

Variability in the control of head movements in seated humans: a link with whiplash injuries?

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1. The aim of this study was to determine how context and on-line sensory information are combined to control posture in seated subjects submitted to high-jerk, passive linear accelerations. Subjects were seated with eyes closed on a servo-controlled linear sled. They were asked to relax and received brief accelerations either sideways or in the fore–aft direction. The stimuli had an abrupt onset, comparable to the jerk experienced during a minor car collision.
2. Rotation and translation of the head and body were measured using an Optotrak system. In some of the subjects, surface electromyographic (EMG) responses of selected neck and/or back muscles were recorded simultaneously. For each subject, responses were highly stereotyped from the first trial, and showed little sign of habituation or sensitisation.
3. Comparable results were obtained with sideways and fore–aft accelerations. During each impulse, the head lagged behind the trunk for several tens of milliseconds. The subjects' head movement responses were distributed as a continuum in between two extreme categories. The 'stiff' subjects showed little rotation or translation of the head relative to the trunk for the whole duration of the impulse. In contrast, the 'floppy' subjects showed a large roll or pitch of the head relative to the trunk in the direction opposite to the sled movement. This response appeared as an exaggerated 'inertial' response to the impulse.
4. Surface EMG recordings showed that most of the stiff subjects were not contracting their superficial neck or back muscles. We think they relied on bilateral contractions of their deep, axial musculature to keep the head–neck ensemble in line with the trunk during the movement.
5. About half of the floppy subjects displayed reflex activation of the neck muscles on the side opposite to the direction of acceleration, which occurred before or during the head movement and tended to exaggerate it. The other floppy subjects seemed to rely on only the passive biomechanical properties of their head–neck ensemble to compensate for the perturbation.
6. In our study, proprioception was the sole source of sensory information as long as the head did not move. We therefore presume that the EMG responses and head movements we observed were mainly triggered by the activation of stretch receptors in the hips, trunk and/or neck.
7. The visualisation of an imaginary reference in space during sideways impulses significantly reduced the head roll exhibited by floppy subjects. This suggests that the adoption by the central nervous system of an extrinsic, 'allocentric' frame of reference instead of an intrinsic, 'egocentric' one may be instrumental for the selection of the stiff strategy.
8. The response of floppy subjects appeared to be maladaptive and likely to increase the risk of whiplash injury during motor vehicle accidents. Evolution of postural control may not have taken into account the implications of passive, high-acceleration perturbations affecting seated subjects.

Bipedal stance, while obviously advantageous from an evolutionary viewpoint, is clearly more unstable than quadrupedal stance. This means that, in humans, various sensors must rapidly detect body movement and trigger fast compensatory reactions when posture is unexpectedly disturbed (Kuo *et al.* 1998; Allum & Honegger, 1998; Buchanan & Horak, 1999). Biomechanical factors play a crucial role in that process. At the onset of a passive body movement, the head remains stable with respect to space because of its high inertia (for review see Allum *et al.* 1997). The vestibular or visual afferent signals related to body motion are therefore minimal, and somesthetic input from the trunk and/or neck remains the main source of information about the stimulus for several tens of milliseconds. Indeed, several authors have shown that the initial postural response triggered by translation of the support surface is very similar in normal subjects and those with profound vestibular loss (Allum & Honegger, 1998; Runge *et al.* 1998). The early proprioceptive information is used to trigger specific patterns of muscle contractions selected from a rich repertoire of motor synergies (Nashner, 1977; Allum & Pfaltz, 1985; Horak & Nashner, 1986; Keshner *et al.* 1988; Allum & Honegger, 1998), which are aimed at adjusting the posture to compensate for the body motion and prevent subsequent falls (Henry *et al.* 1998). At some later time, the head starts to move, activating the vestibular and visual receptors and initiating vestibular and visually related modulation of the ongoing postural responses.

This scenario, shaped by evolutionary pressure, had to be rewritten when new modes of transportation became available. For instance large, passive body accelerations could be experienced in a seated, relaxed posture. While the patterns of sensory signals they generated were rather similar to those triggered in an erect posture, the motor synergies required to stabilise the body became obviously different. Despite that paradox, several laboratory studies of seated subjects submitted to abrupt, unexpected angular (Guitton *et al.* 1986; Kanaya *et al.* 1995; Keshner & Peterson, 1995; Keshner *et al.* 1995; Keshner, 2000) and linear (Viviani & Berthoz, 1975; Gresty, 1989; Forssberg & Hirshfeld, 1994) trunk accelerations have shown that our postural control system had apparently met the challenge, since adequate compensatory responses were initiated. On the other hand, the occurrence of whiplash injuries resulting from the passive body accelerations experienced in car accidents has been (Huelke *et al.* 1981) and remains a major problem (Versteegen *et al.* 1998; Richter *et al.* 1999). These injuries are still a significant threat for car passengers and often cause enduring functional deficits (Sturzenegger *et al.* 1995), not to mention huge economic cost (estimated between 5×10^9 and 10×10^9 US dollars a year in Europe; Castro *et al.* 1997). In brief, whiplash injuries raise some doubt on the seemingly unlimited capacity of the human central nervous system to adapt to our modern life style. The discrepancy between the laboratory results and the

reports of the transportation authorities remains unsolved, and numerous litigations continue to occur following car accidents (Freeman *et al.* 1999).

A particularly intriguing characteristic of whiplash injuries is that they may occur because of car accidents at low velocity (Ryan *et al.* 1993; Castro *et al.* 1997; Davis, 1998; Richter *et al.* 1999). Moreover, the accelerations experienced in these collisions are otherwise harmless in everyday life. For instance, dropping backwards into a chair is associated with peak head accelerations as high as 5.6 *g* in the horizontal plane and 8.5 *g* in the vertical plane, which are similar to those expected in low-velocity 'whiplash'-type motor vehicle accidents (Allen *et al.* 1994; Krafft *et al.* 1998). This led us to suspect that the rate of change of trunk accelerations (i.e. the jerk of the stimulus), and not the amplitude of the body or head acceleration *per se*, might be a critical factor in the explanation of why sensorimotor control sometimes fails to protect the neck. In other words, the time domain would be critical for the study of postural control, as already emphasised by several authors (Viviani & Berthoz, 1975; Guitton *et al.* 1986; Gresty, 1989; Forssberg & Hirshfeld, 1994; Kanaya *et al.* 1995; Keshner & Peterson, 1995; Keshner *et al.* 1995; Keshner, 2000).

In order to test that hypothesis, we conducted experiments where seated subjects were submitted to a brief, abrupt change of acceleration (jerk) by moving the support on which they were seated using a high-performance, custom-designed servo-controlled sled. The stimuli were short-duration, high-jerk steps of linear acceleration delivered to an unrestrained subject seated on a standard chair, and directed along the subject's interaural (sideways impulses) or naso-occipital (fore-aft impulses) axis. The three-dimensional head and trunk rotations and translations were monitored throughout the whole postural perturbation, together with the electromyographic activity of selected superficial neck muscles in about two-thirds of the cases. The activity of the superficial, paravertebral back muscles was also recorded in a few instances at either the upper thoracic or lumbar levels. To our knowledge, this study is the first description of the responses of seated human subjects to *sideways* acceleration, a condition experienced during side-on car collisions. It is noteworthy that even if the incidence of whiplash-type neck injuries is higher for rear impacts (30–40%), a substantial proportion (about 15%) of the people involved in side-on collisions also sustain neck injury (Morris & Thomas, 1996).

Following a first series of experiments, it turned out that the strategies used to stabilise the head-neck ensemble were surprisingly diverse amongst subjects. The head and body movements triggered by the high-jerk, linear acceleration were distributed in between two stereotyped extremes corresponding to two canonical types of subjects, the so-called 'stiff' and 'floppy' subjects (see Results below). For each subject, the 'stiffness' of the

responses triggered by sideways and fore–aft impulses was very similar. This led us to suspect that the mental set of the subjects, which is known to modulate postural reflexes strongly, had not been properly controlled (Guitton *et al.* 1986; Burleigh & Horak, 1996; Runge *et al.* 1998). Two questions were therefore investigated in a second series of experiments using sideways impulses. First, we hypothesised that the different motor responses displayed by stiff and floppy subjects could be linked with the use of distinct spatial frames of reference. To test that hypothesis, a representative sample of subjects was asked to mentally visualise and fixate an imaginary target during the linear impulses. Second, since voluntary control also plays a key role in head stabilisation (Guitton *et al.* 1986; Keshner & Peterson, 1995; Keshner *et al.* 1995; Keshner, 2000), some subjects were asked to perform mental arithmetic during the chair translations. If voluntary control were indeed important, a heavy mental load would be expected to have a major effect on the subjects' postural reactions. Finally, since proprioceptive information coming from the trunk appeared essential to trigger the postural responses, a last set of experiments was designed in which a representative sample of subjects was submitted to sideways impulses while the trunk movement relative to the chair was restrained using padded hip and arm restraints.

Part of these results has already been presented in abstract form (Vidal *et al.* 1999).

METHODS

Subjects

Twenty-four healthy volunteers aged between 8 and 56 years (27.3 ± 10.9 years, mean \pm s.d.) gave their written, informed consent to participate in the experiments. The height of the 11 female subjects ranged from 1.48 to 1.76 m (mean of 1.65 ± 0.09 m), while their weight ranged from 35 to 70 kg (mean of 57.5 ± 12.1 kg). The 13 male subjects were significantly taller ($P = 0.001$) with heights ranging from 1.20 to 1.90 m (mean of 1.76 ± 0.17 m); they were also significantly heavier ($P = 0.006$) with a weight range between 28 and 83 kg (mean of 69.1 ± 14.2 kg). Prior to being tested, all subjects were shown a typical sled input and asked if they wished to continue; all did. No training trials were given, and the trunk and head movements of subjects were always quantified from the first impulse. For the three minors included in the sample (aged 8, 12 and 14 years), the experiments were performed with the understanding and consent of the legal guardian. All experiments were performed according to the Declaration of Helsinki, and the Human Ethics Committee of the University of Sydney, NSW, Australia, approved all procedures used.

