA is caused by increased pelvic and knee muscle stiffness resulting from higher background muscle activity and not from mal-tuned balance correcting activity.

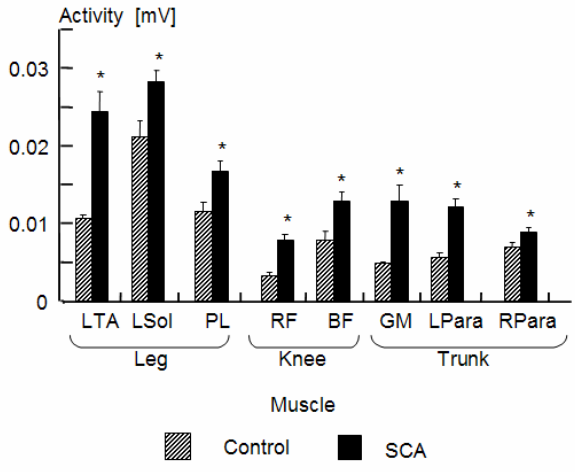

Figure 9: Muscle baseline activity. Mean baseline activity (mean and SEM) measured prior to all stimuli for leg, knee and trunk muscles.* P < 0.05 (post hoc comparison of patients versus controls).

Some features of SCA patients have been observed in healthy subjects fitted with stiffening corsets at the pelvis or trunk (Grüneberg et al., 2004) or at the knees (Oude Nijhuis et al., 2008). Other similarities have also been observed in vestibular loss patients (Allum et al., 2008), in elderly controls (Allum et al., 2002), and in cerebellar ataxia patients (Mummel et al., 1998). The key issue is to what extent comparisons with these previous studies reveal insights into basic aetiology of instabilities, compensatory strategies, or instabilities caused by the compensatory strategies. This issue is particularly important when considering correlations between insufficient knee flexion and either lateral or AP instability. Another issue is the relationship expected between maltuned balance correcting responses and lateral instability. We were unable to establish such a relationship in SCA patients. The key feature of SCA instability seems to be increased knee and pelvis rigidity, which appears to change the dynamic response to lateral perturbations drastically. Patients partially compensate using arm movements, but appear to downscale balance-correcting responses.

Lateral instability

Trunk flexion

The earliest segment movement clearly correlated with COM lateral instability in SCA patients was a reversal of the initial uphill trunk lateral flexion to downhill lateral flexion. Even though downhill motion peaked after the onset of balance-correcting responses in paraspinal and gluteus medius muscles in SCA patients, there was no indication that this muscle activity was responsible for the reversal in trunk motion – a reversal not observed in healthy controls. Activity in downhill paraspinal muscles was less than in controls. Indeed, this decreased muscle activity is a common feature of total leg proprioceptive loss patients who also show a reversal of trunk motion to downhill (Bloem et al., 2002), and young normals fitted with a trunk corset (Grüneberg et al., 2004) who only flexed the trunk downhill. Healthy elderly also show an early reversal of trunk motion to downhill (Allum et al., 2002). Hence, the most parsimonious explanation for the early reversal in trunk motion in SCA is that it follows the common feature of the aforementioned populations: an increased leg and

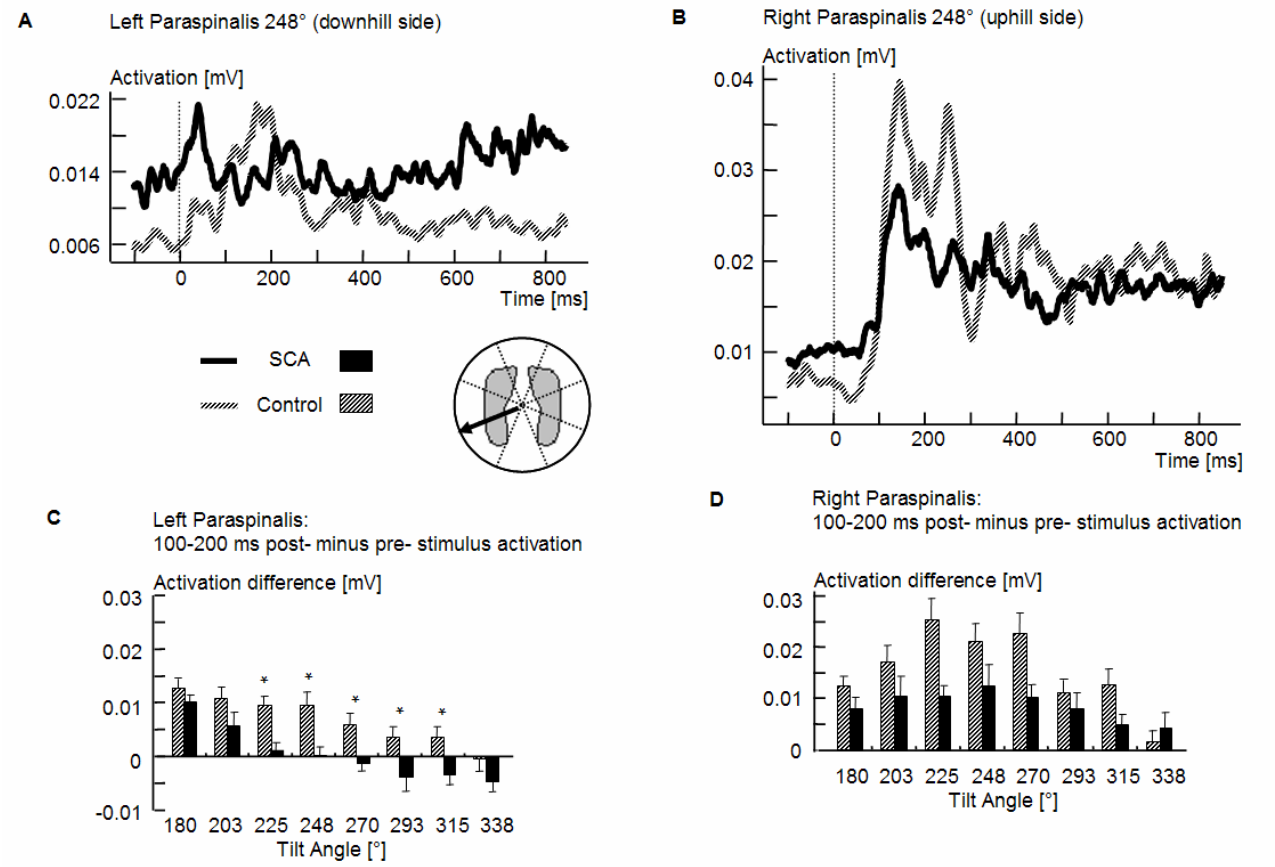

Figure 10: Paraspinalis responses. Mean population traces of both groups are shown for left (A) and right (B) M. paraspinalis in response to a backward-left perturbation. Panels C and D illustrates the averages (and SEM) of muscle activation between 100 ms – 200 ms corrected for baseline activity. Asteriks (*) indicates P < 0.05 (post hoc comparison of patients versus controls).

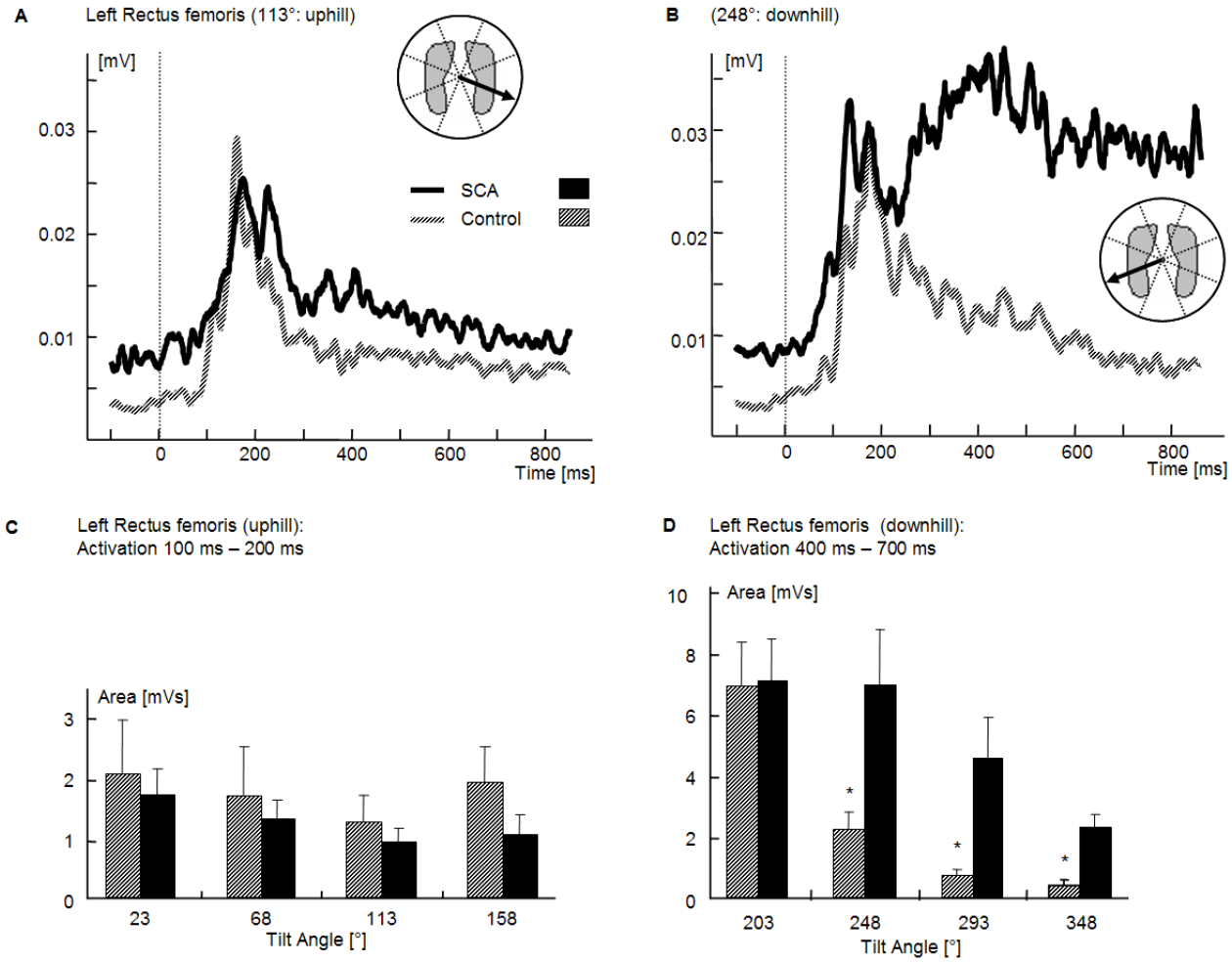

Figure 11: Quadriceps responses. Mean population traces of both groups are shown for the left M. Rectus femoris in response to a backward-right (A) and a backward-left perturbation (B). Average activity between 100 ms- 200 ms (C: mean and SEM) and 400 ms - 700 ms (D: mean and SEM) across directions is shown. * P < 0.05 (post hoc comparison of patients versus controls).

axial muscle stiffness at the level of the pelvis. In SCA patients this appears to result from raised muscle background activity levels (see Fig. 9), that is, an active stiffening prior to the perturbation, and not a reactive stiffening. The lateral truncal hypermetria occurs because support surface tilt is more directly transmitted to the trunk in the form of a whiplash effect and because of uphill balance correcting responses in paraspinal muscles that are down-regulated.

Even though pelvis and leg muscle stiffness appears to be the main cause of the lateral COM instability, the question arises why the uphill trunk muscles are not activated more than controls. We noted less activation over balance correcting response intervals (100 – 200 ms after the onset of tilt) in both the uphill and downhill paraspinal muscles. Less activity in the downhill muscles and more in the uphill muscles would be consistent with a compensation strategy for stiff knees. The lack of increased activation in the uphill paraspinal muscles (Fig. 10C) is surprising given the increased background activity in these muscles. As stretch reflexes were also not increased in amplitude (with respect to background activity levels), we assume that stretch reflexes and balance correcting responses are down-regulated in SCA. Normally, both increase with increased background activity (Allum and Mauritz 1984; Allum and Pfaltz 1985; Diener et al., 1982; Sinkjaer et al., 1988; Weiss et al., 1988).

Reduced knee flexion

Reduced knee flexion in SCA patients was correlated with increased lateral movement of the COM downhill. Both the lack of uphill knee flexion and downhill knee extension contribute to lateral instability. Without an appropriate bilateral knee response, support surface motion is more directly transmitted to the pelvis which is then displaced more downhill. Similar instability was noted in healthy subjects whose knees were bilaterally stiffened using fitted casts (Oude Nijhuis et al., 2008). These healthy subjects had no increased lateral instability compared to subjects without casts due to two compensating mechanisms. Firstly, counter rolling of the trunk with respect to pelvis rotation was greater and thus no differences in trunk roll movement were observed (Oude Nijhuis et al., 2008). Secondly, compensatory arm movements compensated for the increased pelvis roll movement. Counter rolling trunk motion was not present in SCA patients.

We did not observe inappropriate muscular responses as the cause for insufficient knee flexion in SCA patients. Rather, the increased background activity in knee flexors and extensors seems to cause knee rigidity. Measurements of early balance correcting responses (100 – 200 ms) revealed no increases in the amplitudes in SCA subjects despite the greater background activity (Fig. 9 & 11). If anything, there was a trend for lower balance correcting activity in quadriceps muscles. In order to be consistent with an active mechanism causing insufficient knee flexion in the uphill knee, decreased tibialis anterior and hamstrings and increased quadriceps activity should be observed. Increased activity was indeed observed but only after 300ms in the downhill quadriceps muscle (fig. 11 D). Such activity is consistent with the patients attempting to correct their lateral instability, rather than enhancing it.

We can only speculate why patients with SCA have increased background muscle activity. One explanation would be fear of falling, which was indeed increased in our SCA patients and is known to be associated with an active stiffening strategy (Carpenter et al. 2004). A second explanation would be that SCA patients 'lock' their knees to decrease the number of degrees of freedom to be controlled, facilitating their postural control strategy.

Arm Abduction

Arm responses are initially part of the body's early biomechanical response to a tilt stimulus (Allum et al., 2002) and subsequently become an integral part of the balance correcting response (Allum et al., 2002; Mcillroy and Maki, 1995). Some authors have considered the overall effect of arm movements on COM displacements to be small (Patla et al., 2002; Pozzo et al., 2001). Grin et al. (2007) noted however that arm movements significantly change the amplitude of COM velocity. In this study, we correlated arm abduction velocities with lateral COM velocity. Interestingly, these correlations indicated an attempt by SCA patients to stabilise their COM velocity using arm movements.

AP instability

Increased forward flexion of the trunk adopted by SCA patients has been termed a compensatory strategy (Bakker et al., 2006). This strategy leads to excessive posterior movement of pelvis even if it compensates for insufficient knee flexion following anterior perturbations of the COM. Two features suggest that the trunk flexion is a "compensatory" strategy. First, although both a stiffened pelvis (Grüneberg et al., 2004) and stiffened knees (Oude Nijhuis et al., 2008) lead to increased trunk forward flexion for backward perturbations of the support surface, this is not the case for forwards perturbations thereby yielding a major difference to SCA patients. If the increased trunk hypermetria resulted from lower-body stiffness, then the direction of hypermetria for forward displacements should be trunk extension and not flexion.

For forward platform tilt, bilateral knee flexion is normally required. If knee flexion is not possible in healthy subjects, then a compensatory strategy used is forward flexion of the pelvis and trunk (Oude Nijhuis et al., 2008). This thrusts the pelvis and COM backwards, countering the forward tilt of the support surface. SCA patients employ this strategy for both forward and backward tilts (Fig. 8) even though knee flexion is only insufficient for forward directed tilts. We assume that this is an active mechanism involving reduced balance correcting but increased late stabilising activity observed in paraspinal muscles (Fig. 10).

Our current findings indicate that most instability in SCA patients is due to intrinsic muscle stiffness at the level of the knees and pelvis. Questions can be raised concerning which aspects of postural control in these patients should receive more attention based on our results. Clearly it would help to train the use of compensatory arm movements as SCA patients appear to naturally rely on these for stability. Secondly, attempts should be made to "de-train" the trunk flexion response to backward perturbations. The main cause of lateral instability in SCA patients, as was shown by our regression analysis, is the lack of uphill knee flexion. Thus, the primary focus should be on reducing the fear of falling and associated stiffness due to increased background muscle activity, thereby permitting increased uphill knee flexion.

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Control of Roll and Pitch Motion during multi-directional Balance Perturbations

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Control of roll and pitch motion during multi-directional balance perturbations

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Abstract

Does the central nervous system (CNS) independently control roll and pitch movements of the human body during balance corrections? To help provide an answer to this question, we perturbed the balance of 16 young healthy subjects using multi-directional rotations of the support surface. All rotations had pitch and roll components, for which either the roll (DR) or the pitch (DP) component were delayed by 150 ms or not at all (ND). Outcome measures were biomechanical responses of the body and surface EMG activity of several muscles.

Across all perturbation directions, DR caused equally delayed shifts (150 ms) in peak lateral centre of mass (COM) velocity. Across directions, DP did not cause equally delayed shifts in anterior-posterior COM velocity. After 300 ms however, the vector direction of COM velocity was similar to the ND directions. Trunk, arm and knee joint rotations followed this roll compared to pitch pattern but were different from ND rotation synergies after 300 ms, suggesting inter-segmental compensation for the delay effects. Balance correcting responses of muscles demonstrated both roll and pitch directed components regardless of axial alignment. We categorised muscles into three groups: pitch oriented, roll oriented and mixed based on their responses to DR and DP. Lower leg muscles were pitch oriented, trunk muscles roll oriented, and knee and arm muscles mixed.

The results of this study suggest that roll, but not pitch components, of balance correcting movement strategies and muscle synergies are separately programmed by the CNS. Reliance on differentially activated arm and knee muscles to correct roll perturbations reveals a dependence of the pitch response on that of roll, possibly due to biomechanical constraints, and accounts for the failure of DP to be transmitted equally in time across all limbs segments. Thus it appears the CNS preferentially programs the roll response of the body and then adjusts the pitch response accordingly.

Key words: Balance Corrections, Postural control, Muscle responses, CNS motor programs

Introduction

If balance corrections are differently organised in the roll (medio-lateral) and pitch (anterior-posterior) directions, exploring these differences may provide insights into mechanisms underlying falls. A major influence on balance corrections is the biomechanical response of the body which is different in the roll and pitch planes. For a pure pitch perturbation the trunk moves in pitch only. In contrast, across a range of perturbation directions from pure roll to roll combined with pitch, both pitch and roll motions of the trunk occur (Carpenter et al., 1999; Grüneberg et al., 2005). Thus if body motion is different depending on the roll and pitch content of the stimulus, then it might be expected that the CNS takes this into account when executing balance corrections, possibly by relying more on those muscles which act efficiently in the roll and pitch planes to correct the pitch motion induced by a roll perturbation.

There are two opposing viewpoints on the directional control of balance corrections. One viewpoint asserts that no differences exist between the roll and pitch commands issued by the CNS, rather a common movement strategy and muscle synergy is used regardless of perturbation direction (Henry et al., 1998a; Henry et al., 1998b; Park et al., 2004, Jones et al 2008). According to this viewpoint, differences in movement responses or joint torques with perturbation direction can be explained by a simple directional re-weighting of the muscle responses along the body according to the alignment of lines of muscle action with perturbation directions. It was suggested that this re-weighting would take into account the inherent differences in skeletal geometry that lead to different initial responses of the body to the perturbation in the pitch and roll directions. In contrast, the very fact that the timing of trunk velocity is very different in the roll and pitch planes following multi-directional perturbations to stance, led others to believe that there were too many factors to be taken into account for a single directionally re-weighted response synergy to work effectively (Allum et al., 2003; Carpenter et al., 1999; Carpenter et al., 2001). Some of the factors influencing differences in roll and pitch balance correcting strategies are the differences in the arrival

of roll and pitch stimulus-related sensory information used to generate these strategies (Allum et al., 2008), the directional sensitivity of muscle responses (Carpenter et al., 1999) and the need for different knee flexing strategies in the response to roll and pitch tilts (Allum et al., 2008; Oude-Nijhuis et al., 2007).

Thus another viewpoint that has been developed is that the CNS controls roll and pitch joint torques separately. This idea is not new. Winter et al. (1996) suggested separate control of roll and pitch torques during quiet stance and others argued that this is the case for balance corrections (Allum et al., 2008; Carpenter et al., 2001; Matjacic et al., 2001). Matjacic et al. (2001) argued that control in the medio-lateral and anterior-posterior (AP) directions is decoupled based on the observation that net joint torques in pitch only and the roll only directions were identical to those elicited for combined pitch and roll perturbations of the same magnitude. It could however be argued that this does not implicate different control in the two planes and may provide support for the viewpoint that a common torque strategy is utilized regardless of perturbation direction (Henry et al., 1998a,b). Recent studies in the cat, however, also support the concept of separate roll and pitch muscle synergies. Ting and co-workers examined muscles activity in response to several directions of support-surface translation and came to the conclusion that, despite the complex number of muscle patterns involved, these could be resolved into 4 patterns – two for the lateral directions (left and right) and two for AP directions (backwards-forwards) (Ting et al., 2004; Torres-Oviedo et al., 2006). With 3 different synergies required (those for lateral perturbations would be similar for the left and right directions, just opposite in polarity), 2 aligned in opposite directions in the pitch plane (equivalent to differences in the toe-up and toe-down synergies in humans (Allum et al., 2003, 2008), it can be expected that the resultant balance correcting joint torques would have different patterns in the roll and pitch planes too. This would lead to different movement strategies for pitch and roll as concluded on the basis of studies on humans (Carpenter et al., 2001; Grüneberg et al., 2005; Matjacic et al., 2001; Winter et al., 1996).

One way to explore the differences in CNS action for the roll and pitch planes is to delay either the pitch or roll component of the stimulus and compare the response to that with no delay. If the roll and pitch correcting commands are organised separately, a delay in one command should not affect the other. Grüneberg et al. (2005) used only delayed roll tilt stimuli with respect to pitch in order to focus on the different CNS response organization for these two planes. One of the roll stimulus delay times chosen, that with 150 ms delay, was designed to shift the earlier roll trunk motion to the time when trunk pitch motion normally occurs if there is no delay between roll and pitch components of the stimulus. In this way, both roll and pitch commands were forced to act at the same time. This approach worked well in that Grüneberg et al. (2005) were able to show that shifting the roll stimulus merely shifted but did not alter the roll dependent amplitude characteristics of trunk motion or trunk muscle responses. Their results supported the idea that pitch motion is mainly controlled by the ankle muscles and roll motion by the hip and trunk muscles (Carpenter et al., 2001; Matjacic et al., 2001; Winter et al., 1996), but left a number of important issues unexplored. Most importantly they did not explore the effect of delaying the pitch component of the tilts in different directions. The lack of an interaction with pitch movements for delayed roll stimuli might not be true for delayed pitch stimuli. Secondly, Grüneberg et al. (2005) did not explore the effect of the delays on the primary controlled variable, Centre of Mass (COM) movement. Thirdly, they did not explore knee and arm (shoulder joint) motion. At these joints an interaction between roll and pitch corrections could be expected (Allum et al., 2008, Bakker et al., 2006) in addition to any at the trunk. A study of arm and knee joint motion as well as trunk angular motion would seem crucial as these variables show high correlations to COM motion when instability is present (Küng et al., 2009). To explore these issues experiments with both delayed roll and delayed pitch components to tilt stimuli are required with measurements of shoulder and knee joint motion and muscle activity.

Thus the aim of this study was to provide supporting evidence for separate neural control of roll and pitch body motion during balance corrections. For this purpose we investigated the balance corrections following support-surface tilts with delayed roll and delayed pitch stimulus components. One hypothesis we explored was that the biomechanical reactions of the human body in the roll and pitch planes are decoupled from one another and for this reason the CNS controls motion in these planes independently (Grüneberg et al., 2005). We assumed that this could be revealed using delays in the roll and pitch components of tilt stimuli. An alternative hypothesis would be that one command is simply re-weighted by the CNS dependent on direction of body motion (Henry et al., 1998a, b, Jones et al., 2008). Neither hypothesis fit our results, because of the interactions between pitch and roll responses in trunk motion, as well as knee and arm responses, following tilt stimuli with roll components.

