Exp Brain Res (1998) 121:478–494

RESEARCH ARTICLE

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Interactions between vestibular and proprioceptive inputs triggering and modulating human balance-correcting responses differ across muscles

Received: 13 October 1997 / Accepted: 30 March 1998

Abstract Interactions between proprioceptive and vestibular inputs contributing to the generation of balance corrections may vary across muscles depending on the availability of sensory information at centres initiating and modulating muscle synergies, and the efficacy with which the muscle action can prevent a fall. Information which is not available from one sensory system may be obtained by switching to another. Alternatively, interactions between sensory systems and the muscle to which this interaction is targeted may be fixed during neural development and not switchable. To investigate these different concepts, balance corrections with three different sets of proprioceptive trigger signals were examined under eyes-open and eyes-closed conditions in the muscles of normal subjects and compared with those of subjects with bilateral peripheral vestibular loss. The different sets of early proprioceptive inputs were obtained by employing three combinations of support surface rotation and translation, for which ankle inputs were nulled, normal or enhanced, the knees were either locked or in flexion, and the trunk was either in flexion or extension. Three types of proprioceptive and vestibulospinal interactions were identified in muscles responses. These interactions were typified by the responses of triceps surae, quadriceps, and paraspinal muscles. The amplitudes of stretch responses at 50 ms after the onset of ankle flexion in triceps surae muscles were related to the velocity of ankle stretch. The amplitude of balance-correcting responses at 100 ms corresponded more with stretch of the biarticular gastrocnemius when the knee was re-extended at 60 ms. Absent stretch reflexes at 50 ms in triceps surae with nulled ankle inputs caused a minor, 12-ms delay in the onset of balance-correcting responses in triceps surae muscles. Vestibular loss caused no change in the amplitude of balance-correcting responses, but a negligible decrease in onset latency in triceps surae even with nulled ankle inputs. Stretch responses in quadriceps at 80 ms increased with the velocity of knee flexion but were overall lower in amplitude in vestibular loss subjects. Balance-correcting responses in quadriceps had amplitudes which were related to the directions of initial trunk movements, were still present when knee inputs were negligible and were also altered after vestibular loss. Stretch and unloading responses in paraspinals at 80 ms were consistent with the direction of initial trunk flexion and extension. Subsequent balance-correcting responses in paraspinals were delayed 20 ms in onset and altered in amplitude by vestibular loss. The changes in the amplitudes of ankle (tibialis anterior), knee (quadriceps) and trunk (paraspinal) muscle responses with vestibular loss affected the amplitudes and timing of trunk angular velocities, requiring increased stabilizing tibialis anterior, paraspinal and trapezius responses post 240 ms as these subjects attempted to remain upright. The results suggest that trunk inputs provide an ideal candidate for triggering balance corrections as these would still be present when vestibular, ankle and knee inputs are absent. The disparity between the amplitudes of stretch reflex and automatic balance-correcting responses in triceps surae and the insignificant alteration in the timing of balance-correcting responses in these muscles with nulled ankle inputs indicates that ankle inputs do not trigger balance corrections. Furthermore, modulation of balance corrections normally performed by vestibular inputs in some but not all muscles is not achieved by switching to another sensory system on vestibular loss. We postulate that a confluence of trunk and upper-leg proprioceptive input establishes the basic timing of automatic, triggered balance corrections which is then preferentially weighted by vestibular modulation in muscles that prevent falling. The organisation of balance corrections around trunk inputs portrayed here would have considerable advantage for the infant learning balance control, but forces balance control centres to rely on limited sensory information related to this most unstable body segment, the trunk, when triggering balance corrections.