Procedures

Subjects were seated with eyes closed on a standard chair securely mounted to the platform of a servo-controlled, linear sled carriage. An IBM-compatible PC running the LabVIEW (National Instruments, Austin, TX, USA) program was used to drive the velocity servomotor (Baldor ASR Servotron, Fort Smith, AR, USA) and deliver brief passive, unpredictable linear accelerations (peak value of $0.65 g$, i.e. 6.4 m s^{-2}) either sideways or in the fore–aft direction. The stimuli had a sharp onset, with the sled reaching its peak acceleration in about 25 ms (peak jerk of 256 m s^{-3} , see the sled accelerometer trace

in Fig. 1). This is roughly comparable to the jerk experienced in a modest, benign car collision involving a velocity change of $1\text{--}3 \text{ km h}^{-1}$ in the first 50 ms of the impact (see data in Allen *et al.* 1994; Kullgren *et al.* 1995; Krafft *et al.* 1998). A short click gave the subjects an advanced warning of when each impulse was imminent, but the interval between this click and the impulse was automatically randomised, ranging from 0.5 to 5 s. The direction of each impulse was unpredictable (see 'Experimental conditions' below). None of the subjects ever experienced any kind of discomfort and/or pain during or after experimental sessions. For the total duration of the stimulus (3.85 s) the sled chair only moved a total of 416 mm. Subjects were instructed to sit in a natural and relaxed position on the chair, with their head approximately in line with their torso. Although the chair had arms as a safety feature, the subject's arms did not touch the chair arms during the impulse – the subject rested with their hands in their laps or on their knees.

Angular rotation and linear translation of the head and body were measured with respect to space (and with respect to each other) using Optotrak (Northern Digital, Waterloo, Ontario, Canada) sampling at 200 Hz or 400 Hz. Optotrak is a three-dimensional motion measurement system that uses three line-scan cameras to track small infrared, light-emitting diodes (LEDs) with a position resolution of 0.1 mm and an angular resolution of 0.1 deg. The position of the subject's head in space was determined by tracking three infrared LEDs positioned on a tightly fitting headset. Another LED was attached to the sled chair to track sled movement and a fifth LED was attached to a belt worn around the chest to determine position of the torso of the subject in space.

The Optotrak acquisition system was simultaneously used to acquire the signals generated by two accelerometers (see calibration details below), and in about two-thirds of the cases electromyographic (EMG) responses recorded on both sides of the neck with surface electrodes 22 mm in diameter spaced 3–4 cm apart. Surface EMG responses were only recorded from the two sternocleidomastoid muscles in about one-third of the cases. In the other cases, surface EMG responses were also obtained bilaterally from the region of the trapezius and splenius capitis muscles at the back of the neck (which means that four EMG channels were then acquired). Given the size of the EMG electrodes we used, these recordings probably picked up the activity of *both* the trapezius and splenius capitis muscles. Even if the trapezius is more involved in scapular stabilisation than in the control of head movement *per se*, the three muscles we recorded from (sternocleidomastoid, trapezius and splenius) are all activated to various degrees during voluntary head tilts both in the fore–aft direction and towards the ipsilateral side (Keshner *et al.* 1989; Mayoux-Benhamou *et al.* 1997). In a few instances, the EMG channels were used to obtain surface responses from the paravertebral back muscles at either the upper thoracic or lumbar levels.

The accelerometer signals and EMG traces were first sampled at 1000 Hz, but we mostly used a 500 Hz sampling rate that gave similar results while sparing computer memory. The EMG traces were rectified and all signals displayed off-line using the LabVIEW (National Instruments) software. For each sled impulse, the total acquisition time was 8 s; acquisition was systematically pre-triggered to begin 1 s before the onset of the sled impulse so that we could assess the background EMG activity and head and body stability prior to the onset of the sled impulse.

Calibration of the acquisition system

To validate the accuracy of the Optotrak system to measure the latency of the sled movement and subject responses, the values of latency obtained with Optotrak were compared to the latency of changes in acceleration given by high-sensitivity, broad-band linear accelerometers (Analog Devices ADXL IO 5, Norwood, MA, USA;

sensitivity of 12 volts g^{-1} , frequency range of DC to 2000 Hz). One accelerometer was positioned on the headset near the head LEDs, and a second one was positioned on the chair, near the sled LED. The first change in head position measured by the Optotrak occurred just a few milliseconds (< 10 ms) after the head-mounted accelerometer showed a change in acceleration (Fig. 2), which means that the two values were virtually identical given the sampling frequency of the Optotrak system.

On the other hand, the first change in acceleration detected by the sled-mounted accelerometer always occurred 30 ms before the onset of the sled movement, probably because of the high inertia of the sled–chair–subject ensemble (see the accelerometer trace in Fig. 1*B*). The onset of the sled impulse was therefore defined by the time at which the sled accelerometer began to show a change in acceleration (jerk-defined onset). The time at which the sled began to translate from its stationary starting position was measured using the Optotrak records of the sled-mounted LED, and the onset of the sled impulse was automatically set 30 ms before using the LabVIEW program developed to display the data. All measures of latency were calculated with respect to this jerk-defined sled impulse onset.

To normalise the amplitude of the surface EMG responses, subjects were asked to push their head in various directions against a fixed object in order to activate maximally either the sternocleidomastoid or trapezius muscles before and/or after the experimental session. Each muscle activity during linear sled impulses was then to be quantified as a percentage of the maximum EMG activity produced during this calibration procedure. However, as already pointed out in previous studies (Gresty, 1989), the results were highly unreliable because the patterns of neck muscle activation exhibited by each subject displayed huge variations over successive trials. Except for their latency, the EMG responses were therefore assessed only in qualitative terms.

Experimental conditions

For all 24 subjects, a single testing session consisted of at least 10 sideways linear sled impulses, five towards the left ear and five towards the right ear. The direction of each impulse was randomised to minimise the predictability of the stimulus. Sixteen of these subjects were also tested with at least 10 fore–aft linear sled impulses, five directed forwards and five directed backwards.

For 9 of the 24 subjects, the stimulus was systematically modified during a session of sideways impulses by restricting the possibility of trunk movement. Padded thigh–hip and biceps restraints were added to the sled chair to minimise torso movement, while still allowing natural head movements to occur.

The effects of the visualisation of an imagined target on the responses triggered by sideways impulses were evaluated using two different sets of instructions in eight subjects. We asked them to keep their eyes closed, but to visualise a target in space that either moved with the sled or remained stationary with respect to the sled during the movement.

Finally, five of the subjects were asked to undertake a distracting mental arithmetic task during a session of sideways trials (counting backwards in multiples of three from 300) to see whether this distraction modified the responses obtained in the control condition.

Quantification of the trunk and head movements elicited by the sled impulses

Six parameters were used to quantify the trunk and head movements triggered by sideways linear impulses (see Results and Fig. 1). All latency values were measured with respect to the onset of the sled impulse given by the sled accelerometer, as defined in the procedures above.

The latency of the initial trunk movement in space in the direction of the sled movement (L1 in Fig. 1) was evaluated from the signal given by the torso LED. The signals given by the head LEDs were used to compute the latency of the initial head movement in space (L2), which mainly consisted of a head roll in the direction opposite to the impulse (Fig. 1*A* and *B*, upper panel). This head roll was always associated with an initial sideways translation of the head with respect to space in the direction opposite to the impulse (Fig. 1*A* and *B*, second panel). We measured separately the latency to the peak of this head translation in the direction opposite to the sled and trunk movement (L3) and the latency to the peak of the head roll (L4). The delay between the initial movement of the sled and the beginning of the head roll (i.e. the L2 latency) gave the duration for which the subject's response was most likely triggered by proprioceptive inputs. The signals from the head LEDs were also used to quantify the amplitude of the initial head roll with respect to space (head roll amplitude or HRA, Fig. 1*B*, upper panel). The signals given by the head and trunk LEDs were finally processed to evaluate the amount of translation of the head *vs.* the trunk associated with the initial head movement (head *vs.* trunk displacement in the direction opposite to the acceleration or HVTD, Fig. 1*B*, third panel).

The same parameters were used to quantify the trunk and head responses triggered by fore–aft impulses. However, since the initial head rotation was now a head pitch, the HRA was replaced by the HPA (for head pitch amplitude).

Statistical analysis

All processing and statistical analysis of the data was conducted using either the Systat 5.2.1 for Macintosh or SPSS 9.0 for Windows software. All means throughout this paper are given with their associated standard deviation (mean \pm s.d.). Because of the small size of some samples and the obvious deviations from normal distributions observed for the HRA, HPA and HVTD values (see for instance Figs 4 and 6*B*), all statistical analyses were performed using non-parametric tests. Statistical significance was set at $P < 0.05$.

The relation between the latency and amplitude values characterising the trunk and head movements induced by sideways or fore–aft accelerations were assessed using linear regression analysis. The effects of trunk restriction, target visualisation and mental arithmetic on the postural responses triggered by sideways accelerations were all assessed on specific subsets of subjects. Similarly, only 16 subjects were tested with both sideways and fore–aft accelerations. We therefore first checked that the control data obtained for these subsets of subjects were not significantly different from those measured in our whole sample of 24 people. For each specific condition (fore–aft accelerations, trunk restriction, target visualisation and mental arithmetic) comparison with control data was then achieved for each parameter using the Wilcoxon signed-rank test for paired samples.