Materials and Methods

Subjects

16 young healthy subjects without neurologic or orthopaedic deficits were selected (mean age 27 ± 1 (SEM) years; height 175 ± 2.1 (SEM) cm; and weight 69 ± 1.8 (SEM) kg). All subjects gave witnessed informed, written consent to participate in the experiments according to the Declaration of Helsinki. The Institutional Ethical Review Board of Basel University Hospital approved the study.

Protocol

The subjects' feet were lightly strapped across the insteps with backward foot movement blocked by heel guides fixed to the upper surface of a movable platform capable of rotating in the pitch and roll directions. The heel guides were adjusted to ensure that the ankle joint axes were aligned with the pitch axis of the platform. The foot straps prevented stepping reactions when stimuli causing stance perturbations occurred. The roll axis had the same height as the pitch axis and passed between the feet. The stance width was standardized (14 cm) and two handrails of adjustable height were located 40 cm from the sides of the platform centre. Subjects were informed that they were allowed to grasp the handrails if they needed support. One assistant was present to lend support in case of a fall, but no falls, or near falls (defined as a need to grasp the handrail or receive assistance) occurred.

Stimuli consisted of rotations of the platform in 8 different directions with a constant velocity of 60 deg/s and a constant amplitude of 7.5 deg. Pitch and roll rotations of the platform were combined to reach the following resulting tilt directions defined in laboratory coordinates (see schema in Fig. 3): forward right (23 deg, 68 deg), backward right (113 deg, 158 deg), backward left (203 deg, 248 deg), and forward left (293 deg, 338 deg). We chose those stimulus directions for two reasons. Firstly each direction would have a pitch or roll component that could be delayed. Pure roll or pure pitch directions would not have both components. Secondly, to have comparable directions to those used by Grüneberg et al. (2005). For all stimulus directions either the roll or the pitch component of the stimulus could be delayed by 150 ms (delayed pitch, DP or delayed roll, DR) or both components could occur simultaneously with no delay (ND). Each perturbation was presented in random order eight times to the subject. To minimize fatigue, participants were given a 3 - 4 minute seated rest after the 36^{th}, 73^{th}, 108^{th} and 144^{th} trial. Each trial was preceded by a random 5 – 15 secs interstimulus delay which was initiated automatically. During this time period visual feedback of the subjects' own anterior-posterior (AP) and medio-lateral ankle torque was presented to the subject on a cross with light emitting diodes. This visual feedback was used to standardize prestimulus subject position across trials. Subjects were required to maintain AP ankle torque within a range of ± 5 Nm and medio-lateral torque within ± 10 Nm of their preferred stance reference values. In response to each perturbation, subjects were instructed to recover their balance as quickly as possible. The visual feedback was switched off at stimulus onset for 5 secs.

Data collection

Recordings of biomechanical and EMG data commenced 100 ms prior to perturbation onset and were collected for 1 s. To record EMG activity, pairs of silver-silver chloride electrodes were placed approximately 3 cm apart along the muscle bellies of left tibialis anterior, left soleus, left peroneus longus, left rectus femoris, left biceps femoris, left gluteus medius, left medial deltoid (pars acromialis) and bilaterally on paraspinals at the L1 - L2 level of the spine. EMG recordings were analog band-pass filtered between 60 and 600 Hz, full-wave rectified, and low-pass filtered at 100 Hz prior to sampling at 1 kHz.

Full body kinematics were collected using a three-dimensional optical tracking system with 21 infrared emitting diodes (IREDs) (Optotrak, Northern Digital). The Optotrak cameras sampled the IRED signals at 64 Hz and were placed approximately 4 meters in front of the subject. IREDs were placed bilaterally on the following anatomical landmarks: frontally at the lateral malleolus; center of the patella; frontally at the greater trochanter; anterior superior iliac spine; radial styloid process; elbow axis; acromion; chin; angulus sterni; and on a headband placed just above the ears. Three IREDs were placed at the front corners and the left side of the platform to define the pitch and roll movements of the platform. Subjects wore tight fitting shorts and vests to prevent marker movements.

Support surface reaction forces of both feet were measured from strain gauges embedded within the rotating support surface. The strain gauges were located under the corners of the plate supporting each foot. From forces recorded perpendicular to the support-surface by the strain gauges under the left foot and the distances to the centre of ankle joint rotation, the AP and lateral ankle torques were calculated for the left foot. Because a difference in strain gauge measures was used for torque calculations, an influence of the platform mass on the torque measures was negligible. A similar system measured forces and torques applied by the right foot. The torques from the left and right foot were added together and displayed to the subject as described above.

Data analysis

Primary variables of interest were centre of mass (COM) displacement and velocity, trunk angular velocity, shoulder and knee joint angular velocity profiles as well as muscle responses of the legs, arms and the trunk.

Following analogue to digital data conversion, biomechanical and EMG signals were averaged offline across each perturbation direction. Zero latency was defined as the onset of platform rotation. Subject averages were pooled to produce population averages for a single direction. The first trial was excluded from data analysis to reduce habituation effects entering the data (Keshner et al., 1987).

Kinematic analysis

Marker position data from the Optotrak system were digitally filtered at 16 Hz using a zero phase shift 4th order butterworth filter. Total body COM displacement and velocity were calculated separately for the AP, lateral and vertical directions using a 12

body segment adaptation (Visser et al., 2008) of a 14 segment model (Winter et al., 2003). Two trunk segments (upper and lower trunk) were used instead of four. In addition we calculated the following angular displacements: absolute upper trunk angle (roll and pitch), pelvis, and head angle, ankle and knee joint angles. Absolute rotation angles of the planes defined by pelvis trunk, and head body segments and the platform surface were calculated using 3 or 4 markers to define a plane on these segments. The rotation of this plane was calculated yielding an estimate of the segment rotation. Knee and ankle joint angles were calculated using the angle between the body segments either side of the joint. Arm abduction and rotation were calculated as the angle between the upper arm and upper trunk segments (for further details see Bakker et al., 2006, Visser et al., 2008). Stimulus induced changes were calculated with respect to a pretrigger time interval of 90 ms ending 10 ms prior stimulus onset. We concentrated our analysis of body segment motion to that of the upper trunk, the arm angles with respect to the trunk and knee flexion as these motions had been shown to have the strongest correlation to COM linear velocity following tilt of the support-surface (Küng et al., 2008). Peak velocity amplitudes and times for these variables were measured in both population and individual average traces.

EMG analysis

Each EMG response was corrected for background activity by subtracting the average level of prestimulus activity measured over a 90 ms period ending 10 ms prior to perturbation onset. Then techniques similar to those previously employed (Grin et al., 2007; Grüneberg et al., 2005) to determine response areas of balance correcting responses were used for analysis. Basically response areas were defined over intervals from the onset of balance correcting muscle activity until 150 ms later. We considered only the first 150 ms because due to the delay interval of 150 ms of the DP or DR stimuli, earlier short and medium latency activity in the delayed stimulus responses would also have contributed to measured ongoing balance correcting activity. The onset of the balance correcting responses was defined from the population response for the muscle based on the direction showing the greatest peak activity. From the time of peak activity, the analysis algorithm looked backwards in time to locate the moment when the activity was last below the threshold given by the sum of the mean (set to zero after correction for background activity) plus 2.5 times the standard deviations of the prestimulus activity. Starting at this onset, areas were calculated over an interval of 150 ms for each individual response. As seen in figure 7 this interval contains the primary burst of balance correcting activity.

Torque analysis

AP torque was calculated over an interval from 140 to 290 ms post-stimulus onset, when the greatest changes are observed (Carpenter et al., 1999). Torque changes were calculated for left and right feet separately and summed to yield for total AP ankle torque.

Statistics

Our primary analysis concentrated on between-conditions comparisons of no delay (ND), pitch delayed (DP) and roll delayed (DR) responses using a repeated measures ANOVA model (condition × direction). Significant main effects ($p < 0.05$) were further explored using one-way ANOVA and post hoc t-test comparisons with a Bonferroni correction to account for the effect of comparing three conditions at once.

Results

An examination of COM velocity in figure 1 suggests that the stimulus delays of the platform motion were replicated in all body links with an equal delay for roll, but not for pitch. As we will show, the shift in the roll responses could be observed in balance corrections of all recorded body segments, but not in pitch responses. In this respect it is possible to describe the roll responses as decoupled from those of pitch. To highlight the directional differences of roll and pitch responses, in support of separate neural controls for these directions of motion our description of the results has been divided into four sections. First, we present a global picture by considering COM linear and trunk angular motion with respect to the three delay conditions. Second, results for knee and arm joint motions are described in order to reveal whether differences in roll and pitch coupling of motion occurring at these joints match those at the trunk. We concentrated on trunk angular motion as well as knee and arm joint motion because we had identified in previous studies that these motions had the greatest effect on COM motion (Küng et al., 2009; Oude Nijhuis et al., 2007). Third, we examined the roll and pitch components of ankle torque across directions and stimulus delay conditions in order to determine if coupling between roll and pitch responses was present in ankle torque. Finally, we analysed muscle activity at various joints with the aim of correlating this activity to segment motion in the roll and pitch directions and thereby establish a neural correlate as evidence for separate controllers in biomechanical responses.

Biomechanical responses of the COM: comparisons with Trunk, Knee and Arm motion

COM Motion – Timing and vector directions

Figure 1 shows examples of COM and trunk motion for 2 stimulus directions. One direction is 158° (lower graphs of Fig. 1A-D), a pitch tilt almost purely backwards (toe-up) and the other direction is 113°

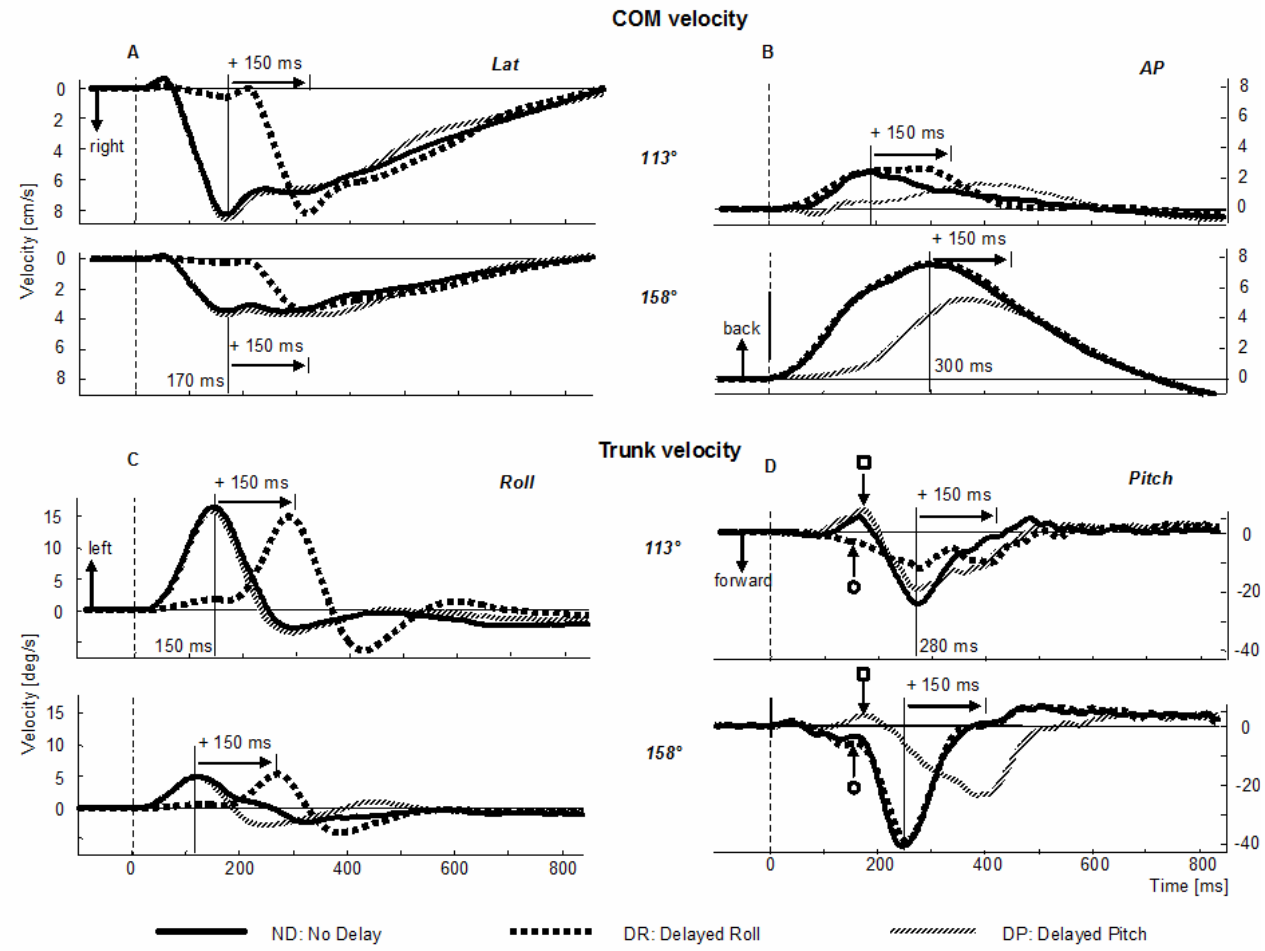

Figure 1: Average velocity plots for COM velocity (A and B), and trunk velocity (C and D). Lateral and roll plots on the left, anterior-posterior and pitch plots on the right. Plots for two directions of platform tilt are shown 113° (right and slightly backwards) and 158° (toe up and slightly right). Each plot for each of the 3 delay conditions is the average of 8 responses from 15 subjects (120 responses). Stimulus onset is marked by a dotted vertical line. The times of peak velocity of each curve for the no-delay (ND) condition is marked by a full vertical line.

(upper graphs of Fig. 1A-D), a roll tilt almost purely right. Directions are illustrated by centre schema in figure 3. In figure 1 the lateral movement of the COM and the roll motion of the trunk were clearly shifted 150 ms with the delayed roll stimulus (DR), for both stimulus directions (Fig. 1A and C). For the two directions shown (and for all other directions) the peak in lateral COM was at 170 ms for no delay (ND) stimuli. This compared to a later peak in AP COM at 300 ms for directions 158° (Fig. 1B) and 203° for ND stimuli. Thus the rationale for the 150 ms delay time, forcing roll and pitch trunk peak velocity to occur simultaneously was achieved. For other directions the peak in AP COM was at different times, compared to the time for near pitch (158°/203°) stimuli and was differently shifted with direction under the delay condition. For example, as shown in figure 1B, for the 113° direction of tilt, the peak in AP COM for DR was shifted almost 150 ms (rather than having no shift as seen for 158° in Fig. 1B) compared to no delay stimuli. Differences in timing shifts across directions observed in figure 1 for AP COM velocities are quantified in figure 2. The time of the AP COM velocity was shifted for DP with respect to ND. For the more pitch directed tilts (23°/338°, 158°/203°) no shift occurred in AP COM velocity peak for DR stimuli as expected (Fig. 2A). However, peaks in AP COM velocity were shifted for DR with respect to ND stimuli for the more laterally directed backward tilts (113°, 248°). These changes in AP COM motion with roll stimulus delay indicate an interaction between roll and pitch responses dependent on stimulus tilt direction.

Despite these changes in timing there were few changes in early vector directions of COM velocities. The polar plots of figure 3A/B indicate the direction of the COM motion at the two time points, 170 and 300 ms, when these COM velocities have peaks in lateral and AP directions for the ND stimuli in stimulus directions 113° and 158°, respectively. If the COM motion is independently controlled in roll and pitch, then firstly, delay of the roll component of the stimulus (DR) by 150 ms should cause the COM motion to be pitch oriented at 170 ms (seen in Fig. 3A). Secondly, if the pitch component is delayed (DP) 150 ms then motion should be laterally oriented at 170 ms (Fig. 3A). However at 300 ms, when a shift in the vector orientation of COM might have been expected with delayed stimulus components, based on the earlier changes in COM velocity at 170 ms with

stimulus delay, only slight differences in the vector orientation of COM velocity between DP, ND and DR stimuli were observed (Fig. 3B). As this result indicates a compensation for earlier changes in COM velocity we examined whether AP COM position at 800 ms was altered with stimuli delay. No change was found [P = 0.940]. However for the DR stimuli, lateral COM position deviated downhill marginally less than for ND stimuli [P = 0.042]. These results suggest that delaying roll and pitch components of the stimuli had no overall effect on control of the COM velocity after 300 ms despite the presence of interaction effects between roll and pitch prior to 300 ms.

Trunk Motion – Timing and Vector directions

Interactions between pitch and roll responses emerged before and after 300 ms for angular motion of the upper trunk, the knee and shoulder joints compared to the linear motion of the COM. At approximately 150 ms, when trunk roll velocity peaked for ND stimuli, DP stimuli revealed that roll component of the stimulus caused backward directed pitch motion of the trunk (marked □ in Fig. 1D). This motion was smaller for the backwards DP perturbations, 158° and 203° with small roll components (Fig. 1D). The pitch responses revealed with DR stimuli also caused trunk motion with a pitch component at 150 ms (marked O in Fig. 1D), which was, however opposite in direction to that revealed by DP stimuli (see figure 1D see traces marked O and □). Moreover, the pitch trunk velocity for roll directed DR stimuli (for example 113° DR traces seen in Fig. 1D), appeared to have two peak values, one due to the pitch component of the stimulus at 280 ms (revealed by DR), the other 150 ms later due to pitch induced by the DR stimulus. This interaction in pitch responses was not seen for tilts in the two backward pitch directions (Fig. 1D, 158°).

In contrast to COM, the vector directions of trunk velocities at 280 ms for DP and DR stimuli were not aligned with those of the ND stimuli except for the two backward pitch directions (158° and 203°). For other tilt directions, vector directions of trunk motion were clearly different for DR and DP stimulus at this time point (Fig. 3D). The earlier timing of the peak in trunk pitch under ND conditions, the shift of the peak in comparison to ND for DR conditions and the lack of a 150 ms shift for delayed pitch (DP) stimuli were common characteristics of the more roll oriented stimuli (68°/293°, 113°/248°, see Fig. 2B). In comparison, the trunk pitch response for more pitch directed stimuli (23°/338°, 158°/203°) was clearly shifted for DP and not changed for DR conditions (Fig. 2B). The differences in vector directions for COM at 300 ms and trunk at 280 ms suggests that roll components of the stimuli induce pitch motion of the trunk which is not mirrored in COM motion due to compensation at other body segments for example, the arms, so that by 300 ms for COM velocity (and 800 ms for COM position) no major differences can be observed in COM motion. The question also arises whether this trunk pitch motion is induced directly on the trunk by the tilt perturbations or is induced on the trunk by earlier movements at other body segments, for example the knees. In the later case, this action would provide evidence of the CNS planning compensatory pitch responses with a roll command.

Amplitudes of COM and trunk angular velocities

Amplitudes of lateral COM velocity and trunk roll velocity were preserved across delay conditions. It

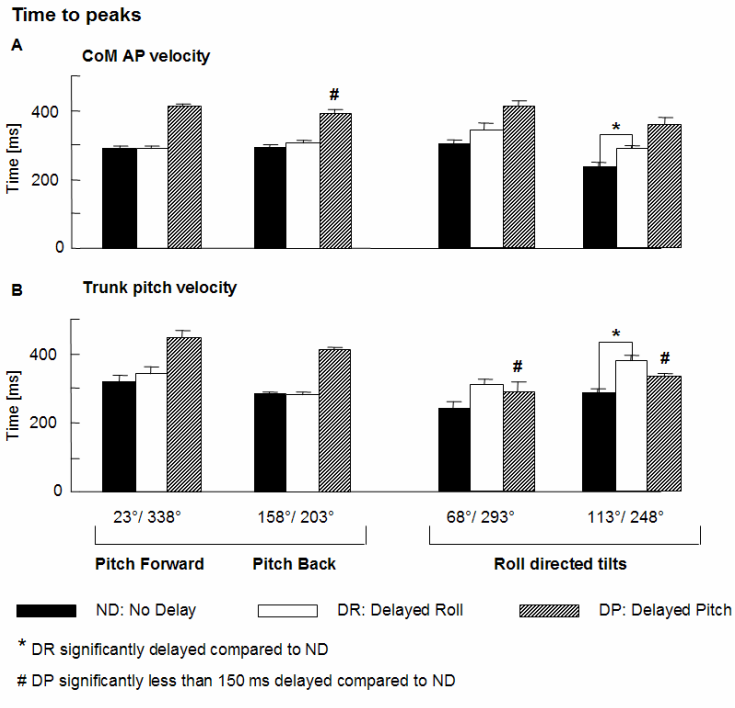

Figure 2: Times of peak COM AP velocity and trunk pitch velocity across directions for the 3 delay conditions. The height of each column represents the mean population value based on each subject's mean response (average of 8 responses) per direction and the vertical bars standard errors of the mean (sem). Responses for directions with the same pitch stimulus component but oppositely directed roll (eg 23° and 338°) have been pooled. The vertical bars on the columns represent the standard error of the mean.

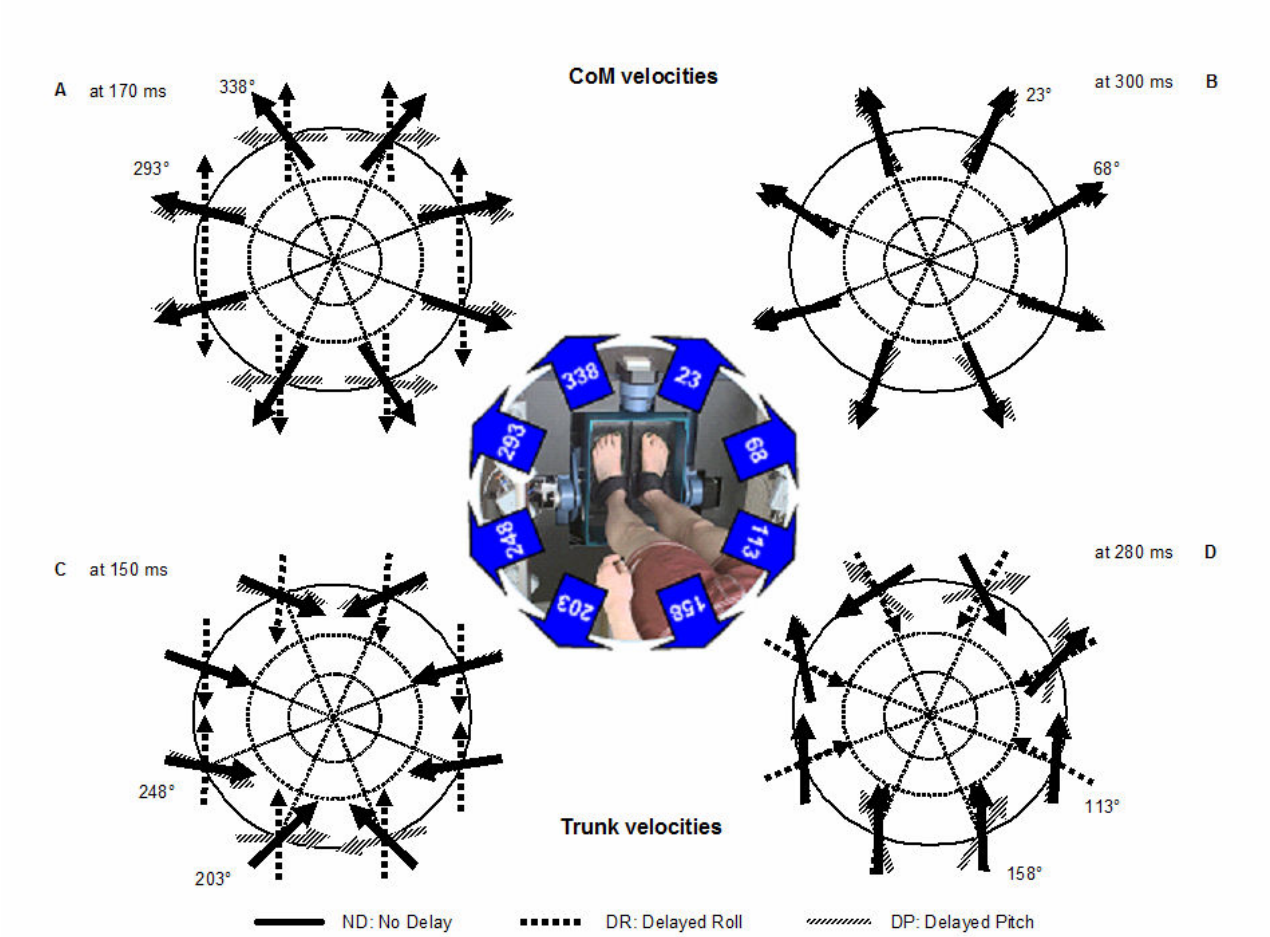

Figure 3: Vector directions of COM velocities at 170 (A) and 300 ms (B), and of trunk velocities at 150 (C) and 280 ms (D) when these velocities peak (see vertical lines in figure 1). For each delay condition the vector direction of velocity computed from anterior-posterior or pitch and lateral or roll velocities for the COM or trunk, respectively, is shown as a polar plot. The directions of the spokes in the polar plot correspond to the directions of tilt indicated in the middle of the figure.

made little difference if the amplitude was examined at the time of the peaks for each subject for each stimulus direction or if the amplitude at the times of the peak in the population average traces was taken (170 and 320ms for lateral COM velocity and 150 and 300 ms for trunk roll velocity for DP and DR stimuli, respectively). There were no differences in the peak amplitudes across stimulus direction [COM: $F(2,84) = 0.067$; $P > 0.5$; Trunk: $F(2,87) = 0.021$; $P > 0,5$].