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Key words Vestibulospinal reflexes · Proprioceptive reflexes · Balance control · Peripheral vestibular loss · Visual modulation of postural responses · Human

Introduction

Two concepts that have emerged from recent studies may well impact our understanding of human balance control and with it the working hypotheses concerning the functional neuroplasticity underlying the recovery processes which compensate for a balance deficit. One of these concepts addresses directly the mechanisms underlying recovery from peripheral vestibular loss. Originally it was thought that a complete unilateral peripheral vestibular deficit (PVD) is centrally compensated to yield normal vestibulo-motor responses in the long term (Galiana et al. 1984); or if recovery is not complete that ankle proprioceptive inputs can be used instead of vestibular signals to establish effective balance control (Horak et al. 1990, 1994; Fitzpatrick et al. 1994). The findings of Halmagyi et al. (1990) could well change these notions. They found that a complete, unilateral PVD yields a permanent impairment of vestibulo-ocular reflex (VOR) responses to the fast head accelerations (over $100°/s^2$) in the pitch and yaw planes (Halmagyi et al. 1990; Aw et al. 1994) typically observed during balance corrections (Allum and Honegger 1992; Allum et al. 1993). That is, a VOR response deficit to fast accelerations is not compensated centrally by a rearrangement of the synaptic efficacy of contralateral vestibular inputs to brainstem vestibular nuclei (Maioli et al. 1983) or by using other sensory inputs (Kasai and Zee 1978). This limitation in the neural plasticity possible with reorganised sensory inputs could also underlie the permanent impairment which occurs after vestibular loss for human vestibulo-spinal responses in the pitch plane (Allum et al. 1988).

Another of the concepts that may impact our understanding of balance control is the idea that the body moves in response to balance perturbations as a damped, multi-linked chain (Keshner and Allum 1990). The movements appear not to be those of an inverted pendulum swaying about the ankle joints (Nashner and McCollum 1985) or those of a two-joint system with motion only at the hips and ankles (Horak and Nashner 1986; Kuo and Zajac 1993). Consistent with the multi-link movement strategy, a set of emerging findings suggest that ankle inputs may not trigger balance corrections at all (Forssberg and Hirschfeld 1994; Allum et al. 1995; Schieppati et al. 1995). Originally it was proposed that ankle inputs first trigger responses in stretched lower-leg muscles and that this trigger signal is then transmitted in a distal-to-proximal fashion upwards to elicit a balance-correcting muscle-response synergy with onsets of 100–120 ms across a number of links (Nashner 1977; Nashner et al. 1982; Horak et al. 1990; Dietz et al. 1988). The movement of the body which resulted was attributed to be like the action of an inverted pendulum – the so-called "ankle strategy" (Horak and Nashner 1986). The first feature of this concept that was called into question concerns the distal-to-proximal triggered activation of automatic balance-correcting muscle responses. Other authors (Keshner et al. 1988; Allum et al. 1993) noted responses in the trunk and neck muscles that occurred at the same time as the proposed first triggered response at 100 ms in gastroenemius (GASTROC) (Nashner 1977; Horak and Nashner 1986). Moreover, the proposed triggered response at 100 ms in GASTROC is delayed compared with the earlier 50-ms response latency of soleus (SOL) muscles to stretch of the ankle muscles (Gottlieb and Agarwal 1979) and the next response observed at 80 ms in stretched quadriceps muscles (QUAD) (Allum et al. 1993). Thus, it appears that proprioceptive reflex systems in other than the ankle muscles could well trigger postural responses with onsets of 100–120 ms. Following this line of reasoning, a number of authors have suggested that rotation of the trunk (Forssberg and Hirschfeld 1994; Allum et al. 1995), rotation of the knee (Di Fabio et al. 1992; Allum et al. 1993) or, more distally, stretch of the intrinsic foot muscles (Schiepatti et al. 1995) could trigger postural responses. In the event that responses were first triggered at the knee and trunk and given that responses appear at a number of body segments in the 100- to 120-ms range (Allum et al. 1993; Keshner et al. 1988), it appears unlikely that the body responds as an inverted pendulum or as a two-joint (ankle-hip) structure except when the balance perturbation specifically rotated some joints into a locked position (Allum and Honegger 1992). Subsequent investigations have in fact established that the body moves as a multi-link structure during many postural perturbations, with extensive knee movements eliciting stretch of upper leg muscles and with rapid head accelerations which excite the vestibular system (Allum et al. 1993, 1997). Thus both knee, trunk and head movements could also provide a trigger signal for balance corrections.