RESULTS

Sideways accelerations

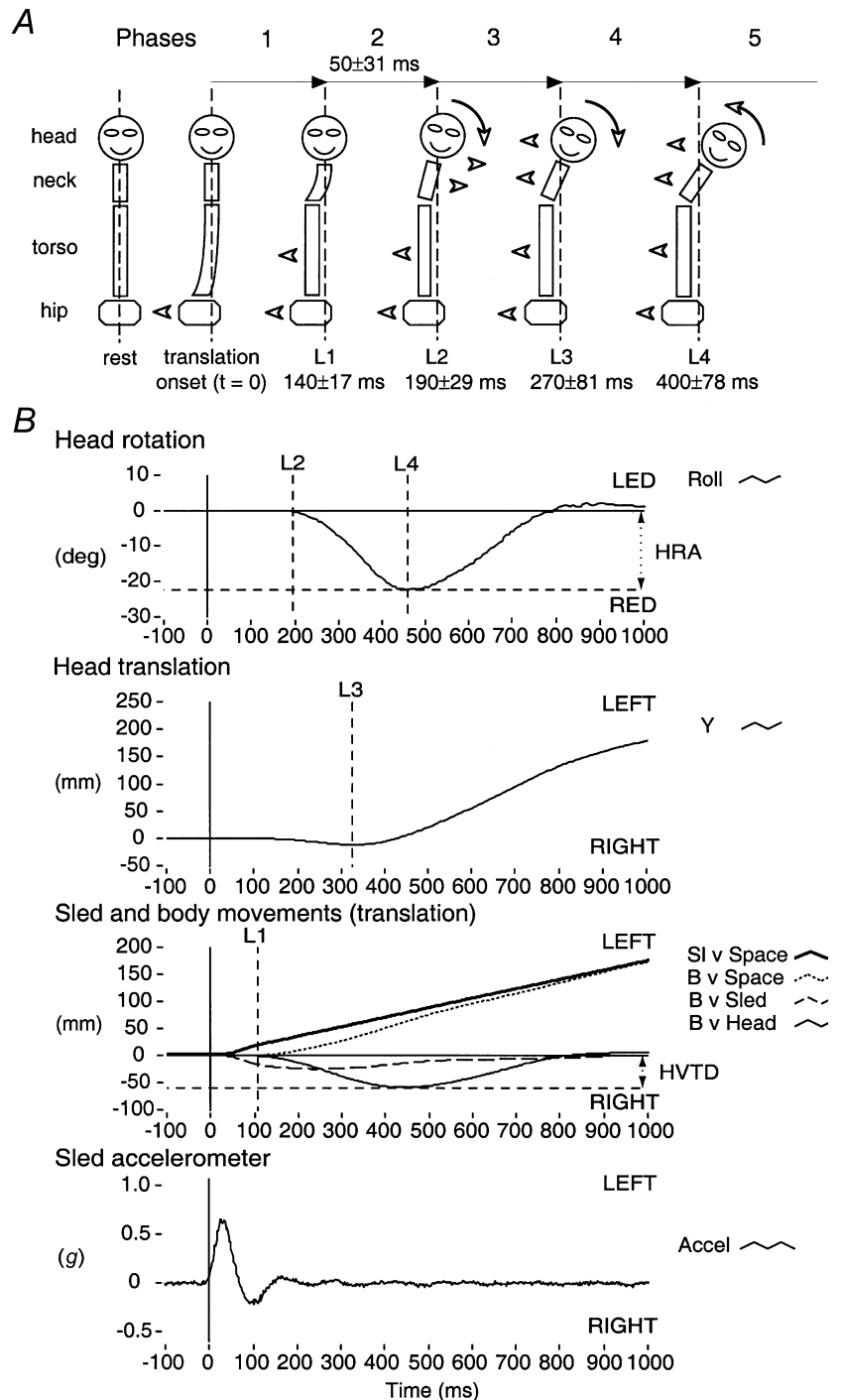
Passive, sideways linear accelerations triggered a rather stereotyped sequence of trunk and head responses in the 24 seated subjects (Fig. 1). At the onset of the sled movement, the hips of the subject moved sideways in the direction of the movement, while the upper part of the torso remained still in space (phase 1). The whole trunk only began to move in the direction of the sled displacement after 140 ± 17 ms (L1), while the head was still stationary in space (phase 2). Head movements lagged the onset of trunk motion by a delay of

50 ± 31 ms. They first included a rapid, jerk-like roll of the head in the direction opposite to the acceleration, which included both rotational and translational components (phase 3), at a mean latency of 190 ± 29 ms (L2). The translation of the head in the direction opposite to the movement of the sled reached its peak on average 80 ms later, i.e. 270 ± 81 ms after the onset of the sled movement (L3), while the head still went on rolling in the direction opposite to the sled displacement (phase 4). Finally, the head roll reached its peak amplitude on average 130 ms later at a mean latency of 400 ± 78 ms (L4), and the head smoothly came back to its normal position on the trunk (phase 5).

The amplitude of the initial head roll was highly variable between subjects (see examples in Fig. 2). Some of them, the so-called ‘stiff’ subjects, appeared to keep their head more or less in line with the torso for the whole duration of the sled movement. They only displayed very small head movements in the direction opposite to the stimulus, with average head roll amplitudes (HRA) as low as 2.9 deg and average total linear displacements of the head *vs.* the trunk (HVTD) as small as 17.1 mm over 10 trials (Fig. 2A). In contrast, the so-called ‘floppy’ subjects displayed very large head movements in the direction opposite to the stimulus. Values of HRA of 32.5 and 32.6 deg were measured in two subjects, associated with respective

Figure 1. Typical head and trunk movements triggered by sideways accelerations

A, schematic drawings showing the sequence of trunk and head movements displayed by seated subjects during sideways impulses. A full description of the sequence is given in Results. The values given for phase duration and latencies are the mean values (± S.D.) obtained for 24 subjects. L1, latency of the initial trunk movement in space; L2, latency of the initial head movement in space; L3, latency to the peak of the initial head translation in the direction opposite the sled; L4, latency to the peak of the initial head roll. B, example of the head and trunk movements triggered by a sideways impulse in a ‘floppy’ subject, as displayed by our LabVIEW program. The L1, L2, L3 and L4 latencies are shown by dashed lines. In the head rotation panel, LED and RED stand for ‘left ear down’ and ‘right ear down’ and indicate the direction of the head roll, while HRA shows the head roll amplitude. The traces showing head movements in pitch and yaw have been removed for the sake of clarity. In the head translation panel, Y corresponds to head movements along the interaural axis, and the other components of head translation have been removed for the sake of clarity. In the sled and body movement panel, HVTD shows the head *vs.* trunk displacement amplitude. Sl v Space: sled *vs.* space; B v Space: body *vs.* space; B v Sled: body *vs.* sled; B v Head: body *vs.* head; Accel: sled accelerometer.



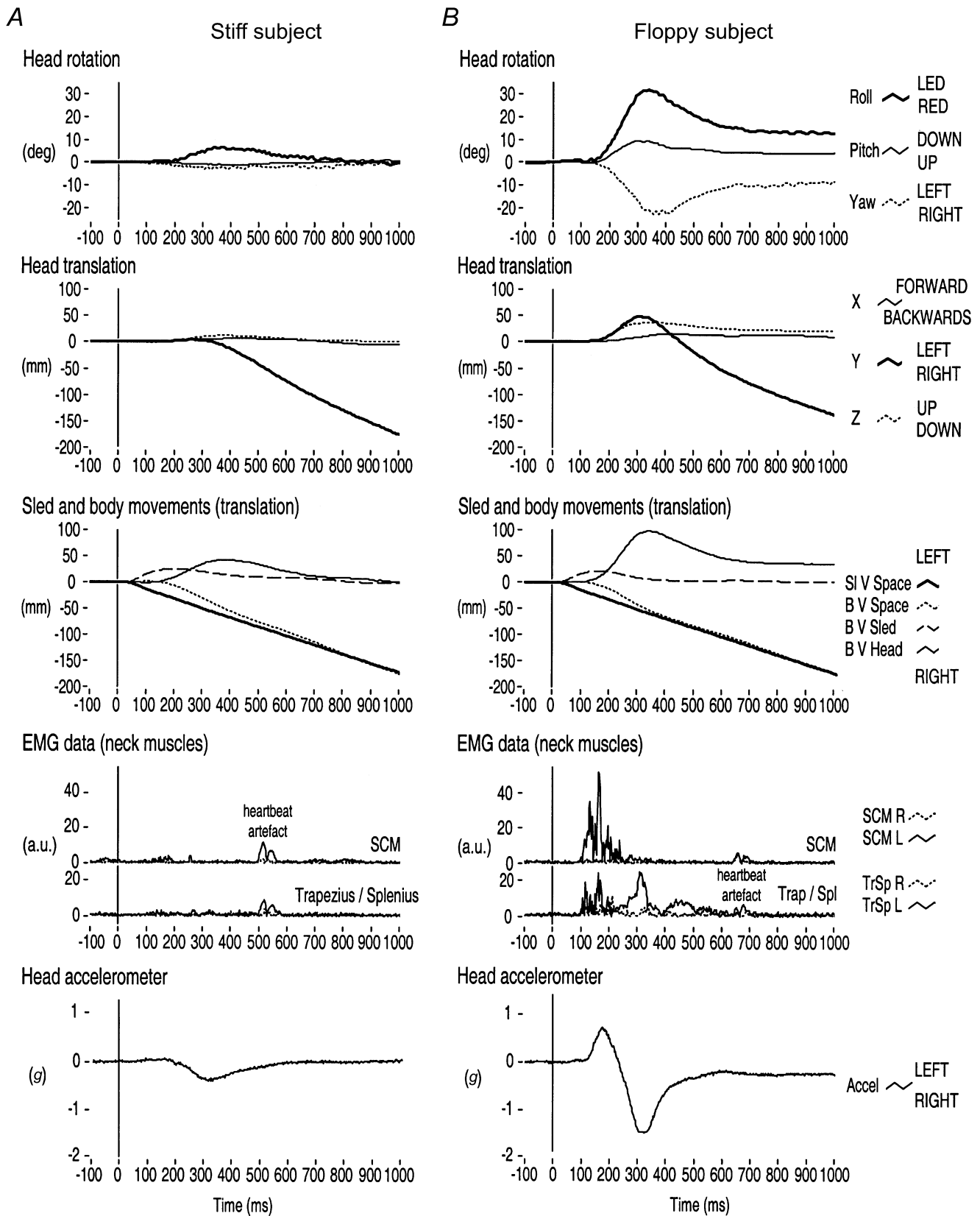


Figure 2. Comparison of the head and trunk movements triggered by sideways accelerations in a stiff subject and a floppy subject