In contrast, delay conditions caused small but significant changes in the amplitudes of AP COM. A consistently reduced AP COM velocity with DP stimuli occurred for forwards (23°/338°) and backwards (158°/203°) directed stimuli (Fig. 4).

In summary, these results indicate that when the pitch component of the tilt stimulus is delayed, then the timing of the peak velocities of AP COM and trunk pitch are not shifted an amount equal to the delay and amplitudes are not preserved. This effect occurs preferentially for roll directed tilts, that is, those with a larger roll component than pitch. These effects contrast with a lack of effects of stimulus delay on lateral COM and trunk roll responses other than a shift of 150 ms for DR stimuli. This provides evidence that the roll component of balance correcting responses can be programmed by the CNS independently of the pitch response but not vice versa. The question arises whether the lack of an effect of the delays on COM motion after 300 ms, despite the clear effect on trunk motion after 300 ms is due to a compensatory action of the CNS for trunk motion using knee and arm responses.

Knee Angular Velocities

In comparison to the trunk, flexion of the knees has the second-most significant influence on lateral and AP motion of the COM (Küng et al., 2009). The uphill knee flexes and the downhill knee extends (up to approximately 2-3 deg to maximum extension) for roll tilts of the support surface (Bakker et al., 2006, Allum et al 2008). Insufficient flexion and extension leads to an unstable COM position and loss of balance (Küng et al., 2009). Appropriate correction for a roll and/or forward tilt is based on the difference in knee movements. Greater flexion of the uphill knee and extension of the downhill knee provides a greater lateral shift of the COM uphill due to a greater difference between the knee movements. On the other hand, knee flexion will also influence AP COM motion (Oude Nijhuis et al., 2007). The greater the

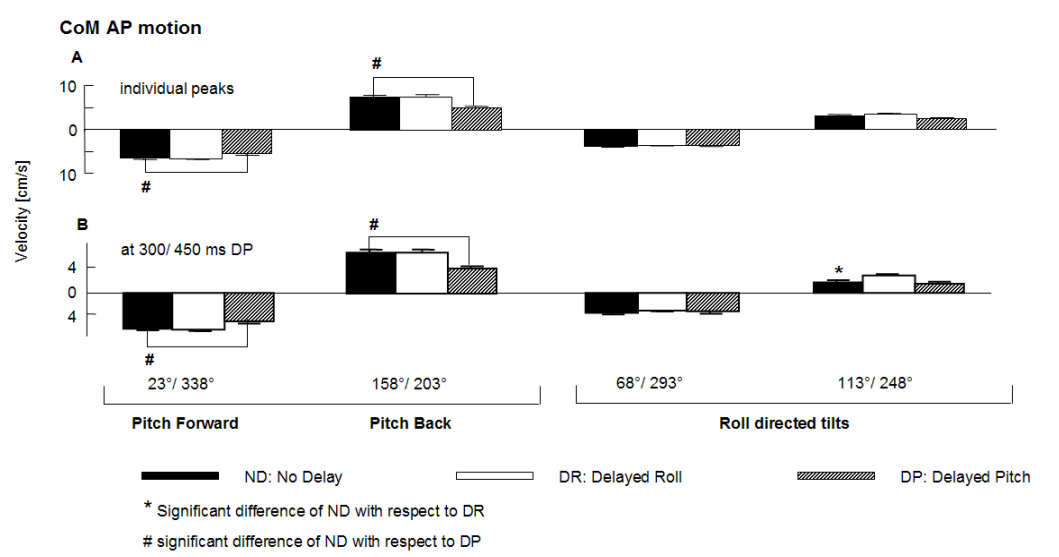

Figure 4: Mean amplitudes of COM AP velocity measured from subjects individual mean response peaks (A). Measurements at 300 ms (for ND and DK stimuli) and 150 ms later for DP stimuli, times when population mean has a maximum value (B). The layout of the figure is identical to that of figure 2.

sum of the two knee flexion movements (zero if one knee flexes an equal amount to the extension of the other) the
greater effect on AP COM motion. Thus we were interested in learning how delaying the roll and pitch components of the stimulus influences knee movements. For this purpose we examined the difference and sum of knee flexion movements.

Knee flexion movements occurred primarily for forward and/or roll tilts. Knee flexion (both differential and summed) was small for backwards tilts (158° and 203°). There were two phases in the difference in knee flexion-extension velocities with the first relative knee extension having a peak at approximately 130 ms (preceding the peak in trunk roll velocity, compare fig. 5A & Fig. 1C). This was followed by relative uphill knee flexion which peaks at approximately 250 ms (see table 1) as the trunk roll velocity uphill reduced to near zero (compare with trunk roll velocity traces in Fig. 1C). Over perturbation directions, the roll velocity directed profiles of knee flexion appeared to be decoupled from a dependence on the pitch effect of knee flexion because profile timing was equal for the ND and DP conditions and shifted 150 ms for the DR condition (see Fig. 5A & Table 1). No changes in the amplitude of differential knee flexion velocity occurred across delay conditions [$F(2,81) = 0.224$, $P > 0.05$].

The sum of the left and right knee velocities divided by 2 is shown for right and forward tilts of the platform in figure 5B. If knee movements were used to predominantly control the pitch rather than the roll displacement of body then across directions all traces of the sum of left and right knee velocities should be similar to those for the 23 deg directed perturbation. For this direction the response to DR stimuli has the same profile as ND stimuli and the response to DP stimuli is delayed 150 ms with respect to ND stimuli. For the perturbations with a greater roll than pitch component (see traces for 68° and 113° in Fig. 5B), the profile of the sum of the left and right knee velocity was different. The response to DP was shifted earlier the response to DR was shifted later (Fig. 5B & Table 1). These changes were consistent with changes in the amount of trunk pitch velocity present under ND and DR conditions for near roll perturbations.

Arm Angular Velocities

Arm movements also have a strong influence on COM movements (Küng et al., 2009). For lateral tilts, both arms moved laterally downhill but the amount of abduction and adduction in each arm varies (Küng et al., 2009), hence we considered the difference in arm abduction velocities (see Fig. 6). For roll and backward pitch tilts both arms rotate forward with most rotation occurring, for backwards tilts (Küng et al., 2009), hence we considered the sum of arm rotation velocities.

Arm abduction velocities, considered as the difference between the left and right arms were shifted exactly 150 ms for DR stimuli across all perturbation directions, with no shifts for DP stimuli (Fig. 6 left). The timing of the peaks in arm abduction velocities was identical to those of trunk velocities, occurring at approximately 150 ms, and prior to the peak in the difference in knee flexion velocities (compare trunk, arm and knee difference traces in Fig. 1C, 5A & 6A, Table 1).

For arm rotation velocities, a significant difference equal to 150 ms in the time of peak velocity was observed in the backward pitch directions (158°/203°) between ND and DP stimuli, with no difference for ND and DR stimuli (Fig. 6B). For near roll stimuli (68°/293° and 113°/248°), the forward arm rotation did not follow this pattern but instead matched the pattern seen for AP directed knee motion (compare responses to 68° and 113° tilts in Fig. 6). The time of peak arm rotation (rotation as seen in the transverse plane) velocity shifted earlier in time as the perturbation direction was directed more forwards for

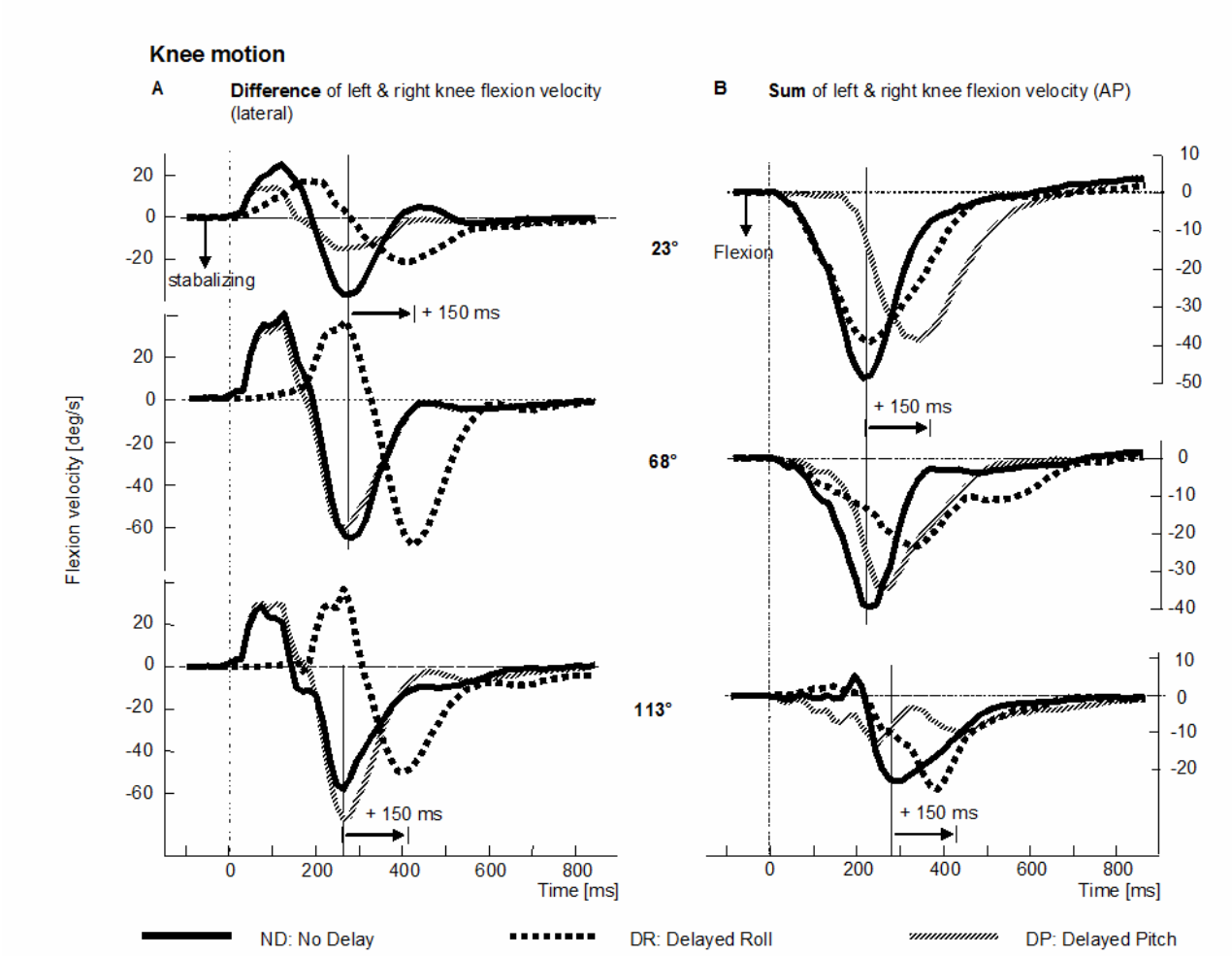

Figure 5: Mean population traces of the difference in right and left knee flexion movements (A) leading to a lateral stabilisation of the body and sum of traces of the right and left knee flexion (B) leading to trunk pitch. Responses for the 3 delay conditions and 3 directions of right tilt are shown. Knee responses for the direction 158° are small. The layout of the figure is identical to figure 1 with peak responses marked by a full vertical line.

Table 1. Times to peak responses in knee and arm velocities

Knees: Difference of left & right: mean (± SE)			
	23° / 338°	68° / 293°	113° / 248°
ND	277.55 (± 11.09)	270.51 (± 12.41)	256.80 (± 18.85)
DR	322.00 (± 19.28)	416.25 (± 13.78)	394.68 (± 16.30)
DP	316.56 (± 22.21)	257.51 (± 17.44)	251.19 (± 19.21)
Knees: Sum of left & right: mean (± SE)			
	23° / 338°	68° / 293°	113° / 248°
ND	253.13 (± 26.76)	231.08 (± 12.27)	266.49 (± 19.92)
DR	256.62 (± 16.66)	318.73 (± 24.74)	361.11 (± 17.14)
DP	343.11 (± 25.11)	278.68 (± 19.11)	236.30 (± 17.98)
Arms: Sum of left & right: mean (± SE)			
	68° / 293°	113° / 248°	158° / 203°
ND	326.86 (± 15.27)	295.43 (± 10.91)	293.81 (± 6.82)
DR	452.01 (± 14.48)	399.46 (± 19.57)	323.07 (± 24.90)
DP	320.36 (± 16.82)	342.03 (± 14.07)	435.76 (± 17.27)
Arms: Difference of left & right: mean (± SE)			
	68° / 293°	113° / 248°	158° / 203°
ND	145.74 (± 4.63)	138.78 (± 2.43)	163.74 (± 13.93)
DR	293.19 (± 1.86)	292.61 (± 2.10)	339.05 (± 29.71)
DP	140.51 (± 1.77)	143.42 (± 4.25)	153.87 (± 17.54)

DP stimuli with no significant difference of the timing being observed for all predominantly roll directed stimuli (68°/293°, 113°/248° – see Table 1). For DR stimuli this peak shifted progressively later in time as perturbation direction was directed more forwards with the difference between DR and ND changing from almost equal for backward stimuli (158° and 203°) to a delay of 150 ms for forward and roll stimuli (68° and 293°). In summary, as table 1 indicates the peak times of arm rotation responses were after those of knee "pitch-inducing" flexion responses, possibly indicating that the arm responses were a compensation for the effects of knee flexion on AP COM velocity.

Ankle Torques

Despite the shifts in knee velocity profiles (Fig. 6), both roll and pitch ankle torques (summed for the left and right foot) were delayed consistent with these torques being decoupled from one another. Lateral torque magnitudes at the ankle were of the order of 1/20 of the AP torques. Summed AP torques from both ankles were shifted 150 ms by DP stimuli but not at all by DR stimuli. Consider, for example, the AP ankle torque profiles for near roll stimuli (68°/293°, 113°/248°). The uphill and downhill ankle torques were changed by the DR and DP stimuli in one leg, however the torque responses of the contralateral legs

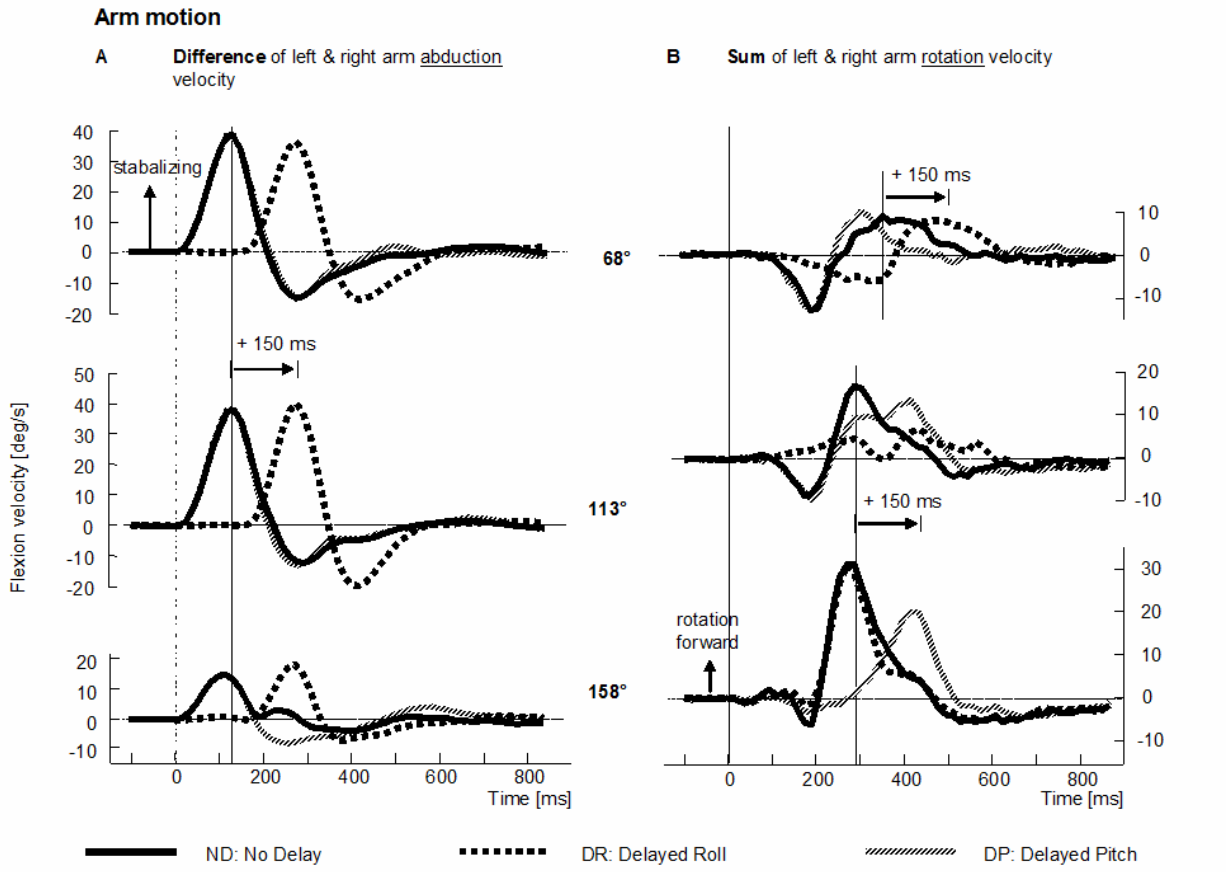

Figure 6: Mean population traces of the difference in right and left arm abduction movements (A), which precede a peak in trunk roll and traces of the sum of right and left forward rotation movements (B) which follow the sum of knee flexion movements. Traces are shown for 3 delay conditions and 3 directions of right tilt. Arm responses for the direction 23° are small. The layout of the figure is similar to that of figures 1 and 5.

had completely opposite polarities. Thus despite these changes at each leg, the effect of the DR stimulus on combined ankle torques was minor even when ankle torque changed rapidly over the balance correcting interval we analysed (140 – 290 ms). Across all directions combined ankle torque was pitch oriented for ND stimuli and remained so for DR stimuli. The DP stimuli shifted ankle torque profiles 150 ms with unchanged amplitudes .

EMG Responses

All muscles we examined had responses with varying sensitivities to the pitch and roll components of the stimulus. Figure 7 provides three examples of these differences to delay stimulus for the tilt directions 158° (backwards) and 248° (sideways). Tibialis anterior had a pitch sensitivity as its maximal response (158° in Fig. 7) was significantly shifted 150 ms for DP stimuli but not by DR stimuli. Gluteus medius has a roll sensitivity as its maximum response (see traces for 248° in Fig. 7) was shifted by DR but not by DP stimuli. Note however that both these muscles were somewhat differently affected by stimulus delay for directions of tilt not eliciting the maximum response amplitude suggesting, for example, tibialis anterior had a roll sensitivity for the 248° direction and gluteus medius a pitch sensitivity for the 158° direction, albeit weak. Responses of an intermediate muscle, rectus femoris with both a pitch and roll sensitivity is illustrated by the middle traces of figure 7.

To characterise the directional sensitivity of muscle responses, figure 8 plots the response amplitudes measured as the area under the curve for the first 150 ms after response onset for different directions of tilt under the 3 delay conditions. Each spoke of these polar plots represents the amplitude for a direction of tilt. It is clear from figure 8 that tibialis anterior shows little difference in response sensitivity for ND and DR stimuli across directions reinforcing its classification as a pitch directed muscle. Likewise, for gluteus medius, there was little difference between ND and DP stimuli justifying its classification as a roll directed muscle, as supported by the direction of its maximum responsiveness for the ND condition (see arrows on polar plots). Nonetheless tibialis anterior had a small roll, and gluteus medius a small pitch sensitivity as indicated by the areas of the polar plots of responses to DP and DR stimuli, respectively, and the arrows indicating the direction of maximum sensitivity under these delay conditions. The polar plot of rectus femoris in figure 8 provides an intermediate picture. For this muscle the area circumscribed by the

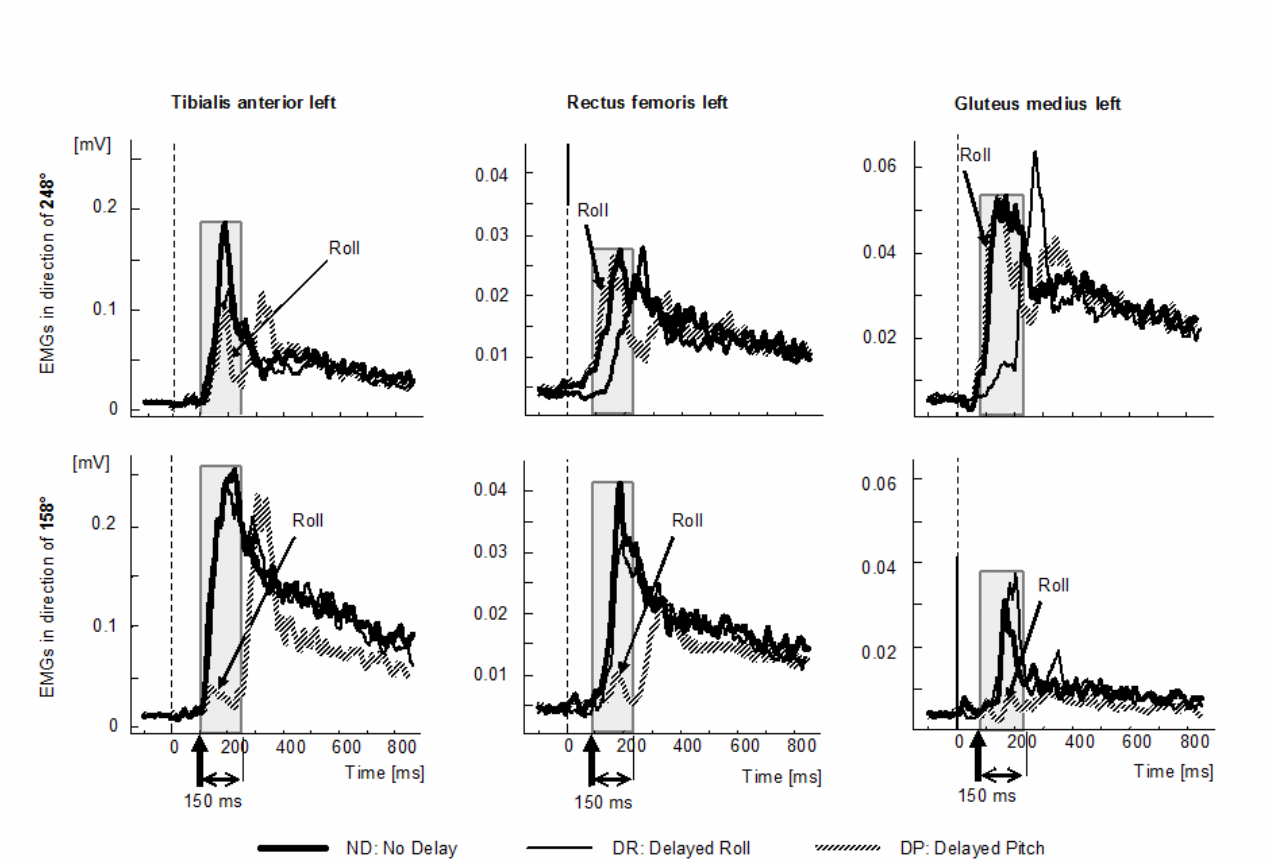

Figure 7: Mean population traces of EMG activity in tibialis anterior (left set of traces), rectus femoris (middle set of traces) and gluteus medius (right set of traces). The upper row of traces is in response to a tilt in the 248° direction, the lower set of traces for a 158° tilt. The onsets of the responses are marked by vertical arrows and the 150 ms averaging interval by a grey box.

polar plot is approximately the same size for DP and DR responses, both of which are less than the area of the plot for ND responses.