Although the vestibular system is sensitive enough to register the supra-threshold (Benson et al. 1986, 1989, Benson and Brown 1992) head angular and linear accelerations occurring in the first 100 ms following support surface movements (Allum and Pfaltz 1985; Allum et al. 1993), evidence that vestibular inputs trigger postural responses in the leg and trunk muscles of standing humans is extremely limited. Comparisons of the postural responses in these muscles between normal subjects and those with profound vestibular loss indicate that response latencies are not altered when vestibular inputs are lacking (Allum and Pfaltz 1985; Keshner et al. 1987; Horak et al. 1990; Allum et al. 1994). For the vestibular-loss subjects, only the response amplitudes were altered, except for a few muscles such as abdominals and trapezius (TRAP) in which timing changes were also present (Allum et al. 1994). The apparent absence of vestibularly triggered responses in the presence of proprioceptive inputs contrasts with response patterns observed when subjects receive a predominantly vestibular stimulus with little proprioceptive input. Responses of 60–80 ms latency are seen in the leg muscles when subjects are suddenly dropped (Greenwood and Hopkins 1976; Lacour et al.

1978). In addition, responses of presumed otolith origin can be generated in a number of muscles with short latencies in subjects with pathologically extensive movements of the middle ear ossicles in response to sound (known as the Tullio phenomenon; see Fries et al. 1993; Colebatch et al. 1994). Such experiments illustrate the capability of the vestibular system to trigger vestibular responses in leg muscles.

The question arises whether or not vestibular triggering and modulating mechanisms operate in a different mode of control when functionally meaningful proprioceptive inputs are also present. Dietz et al. (1988) and later Horak et al. (1994) attempted to answer this question in two ways. First by imposing twice as large (see Fig. 5 of Horak et al. 1994) accelerations over the first 200 ms on the head and upper trunk alone compared with those expected during perturbations applied at the feet by translation of the support surface. Because larger accelerations applied at the head elicited much smaller responses in leg muscles compared with those elicited by support-surface translation, it was argued that fast head accelerations do not affect balance control (Dietz et al. 1988), except to generate head-stabilizing reactions (Horak et al. 1994; Shupert and Horak 1996). If, however, proprioceptive inputs at the legs must occur simultaneously with the vestibular inputs for these later inputs to fully exert their influence in leg muscles, weak responses to head displacements might be expected in leg muscles when proprioceptive inputs in the legs are weak.

A simple way to apply meaningful proprioceptive and vestibular inputs simultaneously is to translate the support surface on which the subject is standing rather than perturb the head. A combination of ankle muscle stretch and head angular and linear accelerations will occur at onset of the support-surface movement (Allum et al. 1993). Based on the theoretical model of the body as an inverted pendulum and the concept that postural responses were triggered by stretch of triceps surae muscles with ankle rotation, the apparent responsiveness of the vestibular system to postural disturbances can be tested by nulling ankle inputs during support surface translation and showing that some leg muscle responses are diminished in amplitude (Nashner 1971; Nashner et al. 1990). The evidence accumulated under these test circumstances (Nashner et al. 1982; Horak et al. 1994) has been interpreted as consistent with the hypothesis that the central nervous system (CNS) switches to using vestibular information when proprioceptive information from the lower legs is unreliable. There are, however, a number of aspects of these experiments which cast doubt on this switching concept. Firstly, stretch reflexes in both triceps surae muscles (soleus and gastrocnemius) were not monitored for a significant reduction with the "nulled" ankle input protocol. Secondly, the vestibular origin of responses after 100 ms was not examined in control experiments with vestibular loss subjects and, thirdly, changes in knee and trunk proprioceptive inputs between the normal and nulled conditions were not examined. If the extension and flexion of both the knee and trunk provide a major triggering and modulating input to balance-correcting postural responses rather then ankle inputs, subtle changes in knee and trunk rotation profiles when ankle inputs are nulled could have profound effects on the amplitudes of balance-correcting muscle responses.