Most of the abbreviations are as used in Fig. 1. In the head translation panels, X corresponds to naso-occipital head movements and Z to vertical head movements. The EMG data show the rectified EMG traces obtained from the left (sternocleidomastoid (SCM) L) and right (SCM R) SCM muscles, and from the

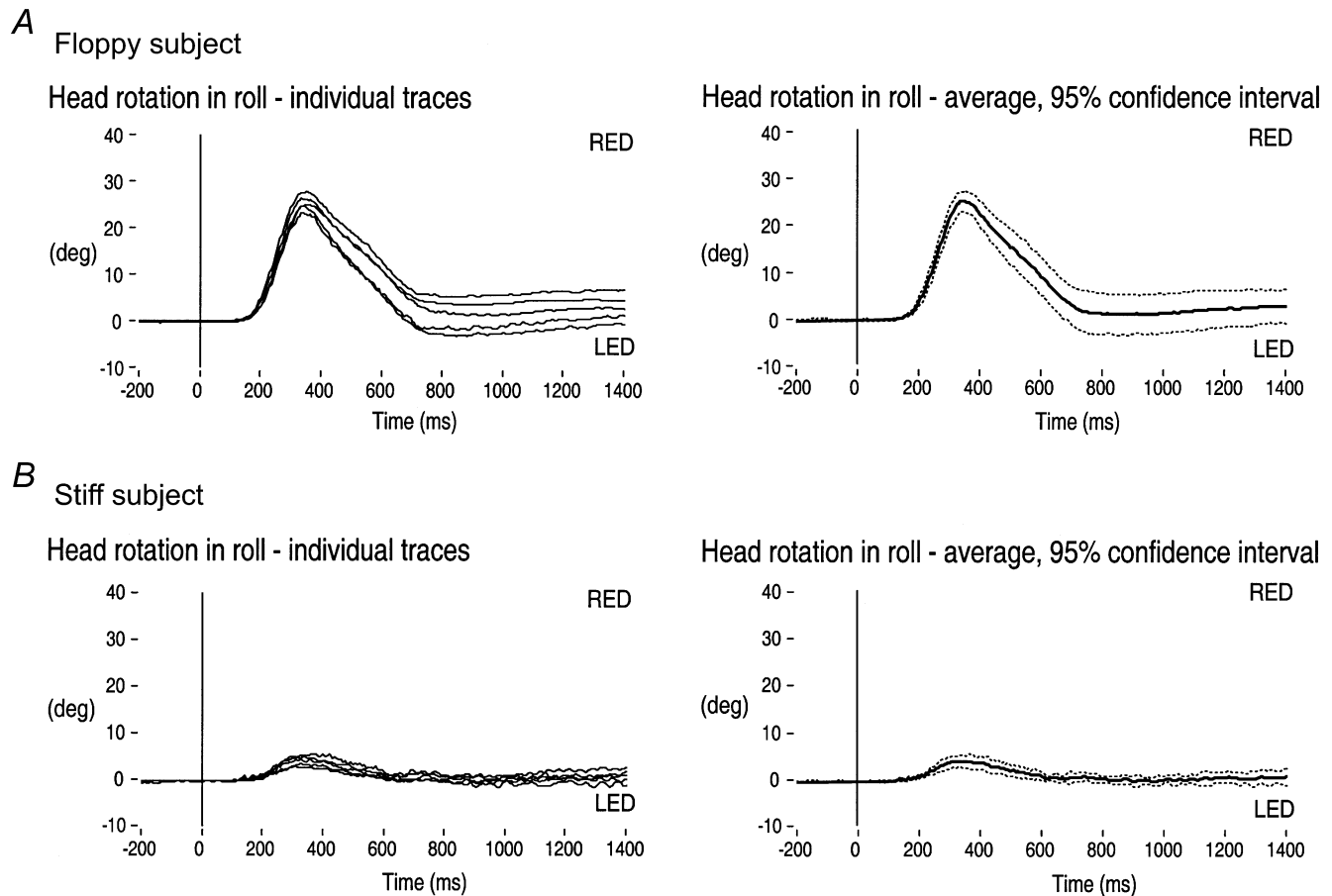


Figure 3. Example of the repeatability of the head movement responses triggered by successive sideways impulses in a floppy and a stiff subject

In each case, the 5 traces corresponding to sideways sled impulses towards the left were obtained over a single test session of 10 successive trials. Each session included 5 leftward and 5 rightward sled impulses delivered in a random order.

HVTD values of 120.5 and 133.3 mm (Fig. 2*B*). The terms ‘stiff’ and ‘floppy’ were defined before any EMG recording was undertaken, and refer only to the amplitude of the head movement observed in these two types of subjects. These terms should therefore *not* be interpreted as assumptions about the level of activation of the surface neck muscles associated with these different responses.

As pointed out in Methods, no ‘training’ trials were given, and the trunk and head movements of subjects were always quantified from the first impulse. The responses of all subjects were checked for any habituation or

learning by comparing the characteristics of the head rolls triggered by the first and last few impulses. Despite the strong inter-individual variability, the responses were highly repeatable for both the stiff and floppy people with most subjects showing very similar traces on successive trials (Fig. 3), or over different sessions of 10 trials given at intervals of several weeks.

The amplitudes of initial head movement displayed by the 24 subjects were distributed in between the stiff and floppy extremes, which were *the end-points of a continuum* rather than two discrete categories (Fig. 4). The average value of HRA for sideways accelerations

regions of the left (TrSp L) and right (TrSp R) trapezius and splenius capitis muscles (Trapezius/Splenius or Trap/Spl). The head accelerometer traces (Accel) are displayed in the lowest panels. The label a.u. stands for arbitrary units. *A*, head and trunk movements displayed by a typical stiff subject. Note the very small amplitude of the head roll and head *vs.* body displacement, and the absence of significant EMG response in either the SCM or trapezius/splenius muscles. *B*, head and trunk movements displayed by a typical floppy subject. Note the large amplitudes of the head roll and head *vs.* body displacement, and the strong EMG activation recorded before the head movement in the left SCM and trapezius/splenius muscles, on the side opposite to the direction of the sled acceleration.

across all subjects was 12.0 ± 8.6 deg for a median of 9.55 deg, while the mean HVTD reached 48.0 ± 31.3 mm for a median of 39.65 mm. Not surprisingly, these two parameters were highly correlated ($r = 0.96$, $P < 0.001$, Fig. 4). Quite logically also, the L3 latency to the peak of the initial head translation in the direction opposite the sled movement was positively correlated with the two parameters characterising the amplitude of the initial head movement in this direction ($r = 0.77$, $P < 0.001$ for the HRA and $r = 0.83$, $P < 0.001$ for the HVTD). The kinetics of the initial head roll were also highly variable, and the peak head roll latency ranged from 313 to 631 ms in the 24 subjects. Interestingly, this peak head roll latency was only loosely (but still significantly) related to the amplitude of the movement ($r = 0.43$, $P = 0.04$ vs. the HRA).

The 'stiffness' of the subjects was not significantly related to their age ($P = 0.22$ for the HRA and 0.14 for the HVTD), and the three minors did not display significantly different results from the adults. In contrast, the stiffness of the subjects did depend on gender (see Fig. 4 for the distribution of females and males along the stiff–floppy axis). The mean HVTD measured for female subjects (35.8 ± 29.8 mm, $n = 11$) was significantly lower ($P = 0.01$) than the average value obtained for male subjects (58.4 ± 29.6 mm, $n = 13$). The same trend was observed for the HRA, which was 9.1 ± 8.3 deg for female subjects vs. 14.4 ± 8.5 deg for male subjects, but the difference was not significant ($P = 0.07$) given the relatively small size of our sample. Altogether, female subjects were significantly stiffer than male subjects in response to sideways accelerations.

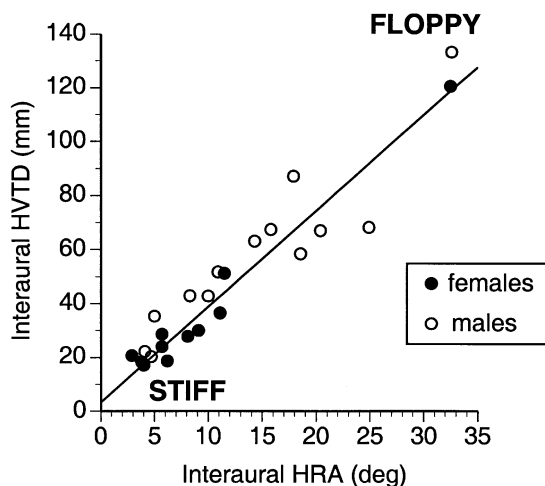


Figure 4. Variability of the head movements displayed by female and male subjects in response to sideways impulses

Note the strong correlation between the HVTD and HRA values obtained for the 24 subjects ($r = 0.96$, $P < 0.001$).

When both males and females were considered together, the stiffness of the subjects was not significantly related to their weight ($P = 0.41$ for the HRA and 0.40 for the HVTD). With the height, there was a trend for the taller subjects to be floppier, but the correlation did not reach significance ($r = 0.40$, $P = 0.054$ for the HRA and $r = 0.40$, $P = 0.052$ for the HVTD). This trend was apparently linked to the fact that the significantly stiffer females were also significantly shorter than the males, because it disappeared when each gender was considered separately (the probability of significance of the correlation with height reached 0.30 for the HRA and 0.41 for the HVTD among women, and 0.27 for the HRA and 0.26 for the HVTD in men).

There was no correlation between the amplitude of the initial head roll (HRA) and its latency ($P = 0.74$). In contrast, there was a significant, negative correlation between the latency of the initial trunk movement and the HVTD ($r = -0.44$, $P = 0.03$). The same trend was observed for the HRA, even if the correlation did not reach significance in this case ($r = -0.39$, $P = 0.06$). In other words, the most floppy people tended to display shorter latency of trunk movement along with the sled than the stiff subjects (see, for instance, Fig. 2).