The area circumscribed in the polar plots such as those of figure 8 was used to categorize the muscle response types shown in figure 9. Thus, pitch sensitivity was defined by the area circumscribed in the polar plot for DR stimuli compared to the area for ND stimuli and that roll sensitivity by DP stimuli compared to ND stimuli. If pitch sensitivity was significantly greater than the roll sensitivity for a muscle we termed this a pitch sensitivity muscle and vice versa, a roll sensitivity muscle. When the two sensitivities had less than a 15% difference in pitch and roll sensitivity we termed the muscles mixed sensitivity muscles. Figure 9 shows how the various muscles we recorded were grouped. The ankle joint muscles, except for peroneus were classified as pitch muscles. The trunk muscles gluteus medius and paraspinals were classified as roll muscles. In contrast, knee (incl. peroneus) and arm muscles were classified as mixed muscles.

Discussion

The results of this work add further evidence to the concept that balance corrections in the roll and pitch directions are executed separately by neural command centres. If this command control is managed within the same neural centre or within closely connected but different neural centres for pitch and roll can not be determined on the basis of this study. We have been able to extend the work of Grüneberg et al. (2005) for delayed roll (DR) stimuli by demonstrating that a delay in the roll component of the stimulus is transmitted faithfully to occur in knee flexion and arm abduction responses at the later time, regardless of the stimulus direction. In the sense that neither the stimulus direction nor the presence or not of a pitch component to the tilt stimulus influenced roll responses, this observation can be interpreted as a control by the CNS of roll decoupled from that of pitch. The findings of Grüneberg et al. (2005), who used different times of delayed roll stimuli, plus earlier work on the biomechanical responses to combined roll and pitch tilts without delays (Allum et al., 2003,2007; Carpenter et al., 1999), led us to develop the hypothesis that pitch motion would be controlled independently of roll motion too, and that

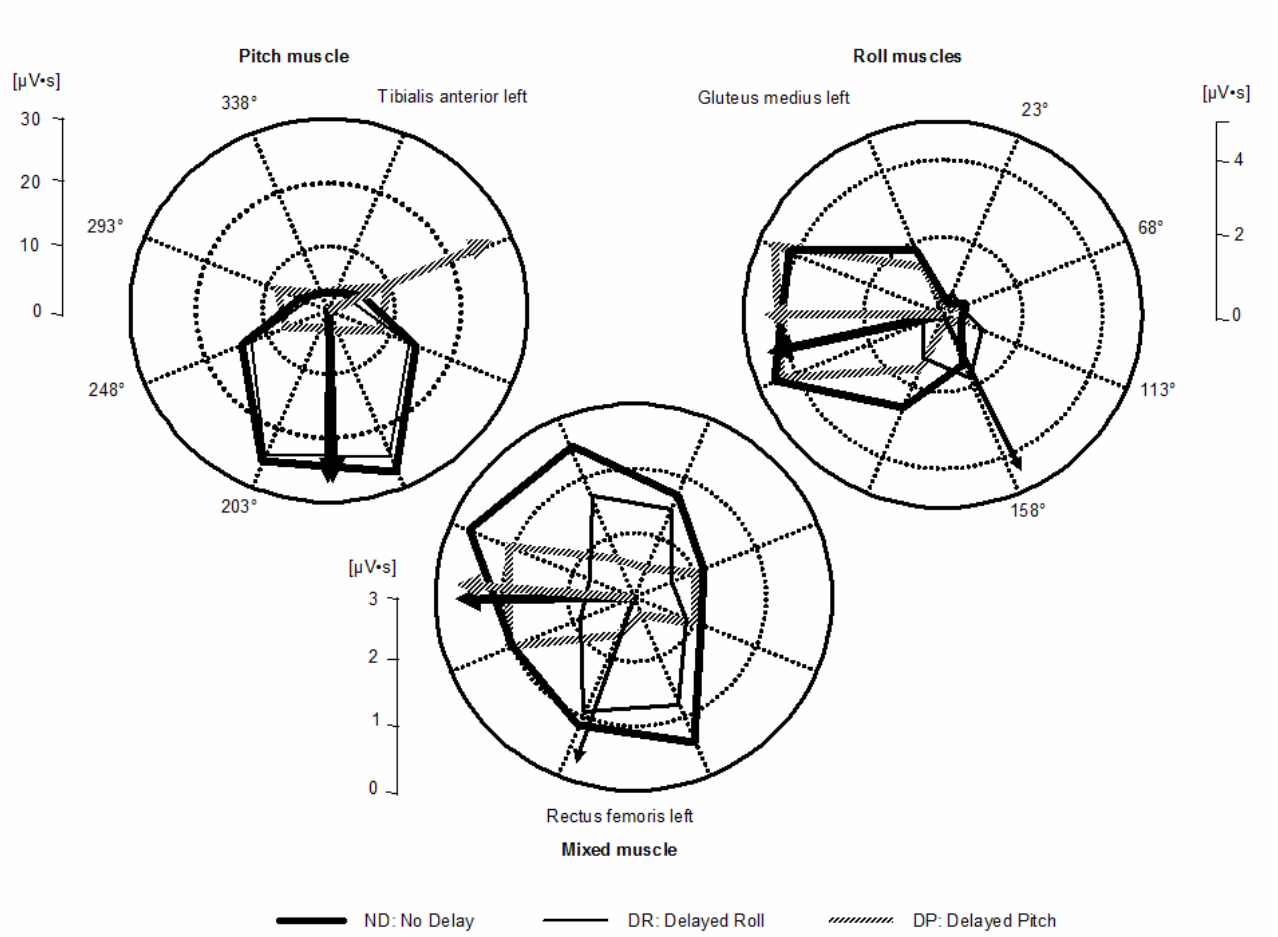

Figure 8: Polar plots of muscle activity over the 150 ms averaging intervals shown in figure 9. The layout of the polar plots is identical to that shown in figure 3. Each spoke represents the direction of tilt and radial distance from the plot centre along the spoke the amplitude of the EMG response area according to the scales next to the polar plots. The amplitudes along the spokes are then joined and the direction of the centroid of the figure defines the direction of maximum response sensitivity as indicated by the arrow. Note the different directions of maximum sensitivity for the 3 stimulus delay conditions for the 3 muscles.

this pitch motion would occur mainly about the ankle and knee joints (Allum et al., 2003; Grüneberg et al., 2005). According to this concept there would be little interaction between the two forms of control. Our current results as well as recent work in vestibular loss subjects (Allum et al., 2008) indicates that pitch control is dependent on roll,with the dependence increasing as the size of the roll component of the stimulus increases. Thus it appears that CNS is not able to program pitch control independently from roll.

When the pitch component of the stimulus was delayed, trunk, knee and arm movements were not transposed faithfully in time by the amount of the delay. Rather the amount of delay in the response profile and its amplitude depended on the stimulus direction (see Fig. 1, 2, 3, 5 and 6). Changes in pitch movements prior to 120 ms in response to roll-directed tilts as shown in right sets of traces figures 1, 5 and 6 are probably due to a purely biomechanical pitch response of the body to roll component of the stimulus. Carpenter et al. (1999) also noted an early pitch response of the trunk following pure roll tilts. An early effect on roll responses in response to the pitch component of the stimulus was not observed in this study and not by Carpenter et al. (1999) for pure pitch tilts. The presence of these early biomechanical changes in pitch for the roll component of the stimulus implies the CNS must take these pitch changes into account when planning the response to the roll perturbation. Most of the later changes in pitch kinematics due to balance corrections could be traced to differences in knee flexion movements between the left and right knees across stimulus directions. The flexion of the uphill knee and exclusion of the downhill knee resisted lateral shift of COM downhill by holding the trunk tilted uphill. If not resisted sufficiently, as in, for example, vestibular loss and spinal cerebellar ataxia (SCA) patients with insufficient flexion of the uphill knee and extension of the downhill knee an unstable lateral motion of the COM resulted (Allum et al., 2008; Küng et al. 2009). Because the differential knee action is insufficient in these patients a reversal of trunk motion downhill occurs. The marked instability in SCA subjects following roll tilts and the inability to program pitch responses to forward tilt adequately (Bakker et al., 2006) would suggest that centres responsible for executing the separate roll and pitch commands,

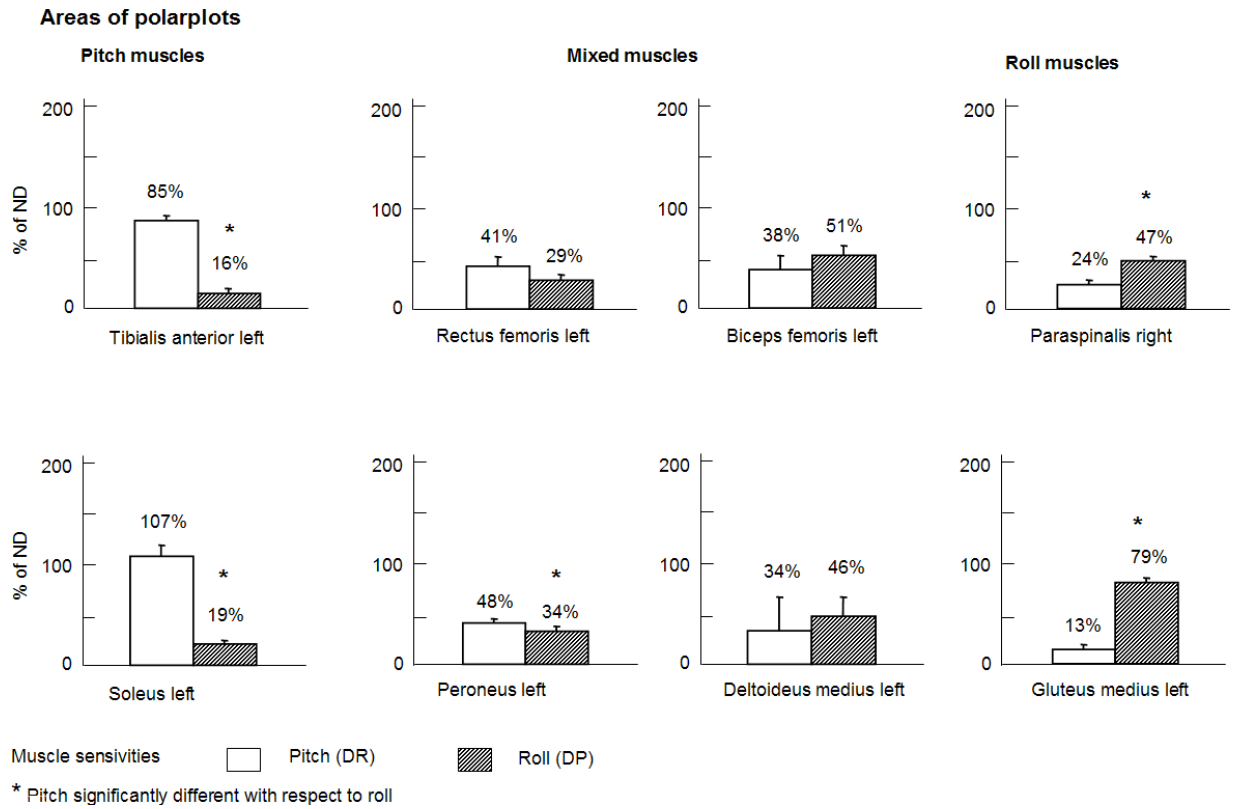

Figure 9: Classification of muscle sensitivity based on response areas in the polar plots under the 2 delay conditions DR and DP compared to the ND condition (ordinate). Each column represents the mean value for the population and the vertical bar the sem.

postulated on the basis of the current results, may lie in the vestibulo cerebellum.

It is interesting to speculate whether our results could be explained as an inability of the CNS to generate roll corrections without the use of knee motion which also induces motion in the pitch plane. It appears that the CNS has other choices in programming rollcorrections. For example, when knees flexion was blocked artificially, subjects successfully corrected for roll perturbations using greater than normal arm movements at expense of greater COM motion (Oude-Nijhuis et al., 2008). Thus differential knee flexion may also help control the amount of COM pitch directed motion.

Our analysis of EMG responses under different stimulus delay conditions indicates, as described before, that ankle muscles predominantly control pitch and trunk muscles roll motion of the body (Grüneberg et al., 2005). Our new findings are that arm and knee muscle responses act on both roll and pitch motion of the body as these are equally sensitive to roll and pitch plane components of the stimulus. We assume this action is the primary manner in which the CNS maintains the controlled variable, presumably COM velocity, at a minimum.

The key contribution of knee movements to controlling roll and pitch motion of the body contrasts with the low sensitivity of early passive knee movements to stimulus direction (Allum et al., 2008). This may have the advantage that the knee muscles help control body motion with efferent signals enhancing later proprioceptive feedback in a feed-forward manner without early proprioceptive knee responses contributing significantly to the sensory signals initiating and modulating balance corrections. A similar function may be exercised by arm muscles in which stretch reflexes relating to tilt stimuli have not been observed (Allum et al., 2002).

It is interesting to speculate why balance control centres coordinate roll balance corrections as if these were totally decoupled from those of pitch but not vice versa. One reason could be simply biomechanical in that roll movement of the trunk occurs earlier than that of pitch following a combined roll and pitch tilt of the support surface (Allum et al., 2002; Carpenter et al., 1999). Furthermore, complete proprioceptive and vestibular information on the stimulus roll characteristics appears to reach the CNS prior to the arrival of pitch directed information (Allum et al. 2008). Thus from timing considerations alone, the CNS may need to carry out the necessary programming and release of the response to roll tilt prior to that for the pitch tilt.

This report presented data on 8 directions of tilt each with a roll and pitch component. For completeness it would have been advisable to have the pure pitch and roll directions as well as 45 deg directions with equal components of pitch and roll for the 3 delay conditions. Data for the pure pitch and roll directions are available in prior publications (Allum et al., 2008; Bakker et al., 2006). Also we have no reason to believe that responses from the directions 45, 135,

225 and 315 in our nomenclature could not be predicted from the current results either side of these directions that is from the current 23, 63, 113, 158, 203, 248, 293 and 338 responses. An expansion of our protocol on the same subjects would probably have been too tiring for them.

These results add further evidence to the differences between the control strategies in centres generating balance correcting responses for bipedal and quadrupedal stance (Allum et al., 2008). Some authors speculated on the similarities based on pitch plane responses (Dunbar et al., 1986, Horak and Macpherson, 1996). Roll responses are fundamentally different between bipedal and quadrupedal stance. Firstly, in humans, the motion of the upper trunk is in the opposite direction to that of the pelvis on roll tilt (Allum et al., 2002,2008). This is a completely different biomechanical response from that of quadrupeds where trunk and pelvis move in the same direction as the roll tilt of the support surface (Macpherson et al., 2007). The movement of the trunk on the pelvis provides a completely different biomechanical situation in biped stance. In quadrupeds the uphill knee must flex in order to shift the body laterally uphill, whereas in bipeds the uphill knee must flex in order to hold the trunk in the uphill position compensating for the lateral shift of the pelvis downhill. The current research indicates that the functional pitch plane effect of the knee action in bipeds is presumably not present in quadrupeds due to stance on four legs. We presume that the presence of a different knee action plus simultaneous forelimb action in quadrupeds leads to a different and possibly reduced pitch motion during roll balance control in quadrupeds. Nonetheless, it would be interesting to explore changes in balance corrections for roll tilt when humans are asked to respond to roll tilt in a quadrupedal position in order to determine if the roll responses can be programmed separately from pitch as indicated here.

In the sagittal plane increasing use of knee movements was seen, dependent on stimulus velocity (Runge et al., 1999). These results provide a conceptional focus different from the notion that the degrees of freedom are reduced in balance control (Bernstein, 1967; Horak and Nashner, 1986). While this concept might hold for backward tilts, in the roll plane the control of the degrees of freedom is quite complex as the knees are controlled independently as are the arms leading to a cross-coupling effect of motion in the pitch plane. The essential question we have tried to address is whether the CNS programs the roll and pitch movement of body independently. The very fact that either the pitch or the roll component of the stimulus could be delayed 150 ms, yet at the completion of the balance correction the overall effect on COM motion was identical to the effect with no delay, would suggest that the CNS programs the roll and pitch motion independently but that both responses interact in a linear manner biomechanically. Interestingly, when individual segments were examined it was very apparent that the underlying segment motions in the roll and pitch planes were not independent of one another. Specifically this is very apparent from knee muscle responses which have almost equal pitch and roll plane sensitivities. The delay of knee flexion movements with respect to trunk roll, and arm movements with respect to the knees, indicates that these movements are not a biomechanical response to the tilt stimulus, rather a compensating balance correction acting to stabilise early trunk motion.

We have assumed here that biomechanical responses prior to 120 ms, interact linearly and that the only effect of the delay was to shift the early biomechanical responses 150 ms. For our 7.5 deg support surface tilts this appears to be a valid approximation, as segment angle changes are maximally 6 deg (Allum et al., 2003). Amplitudes of pitch responses were generally not altered. Roll amplitudes were unchanged with delay conditions. For larger amplitude tilts this may not be a valid approximation. The amplitude of tilt for which the effect of the delay leads to a fundamentally different response needs to be investigated, possibly with modelling techniques. The lack of such an investigation limits the application of our findings to tilt amplitudes greater than those we investigated.

Although there was a clear interaction between the pitch and roll motion of the trunk, knees and arms induced by tilt of the support surface, delaying either the pitch or roll component did not influence the overall COM velocity response of the body after 300 ms. We had not expected that changes we observed with stimulus delays to pitch would lead to interactions at this level and be compensated in COM responses. Again this reinforces our conclusion that the CNS can program balance corrections in the pitch and roll planes independently of one another, even if interactions exist between the two planes at the level of the arms and knees. Interestingly the form interactions took implied that roll control is programmed first and the pitch control must take into account previously occurring effects on pitch due to roll commands. In this sense pitch control is not independent of roll. The question arises within this context as whether the roll first action represents a preferred plane of action. Preferred planes of action for head-neck movements has been suggested as a technique to simplify sensory-motor transformations serving motor control and a way to minimize neural operations (Graf et al., 1995).

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Conflict of interest

The authors declare that they have no conflict of interest, financial or otherwise, related to the submitted manuscript or the associated research

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Incorporating voluntary unilateral Knee Flexion into Balance Corrections elicited by multi-directional Perturbations to Stance

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Incorporating voluntary unilateral knee flexion into balance corrections elicited by multi-directional perturbations to stance

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Abbreviations: AL, anteriorlateral; ANOVA,analysisofvariance; AP, anterior–posterior; APA, anticipatory postural adjustment; CoM, center of mass; CONT, contralateraltotilt; EMG, electromyography; Glut Med, gluteusmedius; IPS, ipsilateraltotilt; IRED, infrared emitting diode; KO, kneeonly; Lat, medio-lateral; KOI: Knee only ipsilateral; KOC: Knee only contralatera; LPo, lateral posterior; Para, paraspinal; PO, perturbationonly; PoL, posterior lateral; SEM, standard error of the mean.

Abstract

Positive effects on lateral CoM shifts during balance recovery have been seen with voluntarily unilateral arm raising but not with voluntarily bilateral knee flexion. To determine whether unilateral voluntary knee movements can be effectively incorporated into balance corrections we perturbed the balance of 30 young healthy subjects using multi-directional rotations of the support surface while they simultaneously executed unilateral knee flexion. Combined pitch and roll rotations (7.5 deg and 60 deg/s) were presented randomly in six different directions. Subjects were tested in four stance conditions: balance perturbation only (PO); cued flexion of one knee only (KO); combined support surface rotation and cued (at rotation onset) flexion of the uphill knee, contralateral to tilt (CONT), or of the downhill knee, ipsilateral to tilt (IPS). Outcome measures were centre of mass (CoM) motion, biomechanical and EMG responses of the legs, arms and trunk. Predicted measures (PO+KO) were compared with combined measures (CONT or IPS).

Unilateral knee flexion of the uphill knee (CONT) provided considerable benefit in balance recovery. Subjects rotated their pelvis more to the uphill side than predicted. Downhill knee bending (IPS) also had a positive effect on CoM motion because of a greater than predicted simultaneous lateral shift of the pelvis uphill. KO leg muscle activity showed anticipatory postural activity (APA) with similar profiles to early balance correcting responses. Onsets of muscle responses and knee velocities were earlier for PO, CONT, and IPS compared to KO conditions. EMG response amplitudes for CONT and IPS conditions were generally not different from the PO condition and therefore smaller than predicted. Later stabilising responses at 400 ms had activation amplitudes generally equal to those predicted from the PO+KO conditions.

Our results suggest that because EMG patterns of anticipatory postural activity of voluntary unilateral knee flexion and early balance corrections have similar profiles, the CNS is easily able to incorporate voluntary activation associated with unilateral knee flexion into automatic postural responses. Furthermore, the effect on movement strategies appears to be non-linear. These findings may have important implications for the rehabilitation of balance deficits.

Key words: Balance Corrections, Postural control, Muscle responses, CNS motor programs

Introduction

Adequate balance recovery plays a critical role in preventing falls (Maki et al., 2007). For rehabilitation focussed on fall avoidance, it is important to know how involuntary or automatic movements of the extremities influence balance control and how additional voluntary movements of the arms and legs may aid balance corrections. Some insights may be obtained by initiating voluntary body motion during quiet stance and studying the subsequent influence on human postural stability. Several studies showed that early anticipatory postural adjustments (APAs) seen just prior to main voluntary contractions in the leg and arm muscles help to offset the destabilising influence of later voluntary movements (Hughey and Fung, 2005; Nashner and Cordo, 1981; Pozzo et al., 2001). This approach, however, provides no direct information on the influence of APAs of voluntary movements on balance corrections. An approach that might provide this information would be to examine automatic balance corrections triggered by a perturbation to stance when these are supplemented with additional voluntary limb movements designed to enhance balance recovery. To achieve stability, the voluntary movements should presumably have similar metrics to those of automatic responses.

Compensatory arm movements have been shown to play a major role in balance recovery (Allum et al., 2002; Carpenter et al., 2004; McIlroy and Maki, 1995). Examples include grasping a handrail (McIlroy and Maki, 1995) to recover balance or breaking the fall by stretching out the arms in the fall direction

(Allum et al., 2002). It has been suggested that automatic arm responses aid centre of mass (CoM) stabilisation over the base of support (Küng et al., 2009a) and/or act as a protective "damping" mechanism in the event of an impending fall (Maki and McIlroy, 2006; Allum et al., 2002). The role of lower limb responses in balance recovery has also been investigated. The main focus was, however, on comparing in-place versus stepping reactions. Stepping reactions expand the base of support in the direction of falling. Thereby, the range of CoM displacement that can be accommodated without loss of stability is increased (Maki and McIlroy, 1997). It is an open question whether balance-correcting responses in the legs, which consist of flexing the uphill and extending the downhill knee (Allum et al., 2008, Küng et al., 2009a) can be enhanced using triggered voluntary movements.

By comparing independent voluntary activation and automatic postural responses elicited during unexpected perturbations, insights into how the central nervous system (CNS) integrates feedforward and feedback information into balance corrections can be obtained. Although they found a number of marked dissimilarities between voluntary and automatic postural responses, Nashner and Cordo (1981) also found similarities in response latencies when voluntary movements were well-practiced, executed in a predictable direction and performed under conditions of postural stability. These studies, however, were restricted to the sagittal plane. When laterally directed movements were studied, more dissimilarities were found between these two kinds of responses (Hughey and Fung, 2005). This effect appeared to be due to the different goals and biomechanical constraints of voluntary activation compared to automatic postural responses resulting from unexpected balance perturbations. For example, during voluntary leg lifts, co-contractions of hip muscles aid stiffening of the pelvis position and thereby reduce medial-lateral movements (Hughey and Fung, 2005). In contrast, the response to a lateral support-surface tilt is to roll the pelvis into the perturbation direction and the trunk in the opposite direction (Allum et al., 2008; Bakker et al., 2006). Thus a dissimilarity between laterally directed voluntary activation and balance control responses appears to be the amount of pelvis stabilisation. If a voluntary leg movement is to aid balance control it would seem important that the voluntary movement and the automatic balance correction have a similar muscle response synergy and movement strategy.