As pointed out in Methods, the amplitudes of EMG responses were assessed only in qualitative terms. The neck EMG recordings performed on 15 of the 24 subjects did not reveal any specific pattern of sternocleidomastoid, trapezius and/or splenius capitis muscle activation associated with the different types of head responses. In all cases, and whatever the stiffness of the subject, the background EMG activity recorded before each impulse was very low, to the extent that the artefactual responses triggered by heartbeats and subsequent blood flow in the carotid arteries were generally clearly visible (Fig. 5A). Whatever the amplitude of their head movement, a larger proportion of the subjects (9 out of 15) did not display any systematic activation of these superficial neck muscles. In particular, the small amplitude of head vs. body movement observed in stiff subjects was in most cases clearly not due to active contractions of their sternocleidomastoid, trapezius and/or splenius capitis muscles (Fig. 2A). Only three of the seven stiffest subjects consistently exhibited EMG activation of the muscles facing the direction of acceleration at short latencies ranging from 84 to 104 ms after the onset of the sled movement, which must have actively contributed to the stabilisation of the head on the trunk. On average, these contractions led the first trunk movement by 55 ms. On the other hand, some of the floppiest subjects (3 out of 8) exhibited consistent bursts of EMG activity in muscles on the side opposite to the direction of acceleration at latencies ranging from 106 to 128 ms after the onset of the sled movement, which were triggered on average 13 ms before the first trunk movement and 61 ms before the head actually moved (Fig. 2B). These contractions,

Table 1. Mean values (\pm S.D.) of the latency and amplitude of head and body movements triggered by fore–aft impulses, and in conditions of trunk restriction, target visualisation and mental arithmetic

Specific testing condition	Age of subjects (years)		Latency of trunk movement (L1, ms)	Latency of initial head roll or pitch (L2, ms)	Peak head translation latency (L3, ms)	Peak head roll (pitch) latency (L4, ms)	HRA or HPA (deg)	HVTD (mm)
Fore–aft impulses	29.1 \pm 13.0 (<i>n</i> = 16)	Fore–aft impulses	86 \pm 14***	158 \pm 35**	250 \pm 53*	356 \pm 94*	12.5 \pm 8.7	58.3 \pm 33.7
		Sideways impulses	138 \pm 17	196 \pm 29	292 \pm 83	402 \pm 85	13.1 \pm 10.1	53.7 \pm 36.2
Trunk restriction	28.6 \pm 10.8 (<i>n</i> = 9)	Trunk restriction	108 \pm 15*	100 \pm 11**	160 \pm 30**	275 \pm 28**	13.0 \pm 4.9	39.8 \pm 9.6*
		Control values	131 \pm 18	175 \pm 26	298 \pm 107	434 \pm 85	16.1 \pm 9.7	59.3 \pm 36.6
Target visualisation								
Stiff subjects	28.0 \pm 4.4 (<i>n</i> = 3)	Target visualisation	136 \pm 28	157 \pm 22	222 \pm 53	316 \pm 23	5.3 \pm 1.2	28.0 \pm 8.3
		Control values	156 \pm 19	175 \pm 39	233 \pm 84	346 \pm 29	5.3 \pm 0.8	27.5 \pm 6.2
Floppy subjects	27.0 \pm 3.1 (<i>n</i> = 5)	Target visualisation	128 \pm 11	179 \pm 8	306 \pm 37	385 \pm 54	10.9 \pm 5.8*	52.2 \pm 16.9*
		Control values	138 \pm 14	184 \pm 16	352 \pm 102	476 \pm 139	21.3 \pm 4.9	78.1 \pm 20.8
Mental arithmetic	28.2 \pm 3.3 (<i>n</i> = 5)	Mental arithmetic	118 \pm 6*	152 \pm 30*	222 \pm 58	381 \pm 49	9.0 \pm 4.0	42.9 \pm 15.3
		Control values	143 \pm 22	184 \pm 31	305 \pm 147	423 \pm 126	11.7 \pm 9.4	54.5 \pm 38.6

Asterisks indicate the values that were significantly different from control values, or from the values obtained during sideways impulses in the case of the fore–aft impulses (**P* < 0.05, ***P* < 0.01, ****P* < 0.001).

which involved both the sternocleidomastoid and the trapezius or splenius capitis muscles, must have actively pulled the head in the direction of the inertia opposite to the sled displacement.

EMG recordings of the paravertebral back muscles were performed in two of the floppiest subjects exhibiting consistent bursts of EMG activities and one of the stiffest subjects, at the upper thoracic and lumbar levels. The background activity of these muscles was always very low, and no consistent EMG response was triggered by the sled impulses (Fig. 5*B*).

Fore–aft accelerations

Sixteen of the 24 subjects (6 females and 10 males) were submitted to fore–aft linear accelerations. The sequence of head and trunk responses triggered by fore–aft sled movements was very similar to the one obtained with sideways stimulation (Fig. 6*A* and *C*). At the onset of the sled movement, the hips of the subject moved backwards or forwards, while the upper part of the torso was still in space. The trunk then began to move in the direction of the sled displacement. Finally, head movements included

a rapid, jerk-like pitch of the head in the direction opposite to the acceleration, before the head smoothly came back to its normal position on the trunk.

The values of the different parameters characterising the head and trunk movements triggered by fore–aft accelerations were compared with the results of sideways stimulation in the same 16 subjects (Table 1). The average latency of trunk motion (L1 in Fig. 6) for fore–aft accelerations amounted to 86 \pm 14 ms, and was reduced by 38% compared with the latency of 138 \pm 17 ms measured for sideways accelerations (*P* < 0.001). Similarly, the average latency of the initial head pitch (L2) amounted to 158 \pm 35 ms and was 19% shorter than the head roll latency measured for sideways accelerations (*P* = 0.008). The delay between trunk motion and head movement (72 \pm 34 ms) was therefore not significantly different compared with sideways accelerations (58 \pm 28 ms, *P* = 0.26). The latency to the peaks of head rotation (in pitch, L4) and head translation in the direction opposite to the sled movement (L3) were also significantly decreased in comparison with sideways accelerations (*P* = 0.04 and 0.02, respectively).

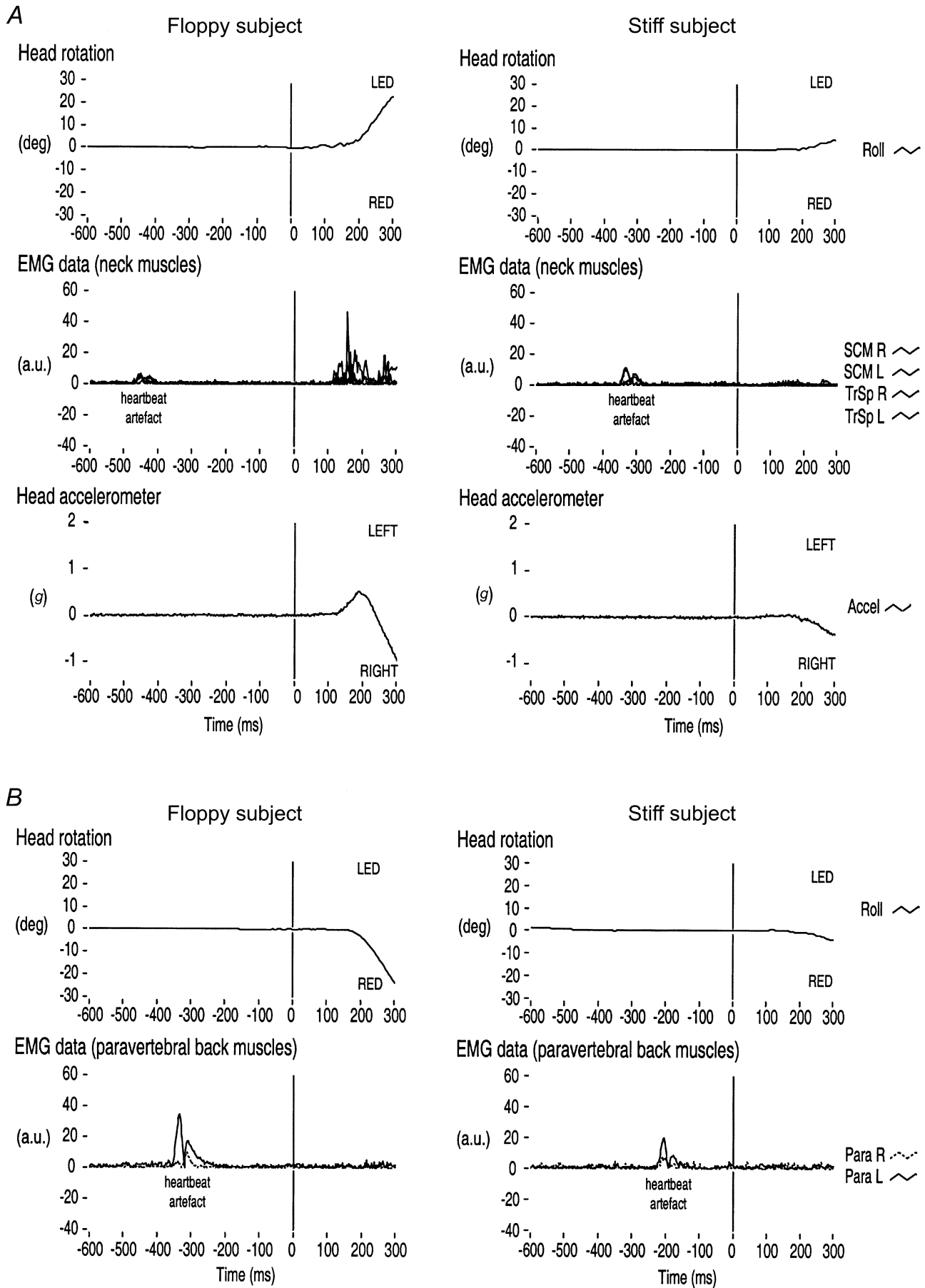


Figure 5. For legend see facing page.

Despite these latency differences, the amplitudes of the head movements triggered by both types of stimulation in the direction opposite to the acceleration were strikingly similar (Fig. 6*B*, Table 1). As for the head roll triggered by sideways stimulation, the head pitch triggered by fore–aft accelerations was highly variable between subjects. The head pitch amplitude (HPA) ranged from 3.6 to 33.5 deg and had an average value of 12.5 ± 8.7 deg (*vs.* 13.1 ± 10.1 deg for the HRA obtained with sideways accelerations), while the HVTD ranged from 28.4 to 160.4 mm for a mean value of 58.3 ± 33.7 mm (*vs.* 53.7 ± 36.2 mm during sideways accelerations). When considering the 16 subjects, there was a high positive correlation between the amplitudes of head movement triggered by fore–aft and sideways stimulation (Fig. 6*B*). In other words, the ‘stiffness’ of each subject did not depend on the direction of the acceleration to which they were submitted. Indeed, the HPA measured during fore–aft stimulation was highly related to the HRA measured for the same subject during sideways accelerations ($r = 0.83$, $P < 0.001$), while the HVTD values obtained in both situations were also very similar in most cases ($r = 0.82$, $P < 0.001$).

The amplitude of the head movements displayed by subjects in the fore–aft direction was again related neither to their age, nor to the latency of the initial head movement. The tendency for the six female subjects to be stiffer than the 10 male subjects persisted (with for instance an average HPA of 9.9 ± 9.6 deg *vs.* 14.0 ± 8.2 deg for male subjects), but was no longer significant ($P = 0.13$), probably because of the overall lower number of subjects submitted to fore–aft accelerations. As for the sideways impulses, there was no significant correlation between the stiffness of the subjects and their weight. The trend for the taller subjects to be floppier persisted ($r = 0.45$, $P = 0.07$ for the HVTD), and actually reached the level of significance for the HPA ($r = 0.50$, $P = 0.042$). Again, this trend was probably linked with the fact that stiffness was related to gender, since it disappeared when males and females were considered separately. The negative correlation between the latency of the initial trunk movement and the amplitudes of head movements observed for sideways stimulation did not persist for fore–aft stimulation.

The EMG recordings performed on eight of the 16 subjects did not reveal more consistent patterns of sternocleidomastoid, trapezius and/or splenius capitis activation than during sideways accelerations. The background EMG activity recorded before each impulse was again very low, and most of the subjects did not display any systematic activation of their superficial neck muscles. Only three of the subjects (1 of the stiffest and 2 of the floppiest) exhibited some consistent patterns of contractions at latencies ranging from 82 to 121 ms after the onset of the sled movement, which led the first trunk movement by 33–57 ms (not shown). These contractions generally occurred simultaneously on the left and right sides of the neck, but their exact functional significance in terms of head movement was difficult to assess. For the eight subjects tested, no clear correlation emerged between the muscle activation patterns triggered by sideways and fore–aft accelerations.

Effects of torso restraint on the head and trunk movements induced by sideways accelerations.

The effects of restriction of the torso *vs.* sled movement on the trunk and head movements induced by sideways accelerations were tested in a subset of nine subjects (2 females and 7 males). The data obtained in the ‘restrained’ condition were compared with those obtained for the same subjects in the normal, unrestrained situation (Table 1). The nine subjects tested were considered as representative of the whole sample, since the average control values obtained for them were not significantly different from those measured for the whole group of 24 subjects (see the details of statistical analysis in Methods). Optotrak recordings confirmed that the torso restraints did substantially reduce the amplitude of movement of the torso *vs.* the sled. While the torso moved by 20–40 mm with respect to the sled in the control situation, this displacement never exceeded 10 mm in the restricted condition.

In all nine subjects, restriction of the torso caused the head and trunk movements to occur at an earlier time. There was a large reduction of the latency of both the trunk and head movements elicited by sideways accelerations. The latency of the trunk movement was reduced by 18%, to 108 ± 15 ms *vs.* 131 ± 18 ms in control conditions

Figure 5. Background activity of the neck and paravertebral back muscles in a floppy and a stiff subject

Most of the abbreviations are as used in the previous figures. *A* shows the background EMG activity recorded from surface neck muscles just prior to a rightwards sled impulse (time 0 corresponds to the onset of the sled impulse). Note the very low level of background activity displayed by both the floppy and the stiff subject. *B* shows the EMG activity recorded from the paravertebral back muscles on both sides of the body just prior to and at the beginning of a leftwards sled impulse. Note the complete absence of background or impulse-related EMG activity in both the floppy and the stiff subject. Para R: right paravertebral muscles; Para L: left paravertebral muscles. Time 0 corresponds to the onset of the sled impulse.

($P = 0.02$, Table 1). The average latency of the head roll in the direction opposite to the acceleration was reduced by 43% at 100 ± 11 ms *vs.* 175 ± 26 ms ($P = 0.008$, Table 1). In this condition, the head began to move with respect to space *before* or at the same time as the trunk in most subjects: on average, the head movement led the trunk movement by 8 ± 14 ms. Both the peak head roll latency and the latency to the peak of the associated head translation in the direction opposite the sled were also significantly reduced ($P = 0.008$ in both cases).

In contrast, the amplitudes of initial head movement were only mildly affected by the use of trunk restraints. The mean HRA was only slightly reduced, and this

decrease was not significant ($P = 0.26$). Only the HVTD was significantly reduced by trunk restriction ($P = 0.04$), from 59.3 ± 36.6 to 39.8 ± 9.6 mm (-33%).

Effects of target visualisation on the responses elicited by sideways accelerations

The effects of the visualisation of an imagined target were tested on a representative sample of eight subjects, including one female and seven males. The data were compared with those obtained for the same eight subjects in control conditions (Fig. 7).

Two types of instructions were actually given in this condition. The subject was asked to imagine a visual

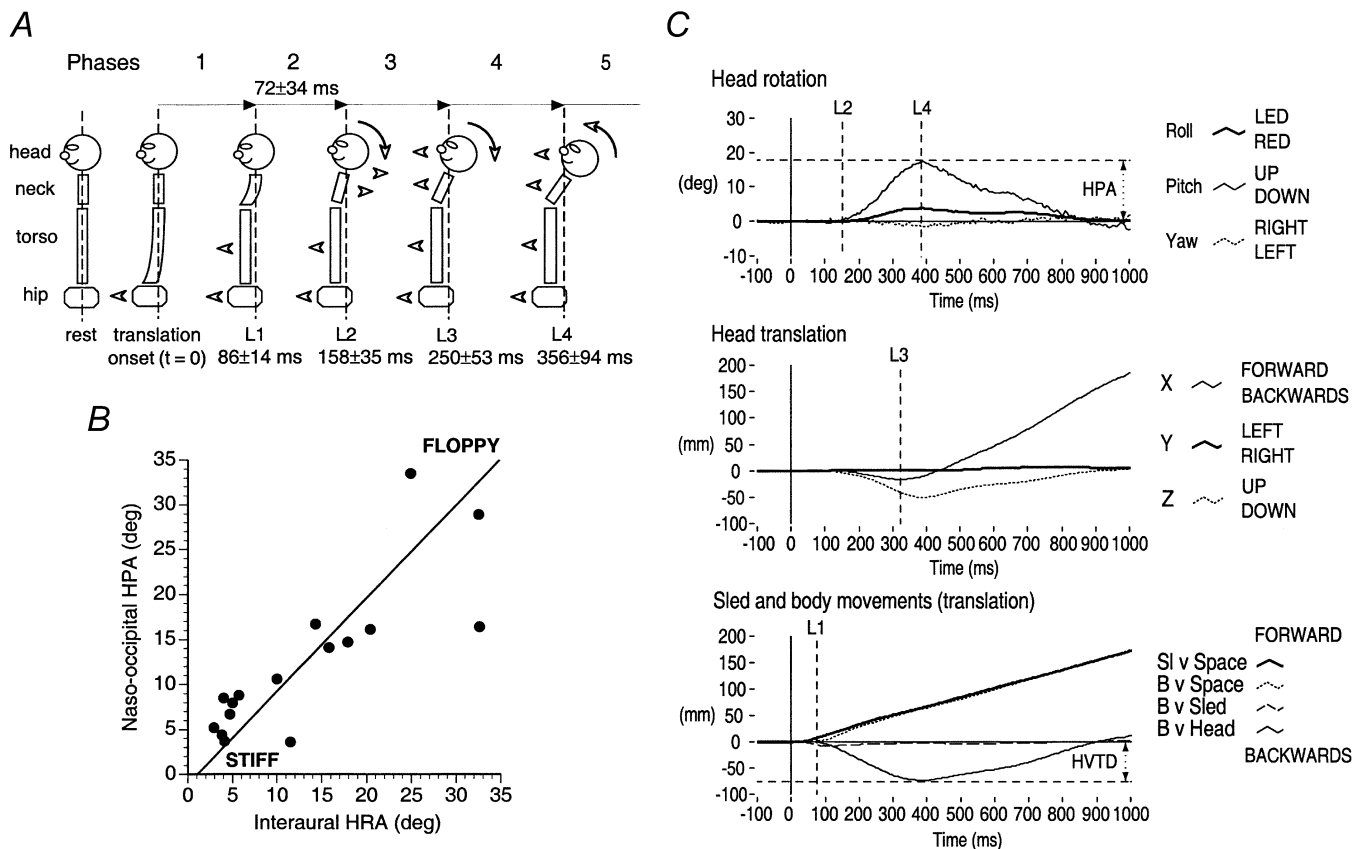


Figure 6. Head and trunk movements triggered by fore-aft accelerations

A, schematic drawings showing the sequence of trunk and head movements displayed by seated subjects during a backward impulse. A full description of this sequence is given in the Results section. The values given for phase duration and latencies are the mean values (\pm s.d.) obtained for 16 subjects. L1, latency of the initial trunk movement in space; L2, latency of the initial head translation in space; L3, latency to the peak of the initial head translation in the direction opposite the sled; L4, latency to the peak of the initial head roll. *B*, graph displaying the correlation between the mean values of HRA and HPA obtained for the 16 subjects who were tested with both sideways and fore-aft impulses. *C*, example of the head and trunk movements triggered by a fore-aft impulse in a moderately 'floppy' subject. The L1, L2, L3 and L4 latencies are shown by dashed lines. In the head rotation panel, LED and RED stand for 'left ear down' and 'right ear down' and indicate the direction of the head roll, while HPA shows the head pitch amplitude. In the head translation panel, X corresponds to head movements along the naso-occipital axis, Y to head movements along the interaural axis and Z to vertical head movements. In the sled and body movements panel, HVTD shows the head *vs.* trunk displacement amplitude. Sl *v* Space: sled *vs.* space; B *v* Space: body *vs.* space; B *v* Sled: body *vs.* sled; B *v* Head: body *vs.* head.

target either moving with him/her and the sled, or staying still in space. Since no significant difference could be detected between the effects of these two instructions, both sets of data were pooled together. In contrast to the effect of torso restraints, target visualisation did not have any significant effect on the latency values characterising the trunk and head movements elicited by sideways stimulation. Peak head roll latency did tend to be slightly reduced, but this decrease was not significant ($P = 0.09$).