Following this line of reasoning, a number of authors have developed the approach of examining whether voluntary movements can be integrated in balance corrections and thereby aid these corrections. Burleigh et al., (1994) showed that there is a similarity between the automatic postural responses to an external perturbation and APAs of voluntary movements. The main difficulty of integrating the latter into balance corrections is that the muscle forces of APAs and voluntary movements may, at the same time, provide sensory inputs that disturb the internal reference needed to plan balance corrections following perturbations to stance (Massion, 1992). An example of this occurs during integration of voluntary bilateral knee bending into the balance correcting strategy (Oude Nijhuis et al., 2007). For a forward tilt of the support surface, Oude Njihuis and colleagues (2007) found that bilateral voluntary knee flexion can be well integrated into the balance correction. But they also found strong support for the notion that the additional APAs created by voluntary movements can disrupt the balance correcting strategy (Oude Nijhuis et al., 2007) by opposing the attempt of the CNS to create oppositely directed limb movements during balance corrections (Hughey and Fung, 2005). The disruption Oude Nijhuis et al. (2007) noted during backward tilts resulted from the dissimilarity between the postural synergy initiating the balance correction and the APAs of bilateral voluntary knee flexion movements. Maintaining balance after a backward tilt of the surface required activation of anterior leg muscles whereas knee flexion is achieved primarily by activation of posterior leg muscles.

In contrast, the incorporation of voluntary activation into balance corrections worked well for voluntary uphill arm raising (Grin et al., 2007). Such voluntary action reduced the lateral downhill motion of the CoM. Given the finding that bilateral knee bending is not well integrated into balance corrections for backward tilts and has no additional stabilising effect on lateral displacement of the CoM (Oude Nijhuis et al., 2007), the question arises whether voluntary *unilateral* knee bending would be better integrated into balance corrections, especially because uphill knee flexion is fundamental to stable balance during lateral tilts (Allum et al., 2008; Bakker et al., 2006, Küng et al., 2009a).

The purpose of the current study was to examine the interactions between balance corrections elicited by unexpected rotational perturbations of the support surface and synergies due to simultaneously executed voluntary unilateral knee flexion. The question arose how voluntary unilateral knee flexion synergies alter the inter-segmental shaping of automatic balance corrections. It could also be that flexing the downhill rather than the uphill knee might suppress the initial automatic postural responses and thereby also alter the pre-programmed balance correcting response synergy. We presumed that additional knee flexion of the uphill knee would reduce the lateral shift of the CoM and be well integrated into balance corrections but that flexion of the downhill knee would not. Thus an overall aim was to investigate whether the muscle synergies for voluntary knee movements and automatic balance corrections were similar in the leg muscles and whether these were well integrated when performed simultaneously. If voluntary unilateral knee movements improve stability, then this study would

provide a basis for patients, with a tendency to fall, to learn an appropriate balance recovery strategy. In those with pathologically reduced knee flexibility, such as patients with cerebellar ataxia (Bakker et al., 2006) or vestibular loss (Allum et al., 2008), such a rehabilitation might prove particularly useful.

Materials and Methods

Subjects

30 healthy young subjects without neurologic or orthopaedic deficits were recruited for this study (mean ± SEM: age 25 ± 0.8 years; height 176 ± 1.6 cm; and weight 67 ± 2.1 kg). All subjects gave witnessed, written informed, consent to participate in the experiments according to the Declaration of Helsinki. The Institutional Ethical Review Board of the University Hospital of Basel approved the study.

Protocol

Subjects stood on a servo-controlled platform that could tilt in the pitch and roll directions. The roll and the pitch axis of the platform had the same height equal to the average distance of the ankle joint to the soles of the feet. The subjects' feet were lightly strapped into heel guides fixed to the upper surface of the movable platform. The heel guides were adjusted to ensure that the ankle joint axes were aligned with the pitch axis of the platform and prevented stepping reactions when stance perturbations occurred. The roll axis passed between the feet. The stance width was standardised (14 cm) and two handrails of adjustable height were located 40 cm from the sides of the platform centre. Subjects were informed that they were allowed to grasp the handrails if they needed support. One assistant was present to lend support in case of a fall, but no falls, and just five near falls (defined as a need to grasp the handrail or receive assistance) occurred for backward perturbations (113 deg, 203 deg, and 248 deg directions, as defined below) when voluntary flexion of the downhill knee was required.

The test protocol was identical to that of Grin et al. (2007). Stimuli consisted of rotations of the platform in 6 different directions with a constant velocity of 60 deg/s and a constant amplitude of 7.5 deg. Perturbation directions were 6 combinations of pitch and roll rotations. Our convention is to define the 0 deg direction as a pure-pitch perturbation with toes down, 90 deg as a pure right tilt, 180 deg a pure-pitch toe-up perturbation. Directions used were forward right (45 deg), backward right (113 deg, 158 deg), backward left (203 deg, 248 deg), and forward left (315 deg). Each perturbation direction was presented seven times in random order to the subject for each task condition. Task conditions were: perturbation of stance using a support surface rotation only (PO); combined support surface rotation and voluntarily flexing the uphill knee (contralateral to tilt: CONT); combined support surface rotation and flexion of the downhill knee (ipsilateral to tilt: IPS); flexing one knee during quiet standing (Knee only: KO). In KO, CONT and IPS conditions, simultaneous visual and auditory triggers prompted the participants to flex the knee.

Simultaneous cues were used to reinforce the command to flex the appropriate knee. The auditory cue consisted of a 50 dB sound pressure level 1000 Hz tone that was produced by either right or left loudspeakers, positioned at the height of the participants' knees, to signal the knee to flex. This auditory cue sounded until it was automatically switched off when movement sensors (light barriers) detected 30 degrees of knee flexion (same auditory cueing used by Oude Nijhuis et al., 2008). Trigger lights were located at eye level, approximately 4 m in front of the participants. A green light stimulus appeared in the left visual field and at the same time a loud-speaker to the left of the participant sounded when the participant was expected to flex their left knee. The instruction to the subject was to flex the expected knee as rapidly as possible in response to the visual and auditory cues, with the specific goal to switch off the sound. Further, flexion of the opposite knee was required to be as small as possible. For consistency, the same auditory cue present in the KO, CONT and IPS conditions sounded at the onset of platform movement in the PO condition as well, but then the specific instruction was to respond naturally to the balance perturbation.

The PO condition was always presented first in order to exclude learning effects of knee flexion on this control condition. For this condition, 43 trials randomised over the 6 directions were presented; one first trial in the 158 direction plus 7 trials per 6 directions. Secondly, 26 KO trials were presented to train unilateral knee flexion to 30 degrees. We chose 30 deg based on pilot experiments. These showed that 30 deg was the maximum flexion subjects were comfortable with (Oude Nijhuis 2009). A small (approximately 0.1 deg), just perceptible (see Beule and Allum 2006), pure roll perturbation was presented with the auditory and visual knee bending prompts in order to have a directional somatosensory cue present without perturbing balance and to duplicate the cue protocol used by Grin et al, 2007. Thirteen of the KO trials were KOI trials presented serially. That is, knee bending was cued to the side of the 0.1 deg tilt (6 left and 6 right plus 1 first trial excluded from analysis). For KOC the small tilt was opposite to the knee cued to be flexed. KOC trials were also presented serially. The order of KOI and KOC series was randomised across participants. Finally 43 trials under CONT and 43 trials under IPS conditions were presented. The order of CONT and IPS presentations was also randomised across subjects. The first trial of each sequence (158, 203, 203 directions for PO, IPS and CONT conditions, respectively) was analysed separately to minimize 1st trial effects on the main body of data (Keshner et al., 1987, Oude Nijuis et al, 2009). To minimize fatigue, participants were given a

3 - 4 minute seated rest after each sequence. Trials were initiated automatically and were preceded by a random 5 - 15 s inter-stimulus delay. During this time period, visual feedback of the subjects' own anterior-posterior (AP) and medio-lateral (Lat) ankle torque was presented to the subject on a cross with light emitting diodes. This visual feedback was used to maintain a standardised pre-stimulus subject position across trials (see Allum et al., 2008).

Data collection

Recordings of biomechanical and electromyographic (EMG) data commenced 100 ms prior to perturbation onset and terminated 1s later. To record EMG activity, pairs of silver-silver chloride electrodes were placed bilaterally approximately 3 cm apart along the muscle bellies of gastrocnemius medialis, biceps femoris (hamstrings), rectus femoris (quadriceps) and unilaterally on left tibialis anterior, left gluteus medius (Glut Med), and on left paraspinals (Para) at the L1 - L2 level of the spine. EMG recordings were analog band-pass filtered between 60 and 600 Hz, full-wave rectified, and low-pass filtered at 100 Hz prior to sampling at 1 kHz.

Full body kinematics were collected using a three-dimensional optical tracking system with 21 infrared emitting diodes (IREDs) (Optotrak®, Northern Digital Incorporation, Waterloo,Ontario,Canada). The Opto-trak cameras sampled the IRED signals at 64 Hz and were placed approximately 4 meters in front of the subject. IREDs were placed bilaterally on the following anatomical landmarks: frontally at the lateral malleolus; center of the patella; frontally at the greater trochanter; anterior superior iliac spine; radial styloid process; elbow axis; acromion; chin; angulus sterni; and on a headband placed just above the ears. Three IREDs were placed at the front corners and the left side of the platform to define the pitch and roll movements of the platform. Subjects wore tight fitting shorts and vests to prevent marker movements with respect to the skin.

Support surface reaction forces of both feet were measured from strain gauges embedded within the rotating support surface. The strain gauges were located under the corners of the plate supporting each foot. From forces recorded perpendicular to the support-surface by the strain gauges under the left/right foot and the distances to the centre of ankle joint rotation, the AP and lateral ankle torques were calculated for the left/right foot. The torques from the left and right foot were added together and displayed to the subject as exclusions on two rows of diodes mounted on a cross 4 m from the subject as described above.

Data analysis

Seventy seven out of total 4650 trials (1.7%) trials were excluded from analysis because of an incorrect voluntary response. Knee flexion was deemed to be in error when the participants bent the knee in the opposite direction to that required by the visual and auditory cues, flexed both knees, or flexed the incorrect knee followed by the correct knee. These errors lead to a minimum of 3 trials out of 7 possible in one direction for 4 subjects.

Primary variables of interest were centre of mass (CoM) movements, arm, trunk, pelvis and knee angle and angular velocity profiles as well as muscle EMG responses of the legs, arms and trunk. Following analogue to digital data conversion, kinematic data and EMG signals were averaged offline across each perturbation direction. Zero latency was defined as the onset of platform rotation. Subject average time traces were pooled to produce population average traces for a single direction for presentation in the figures. Responses for directions with the same pitch stimulus component but oppositely directed roll were pooled for analysis. The pooled measures were termed anterior lateral (AL) for directions 45 deg and 315 deg, lateral posterior (LPo) for 113 deg and 248 deg, and posterior lateral (PoL) for 158 deg and 203 deg.

Kinematic analysis

Kinematic data was digitally filtered at 16 Hz using a zero phase shift 4th order Butterworth filter. Total body CoM displacement was calculated separately for the AP, Lat and vertical directions using a 12 body segment adaptation (see Visser et al., 2008) of a 14 segment model (Winter et al., 2003). Two trunk segments (upper and lower trunk) were used instead of four. In addition we calculated the following angular displacements: absolute trunk angle (roll and pitch), absolute pelvis angle (roll and pitch) and ankle, knee and shoulder joint angles. Absolute rotation angles of the planes defined by trunk and pelvis body segments and the platform surface were defined using 3 or 4 markers on these segments. Arm abduction was calculated as the angle between the upper arm and trunk segment. Knee and ankle joint angles were calculated using angles between segments either side of the joint (Visser et al., 2008). Stimulus induced changes were calculated with respect to values over a pre-trigger time interval of 90 ms ending 10 ms prior to stimulus onset. Kinematic analyses was conducted at the time of maximal divergence of CoM velocity between the IPS and CONT conditions (at 250 and 530 ms), at peak velocities of body segments, at plateau times (750 ms) of the trunk, pelvis, knee angular movements, and at plateaus times (650 ms) of pelvis lateral movement, difference in flexion of the left and right knees, as well as the difference in arm abduction in abduction of the left and right arms.

To analyse the knee motion in detail the onset of voluntary knee flexion with stimulus onset calculated as the divergence of knee velocity from bounds equal to the mean (approximately 0 deg/s) ± 2 standard deviations (SD) of knee velocity during the pre-trigger interval (-100 to -10 ms). To determine if knee velocity profiles were shifted or delayed for the CONT and IPS, conditions with respect to KO conditions, a knee flexion velocity threshold of 50

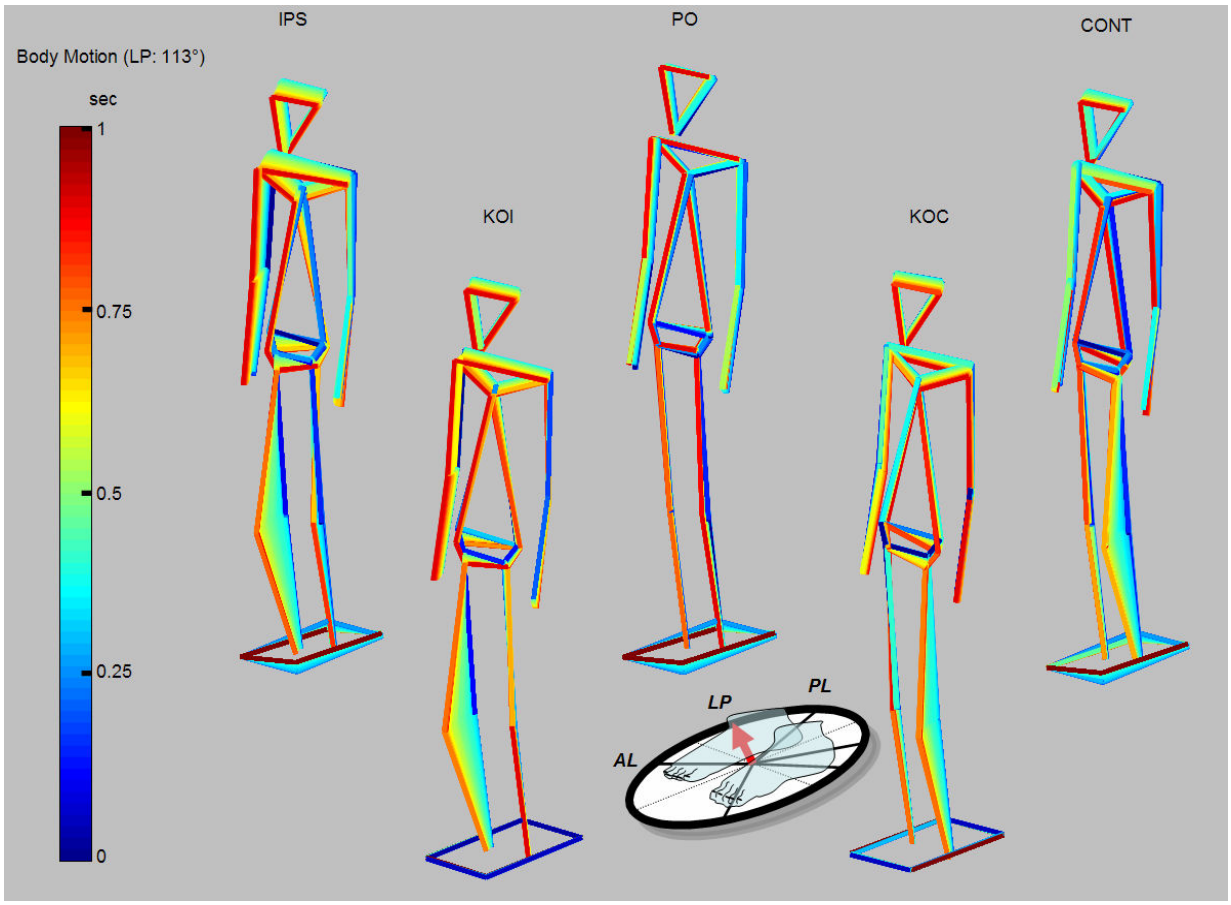

Figure 1: Stick figures of a subject performing voluntary unilateral knee bending alone (KO), simultaneous knee bending at onset of a backward-right perturbation (CONT and IPS) and, the response to perturbation only (PO).

deg/s defined. This threshold allowed for distinction between the automatic knee responses for the PO and CONT, IPS, and KO conditions. The 50 deg/s threshold was based on the maximum flexion velocity for the PO condition. This velocity had a mean of 41.6 deg/s with a standard error of the mean (SEM) of 2.1 deg/s (thus the mean plus 3 SEM equalled 47.9 deg/s) across all directions (population means across directions were: 65.1 deg/s for AL directions 45.9 deg/s LPo, and 33.8 deg/s for PoL). Time to peak knee flexion for the CONT and IPS conditions was determined by subtracting the onset time of 0 deg/s knee flexion from the time of velocity peak.

EMG analysis

Each EMG response was corrected for background activity by subtracting the average level of pre-stimulus activity measured over a 90 ms period ending 10 ms prior to perturbation onset. Then analysis techniques similar to those previously employed (Grin et al., 2007) were used. The onset of activity was defined for each muscle based on the perturbation direction showing the greatest amplitude of activity prior to 250 ms. From the time of peak activity, the analysis algorithm looked backwards in time until activity first reached a level lower than mean pre-stimulus activity plus 2.5 SD. This time was defined as response onset. Then areas were calculated over 130 ms from this onset time for each individual response. A second response area was calculated similarly to Grin et al (2007) over a fixed interval from 500 ms to 800 ms post stimulus.

Statistics

First, three-way repeated measures ANOVA's (side: right vs. left x condition: CONT vs. IPS vs. PO x direction) were conducted using a significance level of 0.05 in order to justifying pooling 45 deg with 315 deg directions (AL directions), 113 deg with 248 deg (LPo) and 158 deg with 203 deg (PoL). Having determined that side did not influence the results our analysis we concentrated on between-conditions comparisons of PO, CONT and IPS using three pre-planned ANOVA's. First, two-way repeated measures ANOVA's (CONT vs. IPS vs. PO x direction) were conducted on all dependent measures using a significance level of 0.05. Then similar two-way ANOVA's compared the differences between combined and predicted responses, that is IPS vs. PO+KOI and CONT vs. PO+KOC, where the summed responses PO+KOI and PO+KOC are the predicted responses for IPS and CONT, respectively. Significant difference effects between knee bending were evaluated within each pooled perturbation direction using Bonferroni post-hoc tests.

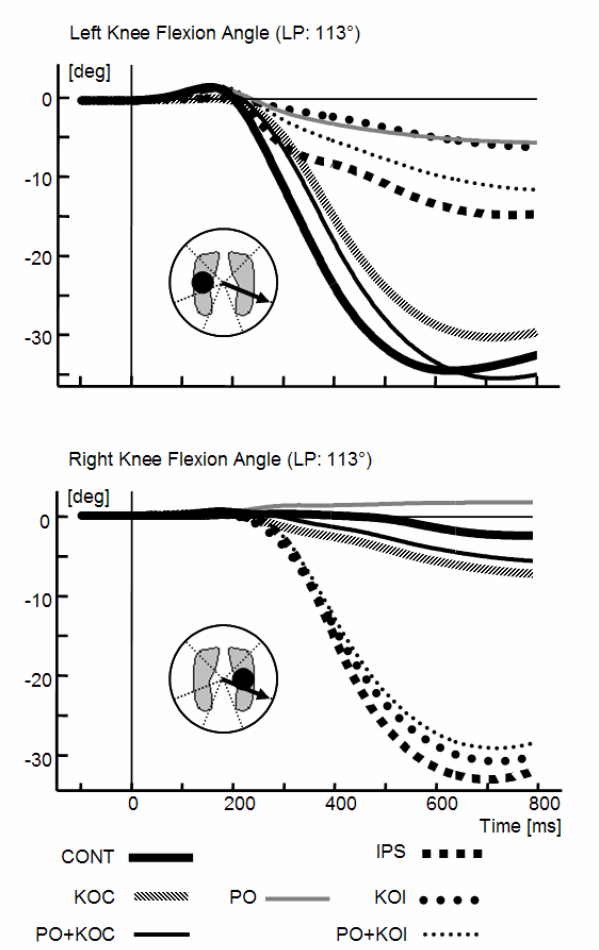

Figure 2: Mean population traces of left and right knee flexion under different stimulus conditions as well as predicted flexion (PO+KOC and PO+KOI traces). Start of the support surface tilt and cue for the knee bending is marked by a vertical line at 0 ms.

Results

Average stick figures of one subject for all conditions in response to a lateral tilt (113 deg) are shown in figure 1. As described below, some kinematic features, for example, the total amplitude of knee bending were predictable from the sum of the PO and KO conditions. However, several body segment velocity profiles, including knee velocities, were not predictable from these two conditions. These differences lead to more stability than predicted for CoM profiles. Nonetheless, when bending the downhill knee for the IPS condition, subjects clearly had more difficulties to maintain balance. Subjects were quite stable under the CONT condition.

Kinematics

Automatic and voluntary knee flexion responses

Subjects flexed their uphill knee slightly under the PO condition some 3–8 deg by 750 ms post stimulus onset (Fig. 2), depending on the perturbation direction (AL: 8.0 ± 0.8 deg; LPo: 5.8 ± 0.7 deg; PoL: 3.2 ± 0.5 deg). Thus a directional effect for knee angle ($F(5,95) = 50$, $P < 0.001$), emerged. AL directions induced the greatest knee flexion. Subjects acquired a mean knee flexion in the KO condition of 32.0 deg (± 0.9 deg). 30 deg of flexion was the goal in the study design. As shown in Fig. 2, when voluntary knee flexion occurred in addition to support surface tilt perturbations, larger knee flexions than 32 deg were obtained for all directions ($P < 0.001$), except PoL. Thus the uphill knee flexion under the CONT condition was for AL 37.5 ± 0.9 deg, for LPo 38.2 ± 0.9 deg, and for PoL 33.2 ± 1.0 deg (PoL flexions were not significantly different from those of KO. A directional effect was present under the CONT condition ($F(5,95) = 590.1$, $P < 0.001$). Our analysis revealed similar changes under the IPS condition, again with a directional effect ($F(5,95) = 218.9$, $P < 0.001$). Overall, the predicted amplitudes of knee flexion, PO+KOC or PO+KOI, were not significantly different from those of CONT and IPS, respectively, with the exception of the uphill knee for the IPS condition (see below).

Although only unilateral knee flexion of the downhill knee was requested under the IPS condition, the uphill knee showed an additional flexion (Figs. 1 and 2). Total flexion of the uphill knee under IPS was for AL 19.6 ± 1.7 deg; for LPo 16.8 ± 1.7 deg, and for PoL 11.8 ± 1.6 deg. These amounts differed from the amount of flexion of the uphill knee for PO ($F(2,176) = 159.2$, $P < 0.001$). The largest difference occurred for AL (11 ± 1.5 deg)

Onset times of knee flexion velocities

Figs. 3A and 3B show the knee angular velocity traces for the right tilt LPo direction, 113 deg, under the various tilt conditions. Panel A in figure 3 shows the knee angular velocity traces of the uphill (left) and B the downhill (right) knee. For the PO condition the onset of uphill knee flexion velocity occurred earlier for forward compared to backward tilt directions (AL: 127 ± 3.8 ms; LPo: 163 ± 6.1 ms; PoL: 193 ± 1.0 ms). For the CONT and IPS conditions similar latency differences across tilt direction were obtained leading to a directional effect for all three conditions (PO: $F(5,353) = 16.3$, $P < 0.001$; CONT: $F(5,349) = 18.4$, $P < 0.001$; IPS: $F(5,351) = 22.5$, $P < 0.001$). In figure 3A the onset times for the PO, CONT and IPS conditions are marked by a single vertical arrow because the onset times for this right LPo direction were very similar (PO: 163 ± 6.1 ms, CONT: 161 ± 4.9 ms, IPS: 160 ± 5.1 ms) as was the case for other directions. In short, for each direction no differences in onset times for uphill knee flexion were found between PO, CONT and IPS conditions. In comparison to the knee flexion onset times for the KO condition, those in the uphill knee for CONT condition were faster in the AL and LPo directions but not for the PoL direction (AL by 63 ± 6.0 ms, $P < 0.001$; LPo by 23 ± 5.5 ms, $P = 0.002$, PoL by 3 ± 6.1 ms, no statistically significant difference). Likewise onset times were faster for the uphill knee under the IPS condition (AL by 76 ± 7.3 ms, $P < 0.001$; LPo by 39 ± 6.8 ms, $P < 0.001$, PoL by 14 ± 8.7 ms, no difference).

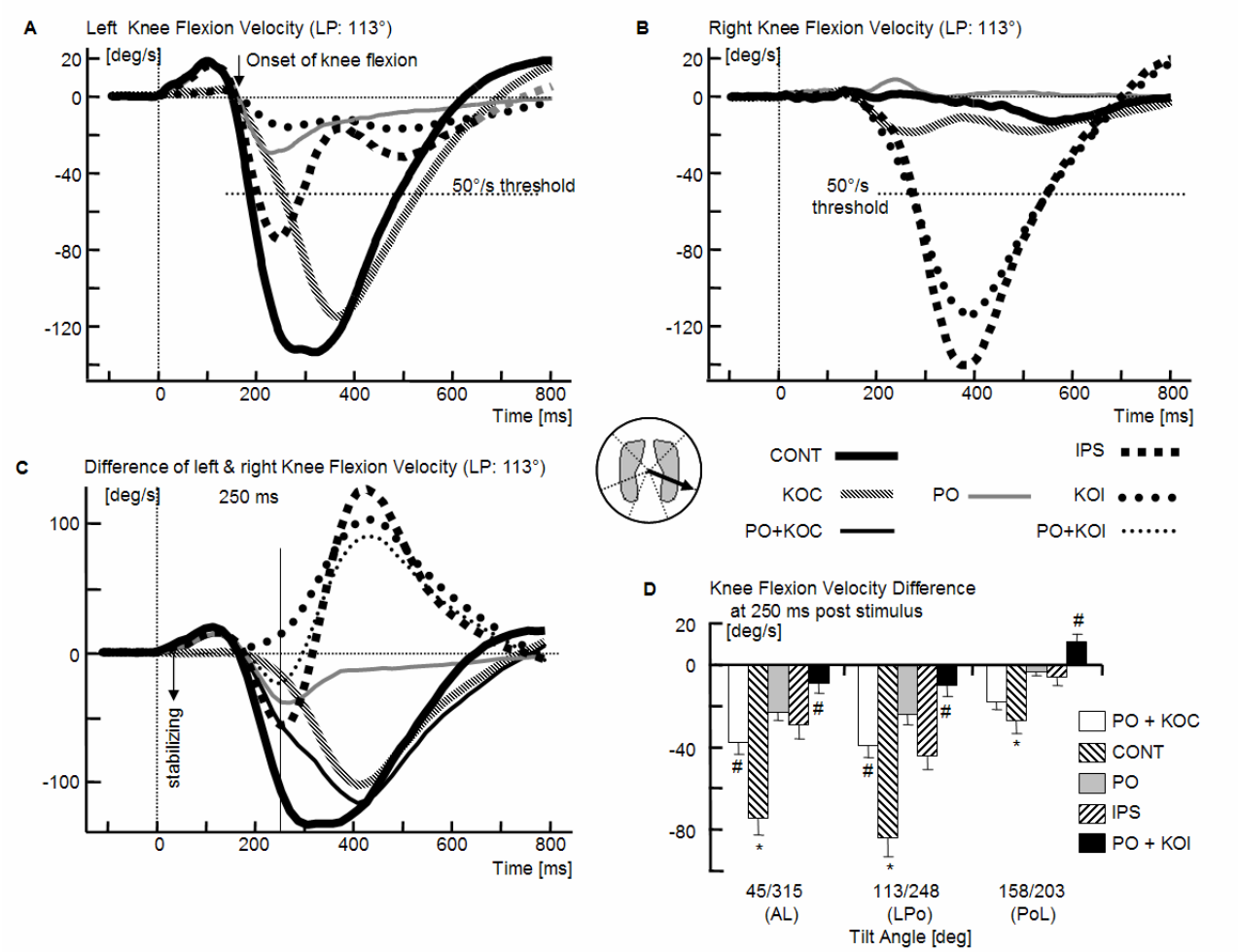

Figure 3: Mean population traces of the left (A) and the right (B) knee angular velocity across conditions for a backward-right tilt. Start of the support surface tilt and the cue for the knee bending, is marked by the vertical dotted line. The short dotted horizontal line marks the threshold used as onset of the voluntary knee bending. C shows the traces of the difference of left and right knee angular velocity across conditions for a backward-right tilt. The difference is quantified in D which shows mean difference in flexion velocity (as columns) based on the value at 250 ms (vertical line in C). Standard errors of the means (SEM) are shown by vertical bars on the columns

When we considered the time to reach a 50 deg/s-threshold (as marked in Figs. 3A & B by a horizontal line) those for CONT were equal to 215 ± 10 ms for AL, 251 ± 8 ms for LPo and 292 ± 10 ms for PoL. CONT knee flexions were faster than those of KO as follows: AL by 108 ± 10.5 ms ($P < 0.001$), LPo by 69 ± 7.8 ms ($P < 0.001$), and PoL by 27 ± 8.1 ms ($P < 0.001$). Thus the times from onset (0 deg/s) to 50 deg/s were also faster under CONT than KOC conditions by 25 to 45 ms with greater differences for lateral directions. IPS knee flexions (in the downhill knee) were not faster than those of KOI concerning the time to the 50 deg/s threshold, except for the AL direction which was faster by 88 ± 1.3 ms ($P < 0.001$). CONT knee flexion velocity reached the 50 deg/s-threshold faster than for the IPS condition for all perturbation directions (AL by 21 ± 11.1 ms, LPo by 69 ± 7.0 ms, PoL by 45 ± 8.2 ms, all $P < 0.001$).

Timing of peak knee flexion velocity

Peak amplitude of knee flexion velocity did not differ between CONT and IPS in any direction, reaching, for example, 150 ± 4.0 deg/s for the LPo direction (see also Fig. 3). The time from flexion onset (0 deg/s time) to the peak of knee velocity was shorter for CONT than for the KO ($F(1,114)= 11.5$, $P = 0.001$): AL by 73 ± 10.3 ms, $P < 0.001$, LPo by 38 ± 11.0 ms, $P = 0.006$, and PoL by 17 ± 10.3 ms, no statistical difference. This time was also shorter for IPS than for KO conditions in the PoL direction by 33 ± 12.4 ms ($P = 0.035$). In the LPo condition no difference was seen, and it was longer for AL by 47 ± 11.0 ms ($P < 0.000$). These CONT vs IPS condition differences with direction occurred because the absolute time to peak velocity was faster under CONT then IPS conditions ($F(1,115) = 9.8$, $P = 0.002$): AL by 28 ± 10.1 ms, no statistical difference, LPo by 42 ± 9.3 ms, $P = 0.01$; and PoL by 53 ± 9.4 ms, $P = 0.001$). The absolute times of the velocity peak under the CONT condition were for AL: 368 ± 13.8 ms, for LPo: 354 ± 14.0 ms, and for PoL: 385 ± 15.5 ms. Thus, generally under the CONT condition, knee flexion started at the same time as under PO condition but flexion was more rapid than under IPS or KO condition. These results on knee velocity timing showed no statistical difference between the left and right knees (before pooling for

AL, LPo and PoL directions). Therefore we conclude that 'footedness' (similar to hand preference) had no influence on the results of the current experiments.

Differences between uphill and downhill knee velocities

As it is the difference in knee flexion velocity (uphill knee flexion greater than that of downhill knee) that creates a lateral stabilising effect rather than the knee flexion velocity of a single leg, (Küng et al., 2009a) we examined this difference in detail. Fig. 3C shows the difference of left and right knee flexion velocity for the LPo direction 113 deg and depicts the direction of bilateral stabilising knee action. After 200 ms, the initial and earlier knee flexion of the uphill knee compared to the downhill knee can be observed for the IPS and CONT conditions. For the IPS condition, the difference in knee velocity reverses after 300 ms to a destabilising direction. At the time of the peak of stabilising action for the IPS condition (250 ms – see vertical line in Fig. 3C), differential knee velocities for IPS and CONT were greater than predicted values (Fig. 3D).

The stabilising effect of the peak difference in knee flexion of the CONT condition occurred earlier than the main destabilisation peak in knee difference velocity for the IPS condition (condition effect: $F(1,118) = 5.6$, $P = 0.02$). For AL no significant difference was seen, for LPo the difference was 50 ± 13.7 ms, and for PoL 59 ± 20.5 ms. The destabilising peak of the IPS condition occurred at a similar time compared to KO (406 ± 12.2 ms). The peak times of the CONT condition were less than for KO conditions in all directions ($F(1,118) = 4.8$, $P = 0.030$): AL by 38 ± 13.5 ms, LPo by 52 ± 13.9 ms and PoL by 21.4 ± 16.7 ms. The stabilising peak for the CONT condition also occurred earlier with respect to 0 deg/s flexion onset compared to KO (AL by 38 ± 13.5 ms, LPo by 52 ± 13.9 ms, PoL by 21 ± 16.7 ms. $F(1,118) = 4.8$, $P = 0.030$). The peak was also earlier than predicted from the PO and KOC conditions as may be seen in figure 3 ($F(1,115) = 14.082$, $P < 0.001$). The difference was 38 ± 7.5 ms in the AL direction, 46 ± 7.7 ms in the LPo direction and 11 ± 6.0 ms in the PoL direction. The peak amplitudes of the difference in knee flexion were the same for CONT and IPS for all directions (mean: 149 ± 3.3 deg/s) and larger than these for KO which had a mean of 122 ± 2.6 deg/s (CONT $P < 0.001$ and IPS $P = 0.040$). CONT amplitudes of the difference in left-right knee velocities were also larger than predicted from the PO+KOC conditions ($F(1,115) = 6.6$, $P = 0.01$) by 17 ± 6.3 deg/s in AL, 30 ± 6.4 deg/s in LPo and by 13 ± 6.4 deg/s in PoL directions. Thus the stabilising effect of the CONT condition, based on differential knee flexion, was earlier and faster than predicted and faster than the destabilising effect of the IPS condition. Nonetheless the destabilising phase of the IPS condition was preceded by a stabilising phase of knee flexion which ended at approximately 300 ms (Figs. 3C and D).

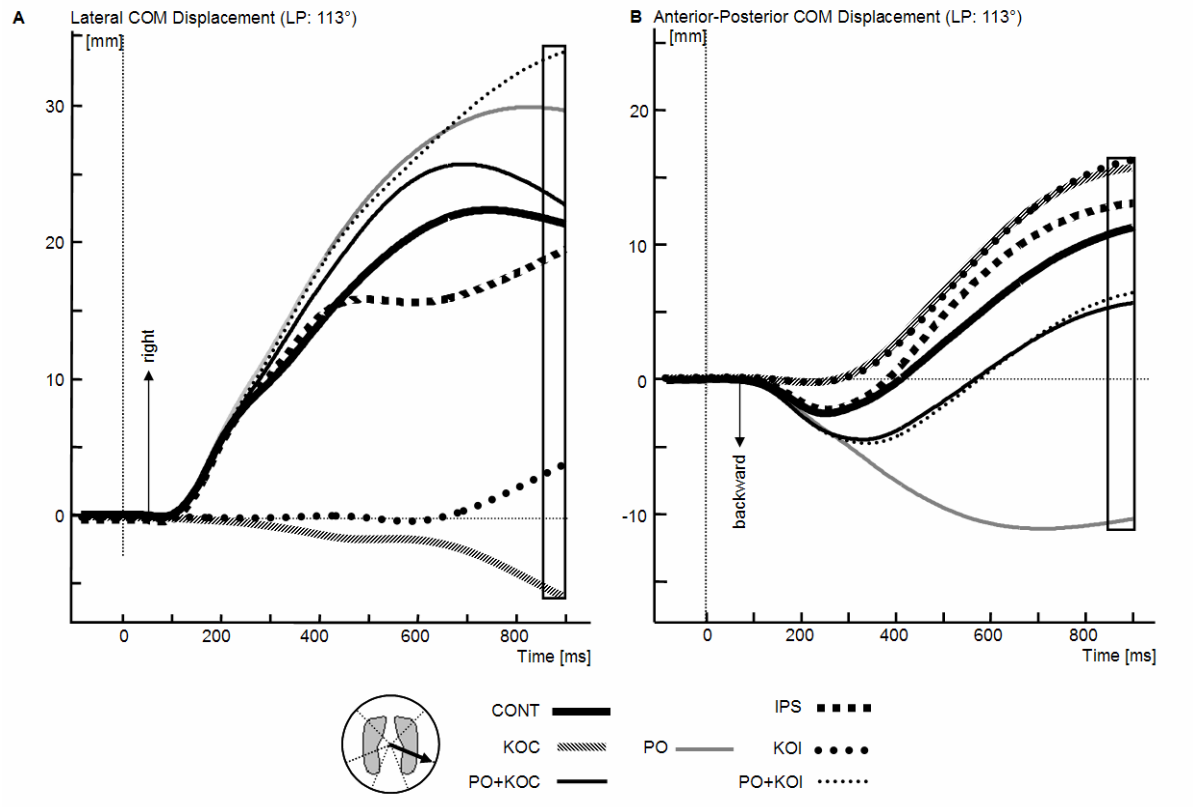

Figure 4: Mean population traces of lateral (A) and anterior-posterior (B) CoM position under 5 conditions (two directions of knee bending only) for a backward-right perturbation.

CoM displacements

Fig. 4A shows the position traces for CoM in the lateral direction and Fig. 4B in the AP direction. IPS and CONT conditions both had a positive effect on the CoM displacement as measured at 850 – 900 ms. Analysis of CoM displacements at these times revealed that flexing the contralateral (CONT) as well as flexing the ipsilateral (IPS) knee was associated with reduced (therefore more stable) deviations of CoM.

Lateral CoM

As Fig. 5A documents, Lat CoM displacement under the CONT condition clearly benefited from unilateral knee bending for all tilt directions with most benefit in the AL direction in comparison to PO values. For the IPS condition, a similar reduction in Lat CoM position compared to PO can be seen (for values at 850 - 900 ms, see Fig. 5A). These differences led to a condition effect when compared to PO condition (CONT: $F(2,2) = 10.0$, $P = 0.002$; IPS: $F(2,2) = 10.0$, $P < 0.001$). Lat CoM displacement was far less (that is more stable) than predicted under the IPS condition ($F(1,2) = 63.7$, $P < 0.001$). CONT showed in comparison to the predicted effect (PO+KOC) a direction by condition effect ($F(1,86) = 5.3$, $P = 0.006$) with AL values for CONT being smaller than predicted by 7 ± 1.5 mm ($P = 0.030$ – see also Fig. 5A).

Our analysis was focused on random series trials with the first trial excluded. CoM measures of the first trial were, however, also examined. The first trial under the IPS condition showed a significantly greater Lat CoM displacement ($p<0.05$) in comparison to subsequent random trials (Fig. 5C). No difference was observed for CONT conditions.

It may be suspected from the CoM position traces in Fig. 4 and previous studies (Grin et al., 2007, Küng et al., 2009a), that earlier changes in CoM velocity profiles accounted for the differences in CoM displacement at 850 - 900 ms. Fig. 6A, for example, displays the condition differences in Lat CoM velocity

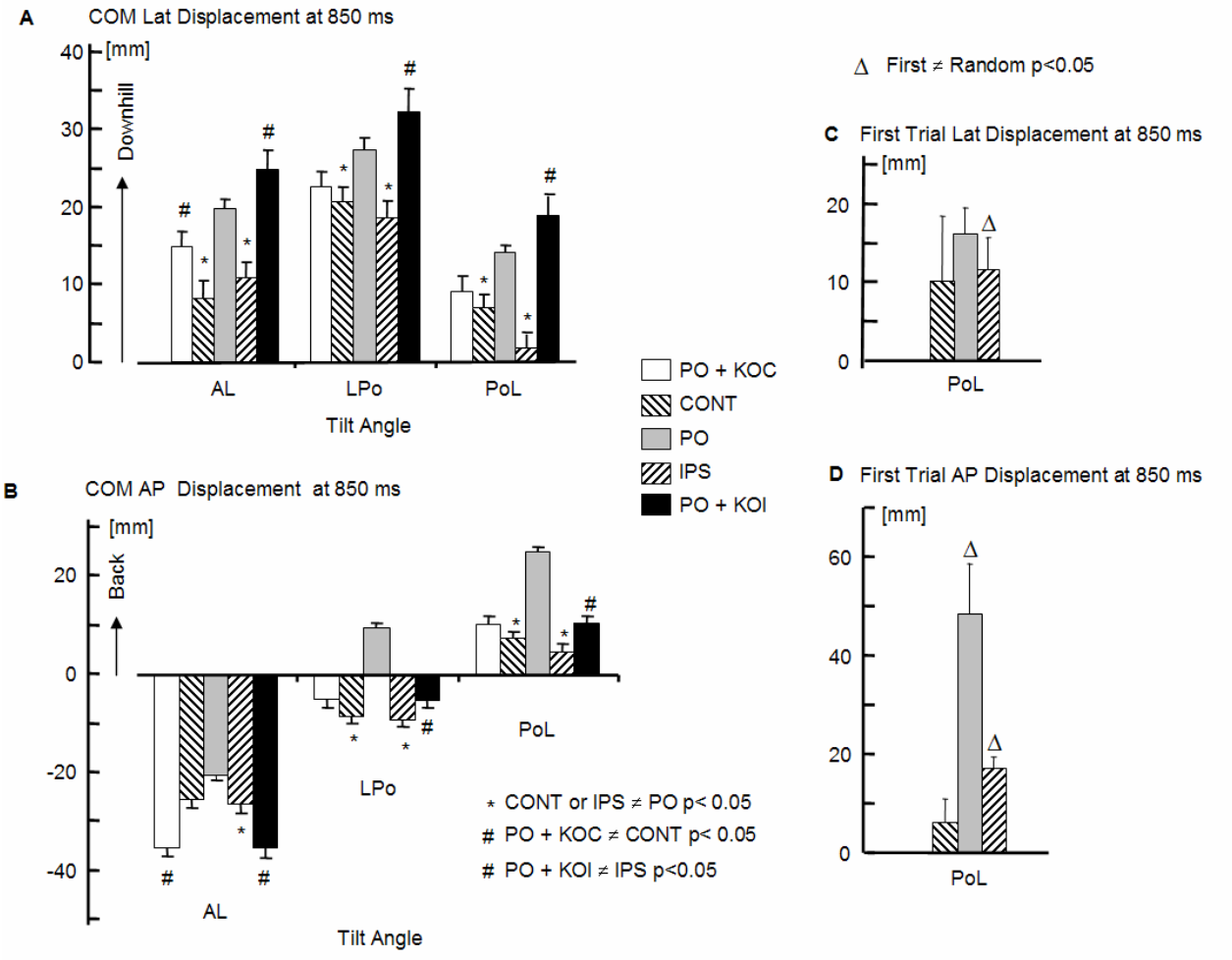

Figure 5 Effect of voluntary knee bending on lateral (A) and AP (B) CoM displacements with the first trial excluded, and for the first trial only (C and D). **A** shows mean values (+ SEM) of lateral CoM displacements in response to all perturbation directions. The columns represent the population means in each condition over the period 850 – 900 ms from stimulus onset. Equal directions of left and right roll for the same pitch direction were pooled together into one dataset. *Asterisks* (*) on the columns indicate significant differences between the PO and CONT or PO and IPS conditions, a gate (#) symbol indicates significant differences between the CONT and predicted (PO + KOC) or IPS and predicted (PO + KOI) means. **B** shows mean values (+ SEM) of AP CoM displacements in response to all perturbation directions. The columns represent the population means in each condition over 850 – 900 ms from stimulus onset. In C and D the mean lateral and AP CoM displacements are shown for the first trial under PO, CONT and IPS conditions. This trial was always in the PoL direction.

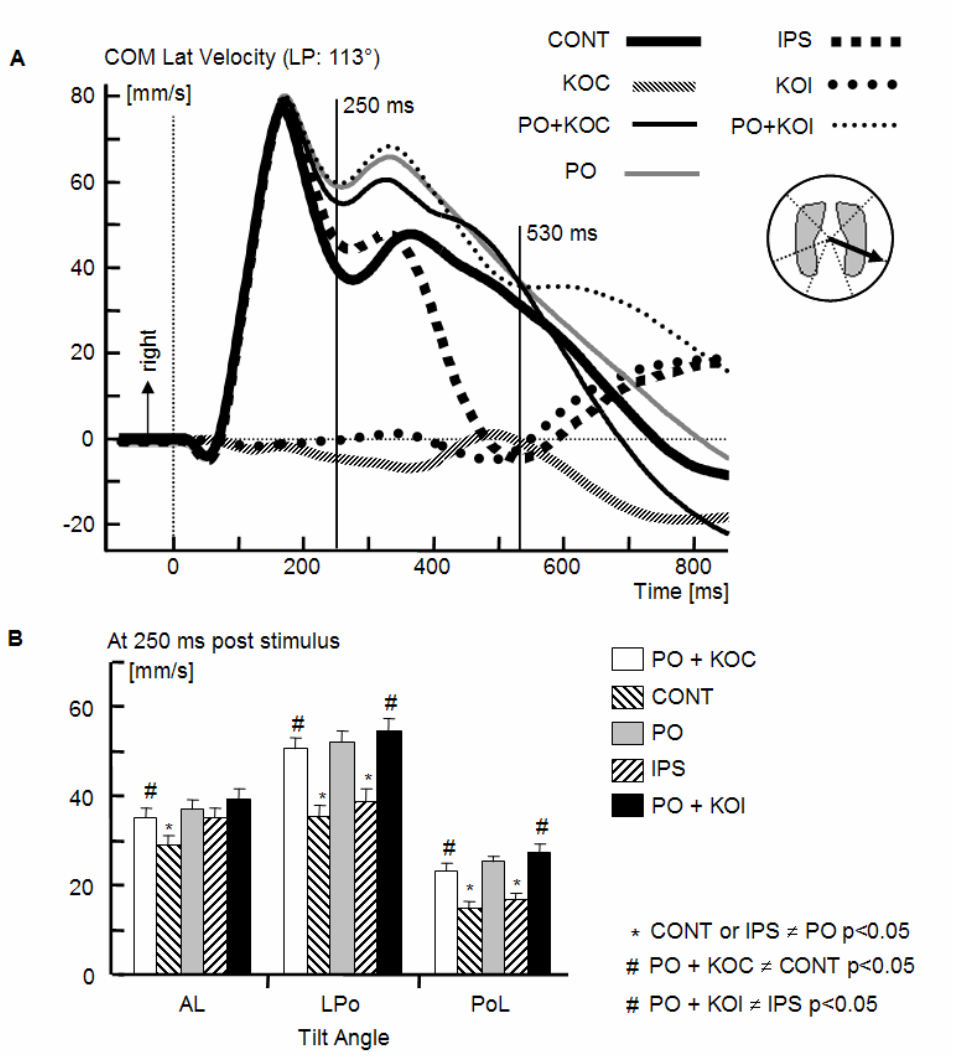

Figure 6: Mean population traces of lateral CoM velocitiy across conditions for a backward-right tilt (A) and mean values of lateral CoM velocity across directions and conditions (B) measured at 320 ms (when a second relative maximum is reached; see *vertical line* in A). For details refer to the legends of Fig. 4A.

profiles for a perturbation backwards to the right (113 deg). At the time of the first peak of the Lat CoM velocity around 170 ms, no change was observed across tilt conditions. However, this first peak is followed by a negative (stabilising) peak at around 250 ms (marked by the vertical line in Fig. 6A). At this time point, significant differences in Lat CoM velocity were evident between conditions for all tilt directions. Fig. 6B shows that under both CONT and IPS conditions, Lat CoM velocity is decreased with respect to that for PO ($F(2, 168) = 12.4$, $P < 0.001$) CONT: AL by 8 ± 1.7 mm/s, LPo by 17 ± 1.9, PoL by 10 ± 1.4 mm/s; IPS: AL by 2 ± 1.5 mm/s, LPo by 12 ± 2.0, PoL by 8 ± 1.4 mm/s). Post hoc tests within each perturbation direction revealed that both bending the contralateral uphill knee (CONT) as well as the downhill ipsilateral (IPS) knee significantly reduced Lat CoM velocity in the direction of the tilt for all directions except the AL direction for IPS (see Fig. 6B). Both ANOVAs and post hoc tests (see Fig. 6) revealed that these changes were greater than predicted (PO+KOC vs. CONT: $F(1,114) = 15.6$, $P < 0.001$ and PO+KOI vs. IPS: $F(1, 111) = 12.6$, $P = 0.001$). The values of CoM velocity at 250 ms post stimulus were correlated with stabilising values of knee velocity marked at 250 ms in figure 2C (CONT: $R = 0.99$, $P < 0.001$; IPS: $R = 0.98$, $P = 0.001$).