On the other hand, the amplitudes of initial head movement were reduced in seven of the eight subjects. The average HRA was decreased by 42%, from 15.3 ± 9.1 to 8.8 ± 5.3 deg (Fig. 7A), while the mean HVTD dropped 27% from 59.1 ± 30.7 to 43.1 ± 18.4 mm (Fig. 7B). The amplitude of this reduction was, however, highly variable between subjects, so that the HRA reduction was only just significant ($P = 0.05$), while the HVTD only tended to be different between the control and target visualisation conditions ($P = 0.12$).

Interestingly, further analysis of the data revealed a different effect of visualisation according to the stiffness of the subjects (Fig. 7). To check whether this trend was significant, the eight subjects were divided into two groups (Table 1). The five people who displayed HRA and HVTD values larger than the medians of these parameters obtained for the main sample of 24 subjects were classified as floppy subjects, while the three others were considered as stiff subjects. As suspected, target visualisation had no effect on the small head movements triggered in stiff subjects (Table 1); their mean HRA stayed exactly the same, and their average HVTD was almost not modified. In contrast (Table 1), the average HRA of floppy subjects asked to visualise a target was halved from 21.3 ± 4.9 to 10.9 ± 5.8 deg ($P = 0.03$), while their mean HVTD was reduced by one-third from 78.1 ± 20.8 to 52.2 ± 16.9 mm ($P = 0.047$).

Effects of mental arithmetic on the responses elicited by sideways accelerations

The effects of mental arithmetic were tested on a representative sample of five subjects. The data were compared with those obtained for the same five subjects in the control condition (Table 1).

The main effect of mental arithmetic was a 15–20% reduction of the latency values for the initial trunk and head movements elicited by sideways acceleration. The latency of trunk movement went from 184 ± 31 to 152 ± 30 ms ($P = 0.04$), while the latency of the initial head roll dropped from 143 ± 22 to 118 ± 6 ms ($P = 0.04$). In contrast with what happened using trunk restraints, the average delay between the trunk and head movement was not significantly modified ($P = 0.50$). The peak head roll latency and the latency to the peak of the head translation in the direction opposite the sled both stayed in the normal range (respective P values of 0.69 and 0.07).

The amplitude of the initial head movement tended to be slightly reduced during mental arithmetic, but this decrease was not significant (Table 1).

DISCUSSION

Comparison with previous studies on postural control in seated subjects

Various studies have described the sensorimotor events underlying postural control in seated subjects submitted to passive accelerations of their support. On the sensory side, Magnusson *et al.* (1999) have shown that stretches and/or unloading of the trunk musculature signal the movement as quickly as 20 ms after the onset of chair acceleration. At that time and for several tens of milliseconds (at least 100 ms in our case), the head remains stable in space because of its inertia. Seated subjects submitted to sinusoidal translations (Gresty,

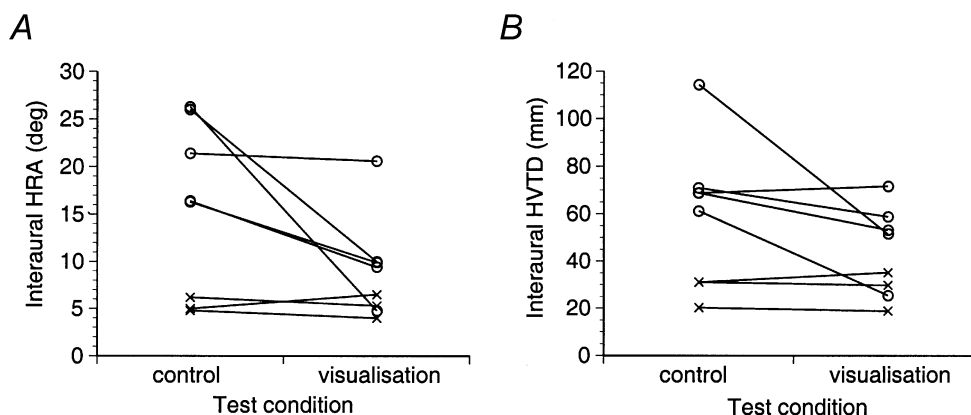


Figure 7. Effects of target visualisation on the head and trunk movements triggered by sideways impulses

The large HRA (A) and HVTD (B) displayed by floppy subjects (O) were significantly reduced by target visualisation. In contrast, the HRA and HVTD of stiff subjects (X) were not significantly modified.

1989), unpredictable tilts of the trunk (Kanaya *et al.* 1995; Keshner *et al.* 1995; Keshner, 2000) or fore–aft acceleration steps (Forssberg & Hirschfeld, 1994) all exhibited large delays between the trunk and head movements. During this period, proprioceptive signals are the main source of information available concerning the ongoing body movement. Then, the head begins to move and the vestibular afferent signals provide input to various brainstem nuclei and cortical areas in about 10 ms (see Fukushima, 1997; Wilson & Schor, 1999; Goldberg, 2000). The visual information associated with head movement becomes available 40 ms later, i.e. about 50 ms after the head begins to move (Azzena *et al.* 1978; Vidal *et al.* 1982). On the motor side, the earliest reflexes are triggered in about 60 ms at the trunk and neck level (Forssberg & Hirschfeld, 1994; Kanaya *et al.* 1995; Magnusson *et al.* 1999; Wilson & Schor, 1999), while voluntary control of the neck musculature is possible about 100 ms after the onset of a postural perturbation (Mazzini & Schiepatti, 1992). In summary, postural control in seated subjects displays two main characteristics: the subject receives sensory information from at least three distinct modalities, but in a sequential way; and both reflex and voluntary mechanisms can be used to restore the postural equilibrium.

Previous studies have shown that in response to postural perturbations, seated subjects triggered motor synergies that successfully stabilised the trunk and head in relation to space, or largely decreased the amplitude of their sway. Three main conclusions were drawn from these studies: first, the variable controlled by the CNS to ensure postural stability was the position of the centre of mass of the body in relation to its support surface. Second, mainly somatosensory signals and not vestibular input triggered the centrally generated patterns of muscle contractions that stabilised the head and trunk (see Allum *et al.* 1997). These somesthetic signals were most probably generated by the stretched (Forssberg & Hirschfeld, 1994; Ito *et al.* 1997; Allum *et al.* 1998; Magnusson *et al.* 1999) and/or unloaded neck and/or trunk muscles (Kanaya *et al.* 1995; Corna *et al.* 1996). Third, at a later stage of the postural response, the somatosensory, vestibular and visual afferent inputs were all able to modulate the centrally generated synergies. Interestingly, this scheme seems also to be valid in standing subjects (Allum & Honegger, 1998).

Variability of the postural responses triggered by high-jerk acceleration steps

In this investigation, large delays were also observed between the trunk and head movements (about 50 ms for both the sideways and fore–aft impulses). When consistent EMG activity was present in the superficial neck muscles, it was always triggered before the head moved, at latencies ranging from 80 to 130 ms after the onset of the sled movement. Therefore, our data confirm

that somesthetic signals play a key role in triggering the earliest, reflex postural synergies observed in seated subjects submitted to passive linear accelerations. In some subjects, the onset of EMG activity even led the trunk movement, which fits well with the hypothesis that the somatosensory signals generated at the pelvic level are instrumental in postural control (Forssberg & Hirschfeld, 1994; Ito *et al.* 1997; Allum & Honegger, 1998). In accordance with these ideas, the reduction of the proprioceptive signals generated at the torso level by trunk restriction significantly decreased the HVTD and tended to reduce the HRA triggered by interaural impulses.

On the other hand, major differences arose from comparisons with previous studies. First, in contrast with the Forssberg & Hirschfeld (1994) study, neither the amplitude of the head and trunk responses nor the latency of EMG activity recorded in the superficial neck muscles (when present) were found to be different according to the direction of the platform translation (sideways *vs.* fore–aft). The latency and kinetics of the head movements triggered by fore–aft impulses were, however, significantly shorter than for sideways impulses, probably because different biomechanical constraints apply.

Second, the pattern of head movement and neck EMG activities triggered by our high-jerk accelerations were very variable amongst individuals. The amplitudes of initial head movement displayed by the subjects varied more than tenfold, and were distributed along a continuum between the two extremes corresponding to stiff and floppy subjects. Despite this variability, the head, trunk and EMG responses displayed by any given individual were remarkably consistent right from the first trial and over successive test sessions.

Third, while the stiff subjects were more or less able to stabilise their head on their body during the postural perturbation, the floppy subjects clearly failed to do so. Some of the floppy subjects even exhibited patterns of neck muscle contractions which suggest that they threw their head actively in the direction opposite to the chair movement, thus accentuating the neck extension caused by the inertia of the head.

We believe that these differences from previous studies arose from the higher jerks experienced by our subjects, which mimicked those observed during low-velocity car collisions. Indeed the body accelerations experienced by our subjects were similar to, if not smaller than, those generated in these previous studies. The behaviour of the floppy subjects shows therefore that, as suggested in the Introduction, it is the jerk of the stimulus and not the amplitude of the acceleration *per se* that would be a critical factor in the occurrence of whiplash injuries at low velocity.