Given the phase of destabilising differential knee velocity described above for the IPS condition, it was remarkable to note a further decrease in Lat CoM velocity for the IPS condition at 530 ms (marked by a vertical line in Fig. 6A). In all directions the Lat CoM velocity at 530 ms for the IPS condition was less than that predicted for the PO and KOI conditions ($F(2,168) = 19.6$, $P < 0.001$), in the direction AL by 32 ± 4.1 mm/s, for LPo by 32 ± 4.3 mm/s, and for PoL by 33 ± 4.0 mm/s. This reduction in CoM velocity at 530 ms was followed by an increase in CoM velocity see Fig. 6A so that the relative velocity between CONT and IPS was reversed at 850 ms with differences ($F(1,168) = 13.5$, $P < 0.001$) as follows: AL by 23.2 ± 4.9, LP by 22.0 ± 5.5, PL by 12.2 ± 4.6 mm/s.

AP CoM

AP CoM displacement (measured at 850 – 900 ms) was shifted forward for CONT & IPS conditions compared to PO (Fig. 4B). As Fig. 5B shows, the shift of the AP CoM displacement compared to PO was

smaller for the AL direction and more forward for the LPo and PoL directions. For the LPo and PoL directions the forward shift was not different from that predicted under CONT conditions. For AL directions, the shift was less than predicted (Fig. 5B). Compared to experiments in which voluntary unilateral arm abduction was combined with a balance perturbation (Grin et al., 2007), the amount of forward shift of the CoM with unilateral knee bending was significant greater in each perturbation direction ($P < 0.05$), but less than that obtained with bilateral knee bending (Oude Nijhuis et al., 2007). From the point of stability, the forward shift was significantly less ($P < 0.05$) for backward directions (PoL) than with bilateral knee bending (Oude Nijhuis et al., 2007).

AP CoM showed a first trial effect under PO and IPS conditions (Fig. 5D). For PO, this result confirmed previous research (Oude Nijhuis et al, 2009).

Pelvis displacements

In addition to knee velocities, we investigated pelvis, trunk and arm movements in order to establish possible segment contributions to the CoM velocity changes described above. We describe these in ascending order, pelvis, trunk and arms.

Figs. 7A and 7C show that when the support surface is tilted laterally, the pelvis initially rolled and was shifted laterally in the same direction as the support surface rotation. After 300 ms, IPS knee flexion resulted in increased pelvis roll downhill as predicted but, importantly, a much less than predicted shift of the pelvis lateral position downhill occurred (Figs. 7B & D). CONT knee flexion reversed the initial pelvis downhill roll. For example in the LPo directions, CONT caused a change in roll of 9.4 ± 0.6 deg with respect to PO, and IPS caused an increased downhill roll of 5.8 ± 0.5 deg. Thus a strong condition effect between CONT vs. PO vs. IPS ($F(2, 177) = 304.0$, $P < 0.001$) was observed. For the IPS condition the pelvis roll was as predicted from the PO and KO conditions (Fig. 7B) but for the CONT condition pelvis roll was always greater than predicted except in the PL direction (Fig. 7B). Fig. 7C illustrates the horizontal lateral deviation of the pelvis, which was always downhill in the direction of tilt. As noted above, the shift downhill under the IPS condition was less than predicted (Fig. 7D). This change occurred because pelvis lateral velocity at 500 ms post stimulus was reversed to uphill. The predicted pelvis velocity from the PO+KOI conditions and that of IPS clearly differed ($P < 0.001$ for all directions, data not illustrat-

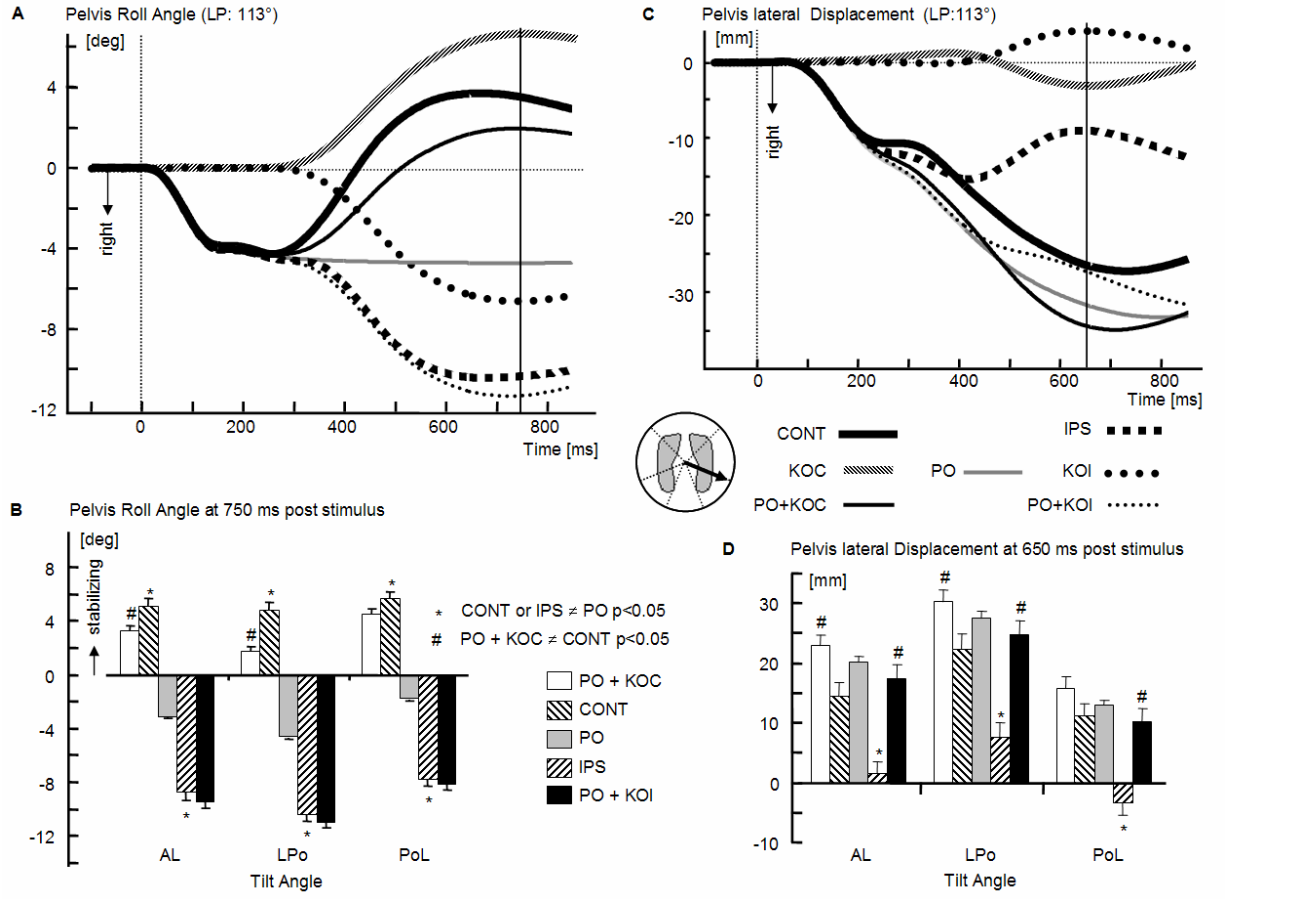

Figure 7: Mean population traces of pelvis roll (A) and pelvis lateral horizontal displacement (C) across conditions for a backward-right tilt and mean values of pelvis roll across directions and conditions measured at 750 ms when pelvis roll displacement plateaued (B) and at 650 ms (D) for pelvis lateral position (see *vertical line* in A). For details refer legends of Fig. 2 and 3.

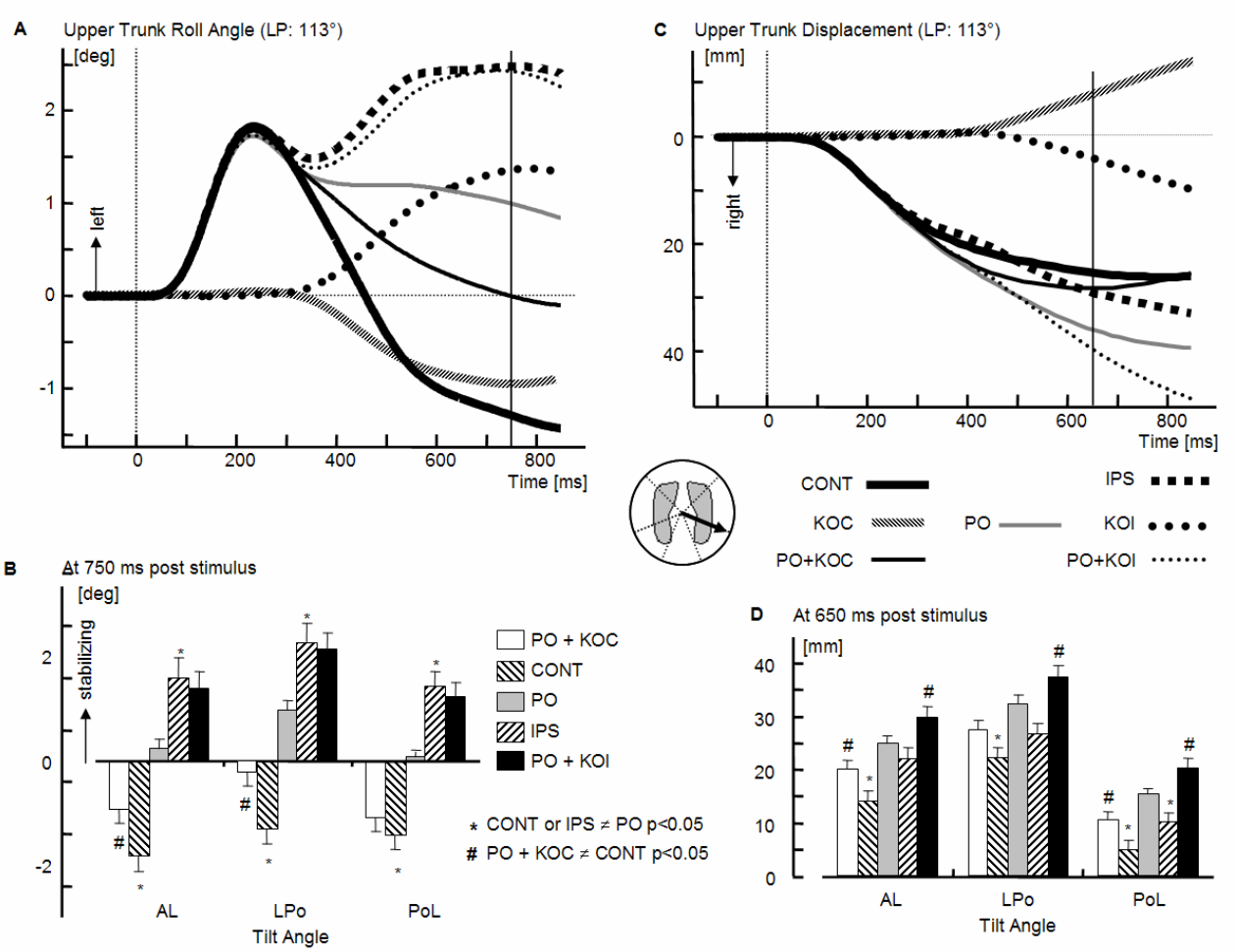

Figure 8: Mean population traces of trunk roll displacements (A) and trunk lateral shift (C) across conditions for a backward-right tilt. **B** shows mean values of trunk roll across directions and conditions measured at 750 ms when trunk roll displacement plateaued (see 2nd *vertical line* in A) and at 650 ms for trunk lateral position (D). For details refer legends of Fig. 2 and 3

ed). Furthermore, the correlation of the difference in predicted (PO+KOI) versus IPS measures for pelvis lateral velocity at 500 ms and lateral CoM velocity at 530 ms was high, R = 0.98 (P < 0.001).

Trunk displacements

Fig. 8A illustrates that when the support surface was rotated into a lateral direction, initially trunk roll occurred in the direction opposite to the perturbation. Bending the uphill knee under the CONT condition caused the trunk to rotate later in the opposite direction, whereas IPS knee bending induced more trunk roll in the same, uphill direction (Fig. 8A). Thus at 750 ms post stimulus, when trunk roll plateaued under the IPS condition, a condition effect (IPS vs. PO vs. CONT) was observed ($F(2, 176) = 38.1$, $P < 0.001$). Fig. 8B illustrates the findings at 750 ms. Post hoc tests show that the predicted trunk roll (PO+KOI) did not differ from that for IPS, whereas under CONT conditions trunk roll was more downhill than predicted (AL by 0.9 ± 0.2 deg and LPo by 1.0 ± 2.4 deg – see Fig. 8B) under CONT conditions. Note, however, that both pelvis and trunk angular movements were greater than predicted, however in opposite directions (compare Fig. 7B with 8B under CONT conditions). Fig. 8C shows that the more than predicted lateral shift of the pelvis under IPS conditions was not counterbalanced by trunk segment lateral shifts due to the trunk rolling in the opposite direction. When we examined the lateral shift of the trunk, its shift was less than predicted, that is, always in a more stabilising direction under CONT and IPS conditions (Fig. 8D). Note that it is the upper trunk segment which is plotted in Fig. 8. A measure of the lower trunk segment, independent of the pelvis and upper trunk segments, was not available due to the choice of marker positions used for motion analysis. Although actual minus predicted measures of stabilising CoM velocity at 530 ms were highly correlated with the same measures IPS-(PO+KOI) for trunk lateral velocity at 500 ms ($R=0.99$, $p<0.001$) the slope of this relationship was 3 orders of magnitude less for trunk than for the pelvis segment, indicating a much lower influence of trunk lateral motion on CoM motion.

Roll perturbation of the support surface and subsequent balance correcting knee flexion simultaneously induces a trunk pitch rotation (Küng et al., 2009b). Thus a greater effect on trunk pitch can be expected with extra voluntary knee bending than

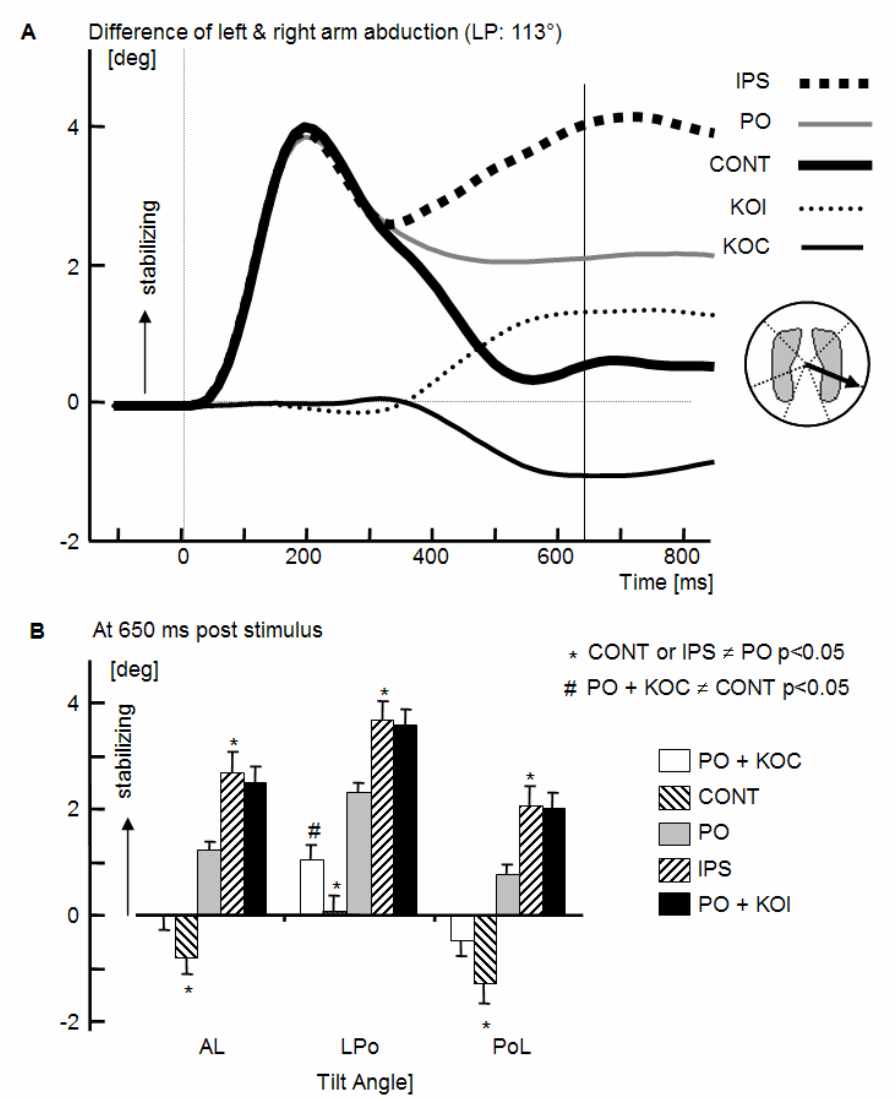

Figure 9: Mean population traces of the difference of the left and right arm abduction angular displacement across conditions (A) and mean values across direction and conditions measured at 650 ms when arm abduction angle difference plateaued (B) (see *vertical line* in A). For details refer legends of Fig. 2 and 3

under the PO condition. Whereas forward rotation of the support surface caused only slight trunk backward pitch (less than 1 deg) and backward perturbations resulted in 2 degs forward pitching of the trunk (direction effect: $F(2, 59) = 77.29$, $P < 0.001$). Bending one knee (regardless if IPS or CONT) induced about 2 deg more forward pitch of the trunk than the PO condition.

Arm abduction

As the plots in Figs. 1 and 9A would suggest, the difference of left and right arm abduction showed smaller stabilising arm motion for the CONT than the PO conditions. For AL directions this amounted to 2.0 ± 0.3 deg less, LPo 2.2 ± 0.4 deg, and PoL 2.0 ± 0.4 deg when measured at 650 ms. There was greater stabilising arm motion with respect to PO for the IPS condition (Figs. 1 and 9A), AL by 1.5 ± 0.4 deg, LPo by 1.4 ± 0.3 deg, and PoL by 1.3 ± 0.3 deg, (CONT vs PO vs IPS effect $F(2,181) = 37.0$, $P < 0.001$). As shown by the column plots of Fig. 9B, arm position for CONT and IPS at 650 ms post stimulus was not different from predicted, except for LPo direction. That is, while arm movements helped stability under the IPS condition this improvement was as predicted and there was no evidence of arm movements accounting for the late shift in CoM stability under the IPS condition.

Muscle responses

Leg muscles

Generally early muscle responses for the CONT and IPS conditions were less than predicted and not different from those of the PO condition whereas later stabilising responses were as predicted. No condition effects, IPS or CONT), compared to PO were observed in the lower leg muscles, tibialis anterior and gastrocnemicus.

Figs. 10 and 11 illustrate the hamstrings (BF) and quadriceps (RF) muscle responses of left leg for the two lateral directions (113 deg, left and 248 deg, right). Over the early 130 ms response interval (86-216 m) covering the first pulse of muscle balance-correcting activity from its onset no condition effect between CONT, PO, IPS conditions for either BF or RF was noted (Fig. 10 and 11, left column plots). The

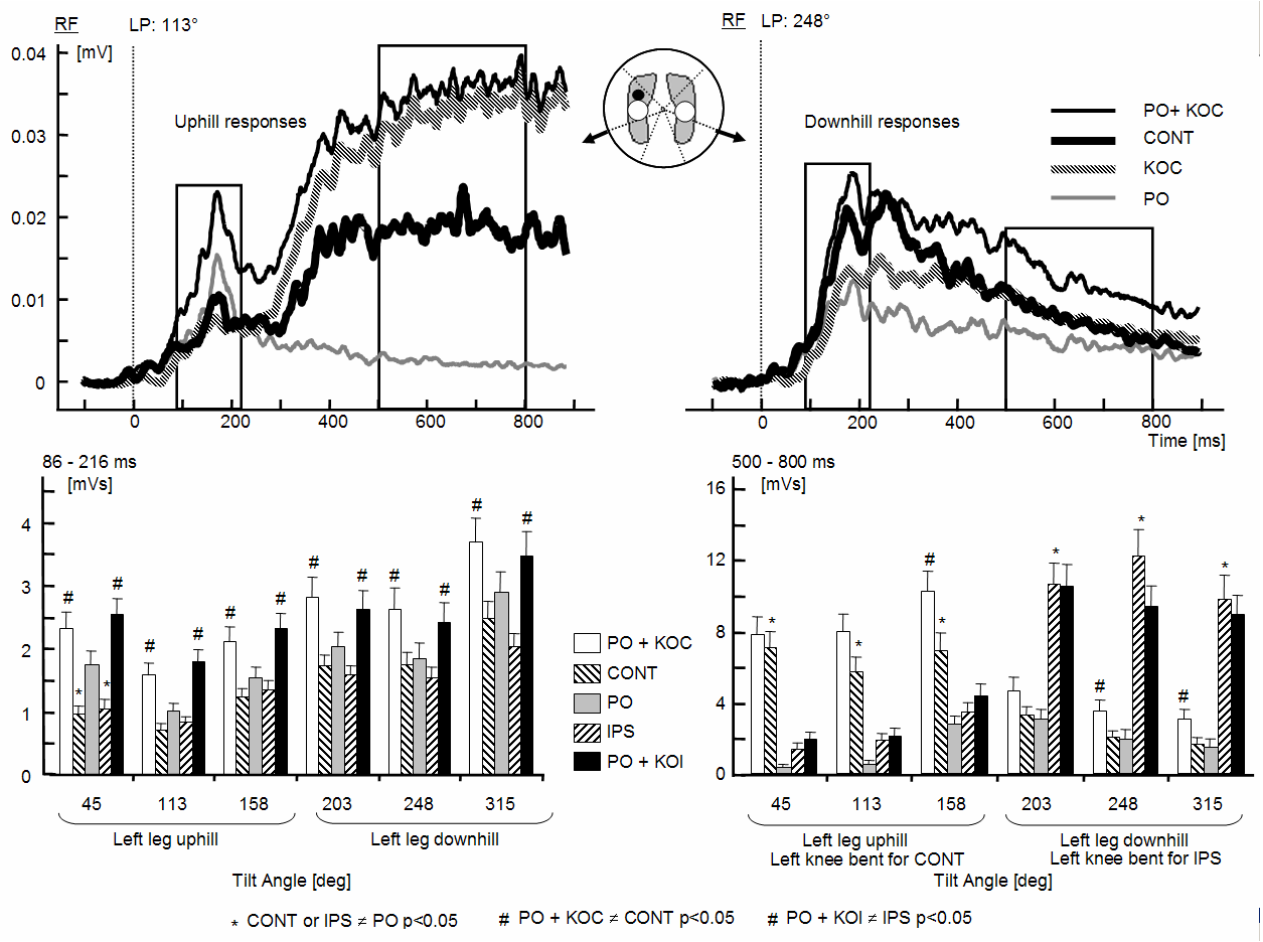

Figure 10: Responses of Quadriceps (RF) muscle with knee bending conditions. The upper sets of population traces are for a backward-right (113°) and backward-left (248°) tilt. Population means and standard error of two different intervals across directions and conditions are shown by column plots.

IPS and CONT responses were less than predicted response amplitudes for this interval, that is the sum PO+KOI or PO+KOC, was greater than the IPS and CONT responses (BF: IPS: $F(1,118) = 8.5$, $P = 0.004$, CONT: $F(1,116) = 8.0$, $P = 0.006$; RF: IPS $F(1,114) = 16.3$, $P < 0.001$, CONT $F(1,112) = 12.9$ $P < 0.001$) for almost every direction (see Figs. 10 and 11). A different pattern emerged for the later stabilising interval 500 – 800 ms post stimulus. A clear effect of voluntary knee bending in BF and RF muscles was present (Fig. 10 and 11, right column plots) consistent with the supporting function of the flexed and extended legs. Thus activity over the 500 – 800 ms interval was greater than for PO conditions ($F(1,113) = 36.2$, $P < 0.001$) and mostly as predicted in the uphill quadriceps for CONT conditions (see Fig. 10 right); although there was a direction by condition effect for CONT vs PO+KOC ($P = 0.001$). In the downhill hamstrings, the CONT late responses were different from those of PO condition (see Fig. 11 right) ($F(1,118) = 34.0$, $P < 0.001$) and CONT vs. PO+KOC responses showed a direction by condition effect ($F(1,117) = 2.6$, $P < 0.001$). When the IPS condition was considered, quadriceps activity was greater than PO and as predicted for the bent downhill leg (Fig. 10), and hamstrings activity for the uphill leg was greater than PO as the knee of this leg needed to be flexed more than under the PO condition (Fig. 11).

Onset times of knee muscle activity were examined in the direction of maximal response (for left hamstrings this was the 45 deg direction, and for left quadriceps, 203 deg). No changes in muscle onsets were detected in CONT and IPS conditions compared to KO for quadriceps. Decreased onsets were seen in hamstrings for which onset times for CONT were shorter than under KO conditions (93 ± 9.6 ms compared with 127 ± 8.0 ms, $P = 0.009$).

Trunk muscles

Similar changes in response amplitudes as seen for the knee muscles were observed in the paraspinal (Para) muscles. An exception was that early balance correcting activity for CONT and IPS was only less than predicted for the AL directions (45 and 315 deg). Late stabilising activity increased as predicted based on the increased trunk lateral flexion (see Fig. 8). That is for left tilts and the CONT instruction (see Fig. 12, upper right), there was more activity required in the left Para as the trunk was held more downhill. In the other trunk muscles from which we recorded, gluteus medius (Glut Med), the changes under various test conditions were similar but less significant.

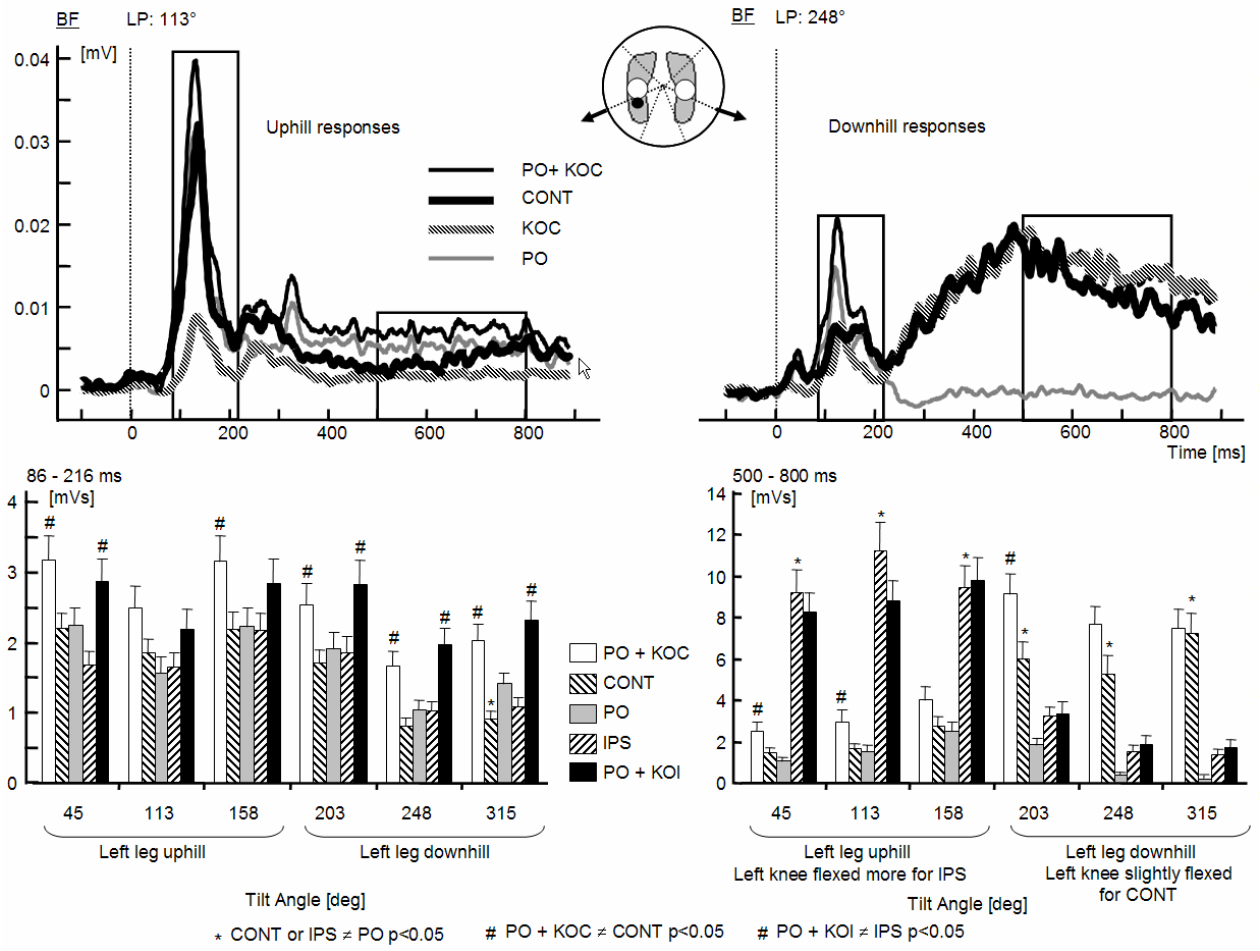

Figure 11: Changes to the left Hamstrings (BF) muscle activity with knee bending conditions. The layout of the figure is identical to that of Fig. 9.

Onset time of muscle activation in the maximal response direction of left Para (113 deg) was clearly faster ($p \leq 0.001$) for CONT, IPS and PO (105 ± 6.2 ms for CONT) than for KO (147 ± 7.4 m) - compare traces in Fig. 12 left - whereas no difference was detected between onset times for CONT, IPS and PO. In the maximal response direction of left Glut Med (248 deg) onset times were faster ($p \leq 0.001$) for CONT, IPS and PO (67 ± 4.0 ms for CONT) compared to KO (104 ± 3.9 ms).

Discussion

In previous studies we have demonstrated that flexion of the knee contralateral to tilt is an integral part of the automatic postural response correcting imbalance on lateral and on forward tilt of the support surface (Allum et al., 2008; Bakker et al., 2006). Without adequate flexion of the uphill knee and extension of the downhill knee, lateral CoM motion is unstable. Interestingly, flexing both knees at once with onset of tilt leads to an instability for backwards AP tilts but little improvement in stability for lateral tilts (Oude Nijhuis et al., 2007). Thus in this study our aim was to determine if unilateral knee flexion aided stability or not across all directions of tilt.

Improvements in stability with unilateral knee bending

Generally we found that simultaneously executing unilateral knee bending at the onset of a perturbation to balance aided stability, as determined by CoM displacements observed at 850 ms, for both lateral and backward directed tilts. The improvement in stability was least for AP displacement during forward tilt. The amount of improvement was mostly equal or slightly less than predicted from responses to the perturbation alone (PO) and knee bending alone (KO) when the knee contralateral to tilt direction was bent (CONT). Surprisingly the amount of improvement was also considerable when the knee ipsilateral (IPS) to the support surface tilt was bent and more than predicted from the PO and KO conditions. The improvement was noted in early changes in CoM lateral velocity with knee bending under CONT and IPS conditions, leading to reduction in downhill directed CoM velocity between 200 and 600 ms post tilt onset. Under the IPS a further reduction in CoM lateral velocity was present peaking at 500 ms. Thus we can conclude that regardless of which knee is flexed, unilateral knee flexion provides an improvement in lateral and AP stability as measured by CoM displacements and velocities. This result is in marked contrast to the unchanged lateral stability for lateral tilts and decreased AP stability when bilateral knee

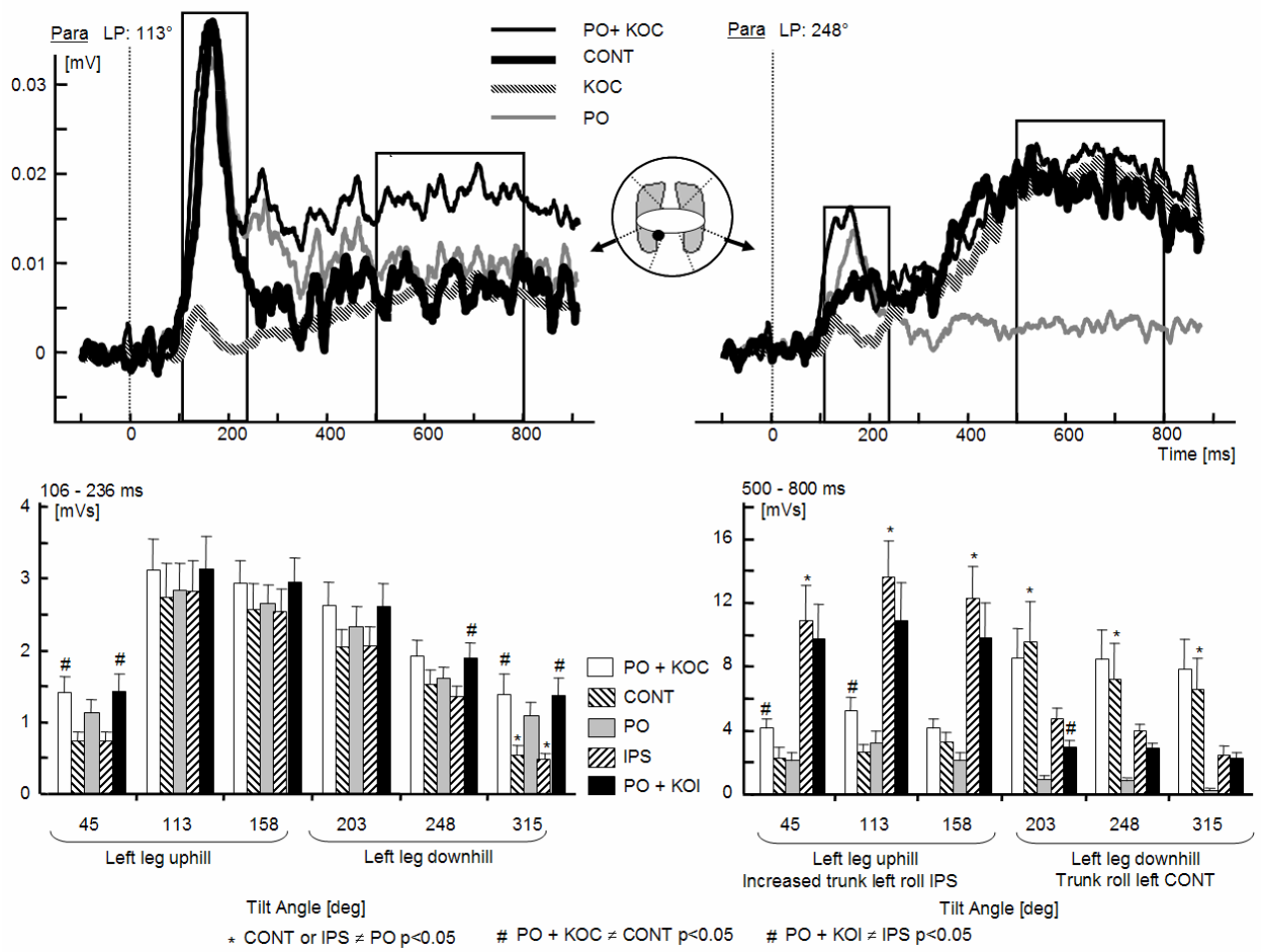

Figure 12: Effect of knee bending conditions on Paraspinal EMG responses.

bending is performed on tilt onset (Oude Nijhuis et al., 2007). This difference is reinforced by the marked reduction in near falls in our study compared to those recorded by Nijhuis et al. (2007). Nonetheless it should be noted that all near falls in the current study were under the IPS condition. Furthermore a destabilising first trial effect on CoM was also present under the IPS but not under the CONT condition. Thus we conclude that training voluntary flexing of the uphill knee (CONT) will be of most use in the rehabilitation of balance deficits.

Similar improvements in CoM lateral velocity as in the current study were obtained when the arm contralateral to tilt was abducted 90 deg (Grin et al., 2007). CoM lateral movements became unstable, however, when the arm ipsilateral to tilt was raised, and no improvement was obtained in AP CoM displacement (Grin et al., 2007). The significant conclusion we have reached regarding fall avoidance is that bending the uphill knee but also flexing the downhill knee will improve both AP and lateral stability more than bending both knees or raising an arm. In the following, we explore the movement strategies and muscle synergies associated with combining unilateral knee flexion and the automatic postural response of balance corrections to support surface tilt.

Measurements of knee velocities revealed that even when a voluntary flexion of the downhill knee was requested, considerably more flexion of the uphill knee occurred than for the PO condition. The opposite was not true when voluntary flexion of the uphill knee was requested. Then a slight flexion of the downhill knee occurred. As the subjects did not know in advance which knee to flex because the directions of platform tilt were presented randomly, we assume this flexion in the contralateral uphill knee when voluntary flexion of downhill knee is requested is an anticipatory reaction, which needs to be executed before the voluntary driven flexion in the downhill knee occurs. Presumably for this reason the main peak of voluntary knee flexion is delayed under the IPS condition with respect to CONT. Regardless of its origin, this early stabilising knee action under the IPS condition leads to a reduction in CoM lateral velocity around 250 ms.

Voluntary arm raising induced major changes in upper body movements when executed with balance correcting responses, but few changes in lower body movements (Grin et al., 2007). Most of the upper body movements involved rotations of the upper trunk with only minor changes to the pelvis rotation in comparison to the PO condition. In contrast, major

changes in the tilt of the pelvis occurred and considerable upper trunk tilt with unilateral knee bending. The combination of these changes led to a less lateral shift of both segments downhill. The shifts had generally amplitudes less than those predicted from PO and KO conditions. That is the result was a more stable CoM motion. The lateral shifts were significantly less for the pelvis under the IPS condition, and less for the trunk under the CONT condition. The major shift in pelvis lateral velocity at ca. 500 ms under IPS condition presumably underlies the stabilisation of CoM velocity observed at this time point. This stability was reinforced by abducting arm movements.

The question arises why greater than predicted knee velocities, pelvis tilts and trunk tilts can lead to reduced lateral shifts of the pelvis and trunk segments and with these shifts improved CoM lateral stability, in contrast to the marked instability with bilateral voluntary knee flexion (Oude Nijhuis et al., 2007). Oude Nijhuis (2007) described a number of factors which influence the stability. Among these, the similarity of the underlying muscle synergies and the approximately equal amplitudes of predicted (PO+KO) and combined (CONT or IPS) responses were considered most important. Specifically, when the initial muscle responses for the automatic postural response were very different in comparison to APA of the voluntary response, instability resulted. Here we would argue in addition that if the tilt responses of the trunk and pelvis are in opposite directions and greater than predicted then considerable margin of safety occurs, enabling more stable control of CoM lateral shift (or AP CoM shift for trunk and pelvis flexion). The angular shifts of trunk and pelvis resulted from the unilateral voluntary knee flexion. It remains to be seen whether similar stabilising lateral shifts of the CoM would be achieved by asking subjects to voluntary laterally flex the trunk at platform tilt onset.

Integration of automatic and voluntary responses

In early studies, voluntary responses implemented with balance corrections were shown to disrupt the automatic postural responses and delay voluntary responses (Alexandrov et al., 1998, Nashner and Cordo 1981). In more recent studies, the automatic postural responses had earlier onsets when voluntary responses were executed simultaneously and in some cases could lead to increased stability (Liu et al., 2003, Grin et al., 2007, Oude Nijhuis et al., 2007). Similar findings were noted in the current study. It has been argued that successful execution of compensatory balance reactions must take into account the unpredictable body motion suddenly induced by the perturbation (Ghafouri et al., 2004; Zettel et al., 2005). When a voluntary movement is added the central neural system must take into account the destabilising effect of the voluntary movement as well and adapt the combined movement strategy to overcome the disruption of the automatic postural response. Nashner & Cordo (1981) suggested that the execution of a voluntary movement is delayed until after the automatic postural response to maintain balance. Voluntary muscle activation only occasionally preceded the automatic postural response to maintain balance if the cue for voluntary movements preceded the destabilising perturbation by ≥ 50 ms. For this reason they argued that a hierarchical relationship existed between voluntary movement and automatic postural responses. In this study we found no such hierarchy presumably because muscle response characteristics for the two actions were similar.

Generally we noted that automatic postural responses consisted of an earlier burst of activity with an onset of approximately 90 ms followed by substained activity which began to increase around 200 ms. The area under the response of the first burst in knee and trunk muscles was less than predicted with simultaneous unilateral voluntary knee flexion. In fact its response amplitude was not greater than that for the PO condition. In contrast, the amplitude for the later substained stabilising activity was as predicted. When both knees were flexed the opposite effect occurred (Oude Nijhuis et al., 2007). The early activity was equal to that predicted and the later activity was either equal to or greater than that predicted, suggesting that lack of sufficient early activity needs to be compensated by larger later activity. These differences pinpoint the advantages of unilateral voluntary knee flexion over bilateral knee flexion when performed at the onset of balance corrections and provide a theoretical basis for considering other voluntary movements as part of a stabilising strategy.

The most important aspect for a stabilising strategy appears to be a marked similarity between the timing of the early APA for the voluntary movement and that of the early balance correcting activity. A marked similarity can be noted in the muscle response profiles of Figs. 10 – 12. As the predicted responses were greater than under the combined (CONT and IPS) conditions then a non-linear summation of the early response must have occurred in which either the early APA or balance correcting response was down regulated. It remains to be investigated whether this down-regulation of efferent activity is associated with changes in CNS controlled interneural activity in the spinal cord so that later stabilising activity can be performed more accurately and with a greater margin of safety. Such a concet raises the question of how APAs are modified based on task conditions (Slijper and Latash, 2000) without apparently modifying stretch reflex gains (Vedula et al., 2008 and current results).

In conclusion, we have demonstrated in this study that voluntary increased knee flexion of the uphill knee executed on onset of a support surface tilt leads to a marked increase in lateral and AP stability compared to that for the balance perturbation alone. As

automatic flexion of the uphill knee is an important contributor to stability we presume that voluntary actions can be pre-programmed to enhance the automatic responses. A condition for such pre-programming appears to be the similarity of APAs of voluntary responses and automatic balance-correcting responses. These results have important implications for the rehabilitation of balance deficits and raises questions whether a hierarchical relationship in fact exists between voluntary and automatic postural responses.

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Conclusions

Conclusions

This research first focussed on Spinal Cerebellar Ataxia (SCA) patients as this group provide a model population for studying balance instabilities.

The current findings indicate that most instability in SCA patients is due to a failure to flex the knees adequately, and the presence of pathological trunk responses. They use the arms more than normal controls to regain mediocre stability. Questions can then be raised concerning which aspects of postural control these patients and others with balance problems should receive more attention. Clearly it helps to train the use of compensatory arm movements as SCA patients appear to naturally rely on these for stability. Attempts should also be made to "de-train" pathological trunk flexion responses to perturbations in every direction. The main cause of lateral instability in SCA patients, clearly correlated with centre of mass (COM) instability, is the lack of uphill knee flexion. Thus, the primary focus should be training knee flexion, and then reducing the fear of falling and its associated stiffness due to increased background muscle activity, because such stiffness inhibits knee flexion.

In the context of rehabilitation it is important to know if there is independent control or not of responses to roll and pitch of the support surface. Although it was demonstrated that there this interaction could be compensated by the central neural system (CNS) in the latter phases of the balance correction, there was a clear interaction between the pitch and roll motion of the trunk, knees and arms induced by pitch but not roll tilt of the support surface. This reinforced the conclusion that the CNS can program balance corrections in the pitch and roll planes independently of one another. Interestingly the form interactions took implied that roll control is programmed first and the pitch control must take into account previously occurring effects on pitch due to roll commands. In this sense pitch control is not independent of roll. For this reason it was concentrated on controlling roll motion by training voluntary motion of the knees or trunk to occur simultaneously with the automatic balance and compared these rehabilitation strategies with the effectiveness of raising the uphill arm.

The most important aspect for a stabilising strategy appears to be a marked similarity between the timing of the early anticipatory postural adjustment (APA) for the voluntary movement and that of the early balance correcting activity. As the predicted knee flexing responses were greater than the actual responses then a non-linear summation of the early response must have occurred in which either the early APA for the voluntary action or the balance correcting response was down regulated. It remains to be investigated whether this down-regulation of efferent activity is associated with changes in CNS controlled interneural activity in the spinal cord so that later stabilising activity can be performed more accurately and with a greater margin of safety. Such a concept raises the question of how APAs are modified based on task conditions without apparently modifying stretch reflex gains.

Flexing the uphill knee provided the best strategy for two reasons. Firstly the improved COM stability was in both lateral and anterior-posterior directions. Secondly, flexing the downhill knee also provided some stability whereas flexing the trunk downhill or abducting the downhill arm none.

These findings provide a positive impulse for the development of rehabilitation programs, by showing which types of voluntary movement can be productively integrated into balance corrections. Indeed, several body segments (arms, knees and trunk) have a positive effect on COM sway and can be trained to help to recover balance. Thus, an individual training program

can be composed. Further, training in two axes (anterior-posterior and medial-lateral) covers all fall directions.

Following a tilt of the support surface, the COM sways downhill, in the direction of tilt.

- The whole balance correction occurs within the first second after the perturbation starts.
- For a tilt to backward right, the COM sways back to the right.
- The most critical direction is that that induces COM motion backwards.
- The CNS reacts instantaneously to destabilizing movements and initializes a counter reaction to correct for imbalance.
- Subjects normally compensate for imbalance by raising the arms and bending the knees.
- If subjects actively or passively stiffen up their knees, the balance recovery is impaired but this can sometimes be compensated by larger ranges of movements in other body segments.
- The mass and velocity of the moving body segment and its coupling via joints to other segments are crucial for an effective recovery reaction.
- Stiffening a joint couples the mass of two segments to one of combined inertia.
- Coupling two segments increases the lever effect on neighboring segments.
- As the result of joint morphology and orientation relative to the body axes, one differentiates between roll, pitch, and mixed muscle action.
- Training of compensatory arm, knee, and trunk movements can result in stable more optimal balance corrections.
- Similarities in muscle activation are crucial for combining two motion patterns.
- Body control represents a highly complex coordination muscle patterns due to the numerous joints with many degrees of freedom involved.

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Doing a PhD thesis in the laboratory of experimental Neuro-Otology was a great challenge for me. Studying the movement of the whole human body during daily living activities is stimulating for a Human Movement Scientist. Thus, I took my chance and got this job as a scientific assistant in posturography. Working in this laboratory gave me quite big responsibilities running the studies that are described in this work.
When I started my job at Prof. Dr. John Allum's laboratory it was clear for him to give me the opportunity to do a PhD thesis. However, no PhD program for Human Movement Scientists existed at the University of Basel at that time. Despite that, I launched my challenging job as scientific assistant in posturography. About two years later Prof. Dr. Bert Müller started his Biomaterials Science Center at the University of Basel. He had the ambition to establish a PhD program in Biomedical Engineering at the Medical Faculty of the University Basel and I got the chance to attend this program right from the start in 2008. Thus, I could begin my official PhD thesis at the University of Basel based on my previous and current work under the supervision of Prof. Allum and with Prof. Müller as my mentor.

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Research in the field of human movement science means working in an interdisciplinary team. The first step is to collect data. Thus, several hours per person/of my co-workers were needed to finish a whole study protocol in which lots of sensors were placed, tape was used, but moreover, endurance was asked.
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Journal Publications

Küng UM, Horlings CGC, Honegger F, Kremer HPH, Bloem BR, Van de Warrenburg BPC, Allum JHJ (2009) Postural Instability in Cerebellar Ataxia: Correlations of Knee, Arm and Trunk Movements to COM velocity. Neuroscience 159:390-404

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