Stiff and floppy subjects, functional implications

Although very diverse strategies were observed among subjects, the head and trunk responses displayed by any given individual were remarkably consistent both over successive trials and over distinct test sessions performed even several weeks apart. Despite the fact that no training trials were given, each subject displayed highly stereotyped head and body movement in response to the sled impulse from the first trial, and no sign of habituation or learning was observed (Fig. 3). We interpret this as an indication that the different motor responses were selected according to deeply rooted strategies of postural control, and did not solely arise from the activation of specific, lower-level proprioceptive reflexes that could be progressively modified over time. The fact that the amount of head movement displayed by each subject did not depend on the direction of the acceleration and was similar for sideways and fore–aft impulses is also in accordance with this idea.

The variability of the subjects' responses could arise from the fact that some people were able to relax more than others while waiting for the impulse. Whatever their stiffness, however, all subjects displayed very low levels of background activity in their superficial neck muscles, and we checked on a few stiff and floppy subjects that the same was true for the paravertebral back muscles (Fig. 5). Besides, since subjects always had an advanced warning of when each impulse was imminent, we believe that all of them had a roughly similar anticipation of what was going to happen. Indeed, very similar responses to each impulse were obtained for any given subject despite the fact that the delay between the advanced warning and the onset of the impulse was randomised, and that the direction of each impulse (i.e. left or right, or frontwards or backwards) was totally unpredictable. Altogether, this makes the possibility that the variations of the subjects' responses might be due to differences in their level of anticipation of, and/or attention to the stimulus very unlikely.

During postural perturbations, the trunk and head dynamics depend on their inertial and viscoelastic properties, but should also be modulated by the ongoing activity of axial muscles. However, we did not observe any systematic correlation between the EMG recorded from the superficial neck muscles and the various types of head movements displayed by our subjects. We do not think this variability of the muscle activation patterns was related to differences in the resting posture adopted at the onset of the impulses (which has been shown to happen in cats, see Thomson *et al.* 1996). Indeed, all subjects were first instructed to sit with their head approximately in line with their torso (see Methods). Then, as stated above, the head and trunk responses displayed by each individual were remarkably consistent over 10 successive trials, despite the fact that the resting

posture resumed between each impulse varied from trial to trial. Hence, it is likely that the variable stiffness of the subjects' upper bodies was actually related to different patterns of activation of their deep, paraspinal musculature (Winters & Peles, 1990). In particular, we think that the stiff subjects might use bilateral contractions of these muscles to actively maintain their head in line with their body. Unfortunately, surface EMG electrodes were not able to pick out the activity of deep neck muscles in humans. Nevertheless, the early onset of the compensatory EMG activity recorded from the surface muscles of some stiff subjects supports that hypothesis.

Despite the fact that the role of co-contraction in postural stabilisation is unclear, increasing the stiffness of the neck makes sense for coping with potentially harmful head trajectories. Keshner (2000) has recently shown that in seated, elderly adults where the sensory information normally used to evoke appropriate, direction-specific responses to postural perturbations was probably reduced, the central nervous system tended to contract multiple neck muscles simultaneously in order to make the neck more rigid. She pointed out that while 'the clinical literature continues to focus upon co-activation of opposing musculature as a deleterious factor in joint motion, ... co-contraction should be the strategy of choice in the elderly subjects'. Our results suggest that this strategy can also be spontaneously used by younger adults and children exposed to complex patterns of sensory stimulation. In the case of car crashes, the awareness of what is going to happen clearly reduces the occurrence of neck injuries (Ryan *et al.* 1993; Sturzenegger *et al.* 1994), which suggests that anticipatory tensing of the body has a beneficial effect. In contrast, the people who are fully relaxed at the time of the collision are probably less prone to actively reduce their head movement, and suffer a higher incidence of lesions. We therefore think that the strategy adopted by the floppy subjects while seated can be considered as 'maladaptive' compared with the stiffening strategy.

We propose that the strategy adopted by the floppy subjects would have made sense if the subject had not been placed in an odd, unnatural postural context. The sensory activation patterns experienced by seated subjects submitted to high-jerk, linear accelerations of their support must be very similar to the patterns generated by active or passive thrusts exerted on the trunk while standing. In the latter case, moving the head back in the direction opposite to the acceleration is functionally meaningful because it drives back the centre of mass of the body within the subject's support polygon. However, the same strategy becomes detrimental when the movement is triggered by sudden translation of the chair in seated subjects. In other words, unusual situations from an ecological viewpoint would lead to the

selection of wrong strategies in at least part of the subjects.

Stiffness vs. floppiness: a question of frames of reference?

Ultimately, we think that seated subjects submitted to high-jerk linear accelerations adopted three broad types of strategies. (1) The stiff subjects probably relied on bilateral contractions of their axial musculature to efficiently keep the head–neck ensemble in line with the trunk for the whole duration of the movement. (2) About half of the floppy subjects, in whom no consistent activation of the superficial neck muscles was observed, seemed to rely only on the passive biomechanical properties of their head and neck to compensate for the perturbations. (3) The remaining floppy subjects displayed stretch-activated, reflex muscular synergies that tended to exaggerate the inertial head movement triggered in the direction opposite to the acceleration, and would be detrimental ('maladaptive') from a functional point of view.

We propose as a working hypothesis that the central nervous system adopts one of these various strategies according to the specific frame of reference normally used by each subject. Various authors have reported that some people used visual cues to improve their balance (thus using an 'allocentric' frame of reference), while others did not (Crémieux & Mesure, 1994; Collins & De Luca, 1995; Isableu *et al.* 1997). The subjects who do not use visual cues would rather rely on gravitational and/or 'egocentric' cues (Harm & Parker, 1993; Luyat *et al.* 1997). In our case, despite the fact that no visual input was allowed, we suspect that the stiff subjects might correspond to the subjects who rely on visual cues. By increasing the stiffness of their axial muscles, they would actively keep their head stable relative to the external space while dealing with the postural perturbation. On the other hand, the floppy subjects may favour the use of an egocentric frame of reference. Indeed, whether they just relied on the inertia of their head–neck ensemble or used stretch-activated synergies to actively enhance this passive movement, the floppy subjects always tended to bring back their head to its initial position in space.

We actually demonstrated that the visualisation of an imaginary point of reference in the external space during sideways impulses could significantly change the behavioural strategy adopted by floppy subjects. Whether this imaginary target stayed fixed in space or moved together with the sled, the large head roll normally exhibited by floppy subjects was strongly reduced. This suggests that the adoption by the central nervous system of an extrinsic, allocentric frame of reference instead of an intrinsic, egocentric one is instrumental for the selection of the 'stiff' strategy. On the other hand, the visualisation of an external point of reference did not affect the latency of the head and trunk

movements of the subjects, which suggests that the use of a given frame of reference sets the stiffness of the postural responses without interfering with their kinetics. Accordingly, no significant correlation was found between the latency of the head roll or pitch evoked by linear impulses and the stiffness of the subjects. Altogether, we presume that the kinetics of the responses obtained in each individual mainly depend on the specific biomechanical properties of their head–neck ensemble.

Automated balance corrections or voluntary control?

As pointed out above, voluntary neck muscle contractions can be triggered about 100 ms (Mazzini & Schiepatti, 1992) after an unexpected postural perturbation, which suggests that voluntary control might play an important role in preventing head movement. This was indeed the case for subjects submitted to low-jerk, low-frequency (below 1 Hz) randomised trunk movements (Guitton *et al.* 1986), whose head stabilisation was strongly impaired when a mental load was added by asking them to perform mental arithmetic. However, mental load did not affect the responses triggered by perturbations including high-frequency (high-jerk) components. In this condition, the subjects' motor reactions appear to rely predominantly on stereotyped, predefined strategies, since mental arithmetic had only a minor impact on their performance (Keshner & Peterson, 1995; Keshner *et al.* 1995; Keshner, 2000). Similarly, mental arithmetic had no impact on the amplitude of the head movements triggered by our high-jerk, linear impulses. We suggest that depending on the frame of reference adopted by each subject, distinct predefined motor synergies would be selected to compensate for the high-frequency perturbations that cannot be efficiently handled by voluntary control.

On the other hand, mental arithmetic during testing sessions induced a significant decrease in the latency of the initial trunk and head movements triggered by sideways impulses. This effect of mental load suggests that in normal conditions, subjects use voluntary control to delay the movement of their trunk along with the sled in the direction of the acceleration.

Conclusion

During car accidents, seated subjects are prone to whiplash injuries due to neck hyperextension, particularly at the level of the C6–C7 cervical vertebrae (Deng, 1989; Grauer *et al.* 1997; Kaneoka *et al.* 1999). Our results suggest that the stimulus jerk is probably a crucial factor in the occurrence of such spinal injuries. However, during boxing matches, fist impulses can generate, in a few tenths of milliseconds, accelerations of the target head as high as 50 *g*, which do not cause significant damage to the cervical column at least on a short-term basis (Atha *et al.* 1985; Schwartz *et al.* 1986). Clearly, then, jerk cannot be the only factor involved. We therefore propose that car collisions may generate ambiguous patterns of proprioceptive inputs in seated humans. These inputs

would trigger heterogeneous motor responses depending on the frame of reference each subject normally uses to control his posture. In stiff and intermediate subjects (about two-thirds of the cases), bilateral contractions of the axial musculature would limit movement of the head relative to the trunk and keep them more or less in line with each other and stable in space. The remaining population (floppy subjects) would display either a fully passive behaviour, or even trigger seemingly inappropriate muscular synergies. In the latter case, subjects apparently add the thrust of their own neck muscle contractions to head inertia in a potentially harmful combination. The fact that the awareness of what is going to happen reduces the occurrence of neck injuries in car crashes (Ryan *et al.* 1993; Sturzenegger *et al.* 1994) supports the idea that the central nervous system is able to switch strategies depending on the behavioural context. This scheme might explain why whiplash injuries can be produced by low-amplitude accelerations, and why the neck injuries observed among different passengers of the same car can be very variable (Sturzenegger *et al.* 1994; Versteegen *et al.* 1998).

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