Perturbation of the support surface backwards causes characteristic changes of hip and knee angles accompanied by downward linear accelerations of the head as the knees bend, together with anterior-posterior linear accelerations and rotational accelerations of the head (Allum and Honegger 1992; Allum et al. 1993). Of the two sets of proprioceptive inputs occurring at the knee and trunk, the knee proprioceptive input is earlier and involves larger velocities (Allum et al. 1993). Surprisingly little attention has been paid to the role of knee inputs in postural reactions, despite the known importance of knee muscle strength in preventing falls in the elderly (Sauvage et al. 1992) and the major rearrangement of the timing of postural responses following significant knee joint instability (Di Fabio et al. 1992). Recent investigations have, in fact, documented the importance of knee inputs in reflex control from both a biomechanical and a neurophysiological standpoint. Biomechanically, the biarticular nature of many muscles acting across the knee causes either knee or ankle rotations (or joint torques) to influence the recruitment of medial gastrocnemius motor units directly. Via neural feedback, indirect interactions between forces in a number of ankle and knee muscles occur via disynaptic (Ib afferent) inhibitory feedback (Bonasera and Nichols 1994; Meunier et al. 1994). If, as postulated above, both proprioceptive and vestibular inputs must be present in leg muscle motoneurons to establish a normal vestibular modulation of postural responses following a balance perturbation, knee muscle responses and/or ankle and trunk muscle responses may be strongly influenced by the absence of a vestibular input and therefore could, in this event, lead to unstable responses.

Given these unanswered questions on the interactions between proprioceptive and vestibular inputs contributing to balance corrections, the goals of this study were twofold. First, to investigate the metrics of reflex responses in muscles at the ankle, knee and hip joints compared with the metrics of subsequent balance corrections in the same muscles in order to determine whether proprioceptive reflex responses in knee and trunk muscles might provide suitable candidates for triggering and modulating balance corrections. Second, to determine whether the absence of vestibular inputs causes the CNS to switch to proprioceptive inputs in order to appropriately modulate balance corrections or whether modulation is simply absent with vestibular loss and balance instability then occurs. To achieve these goals, we compared the responses of normal and vestibular-loss subjects to backwards translations of the support surface with no and with large ankle rotations to determine the relative role of knee and trunk inputs when ankle and vestibular inputs were present or absent. Second, we compared these responses to those elicited by dorsi-flexion of the support surface which causes little

knee flexion to determine the effect of reduced knee or vestibular inputs on balance responses. Surprisingly both normal and vestibular-loss subjects produced adequate balance-correcting responses in leg muscles without ankle and knee inputs. In trunk muscles, balance-correcting responses were delayed following vestibular loss. This leads us to postulate that primarily trunk inputs trigger balance corrections, with secondary assistance, when present, from vestibular and knee inputs. Vestibular modulation of balance-correcting responses appeared to be preferentially directed to those muscles preventing backwards falling and when absent was not compensated by either proprioceptive or visual inputs.

Materials and methods

This study examined the effect of three different balance perturbation protocols under two different conditions (eyes open and eyes closed) on the stretch reflex, balance-correcting and stabilizing responses in muscles of two groups of subjects. One group of 15 normal subjects had no known vestibular, neurological or orthopaedic problems. Vestibular deficits were excluded on the basis of symmetrical responses to caloric irrigation of each ear at 44°C and 30°C and tests of the horizontal VOR using whole-body 20°/s^2 rotations about an earth-vertical axis. The other group of five subjects had acquired, idiopathically (generally after a viral infection), bilateral peripheral vestibular deficits (vestibular loss) as adults. The deficit of at least 5 years duration was determined by no response to caloric irrigation of each ear, and responses to whole-body accelerations steps of 80°/s^2 which were less than the 5% percentile limit of normal responses. Neurological deficits were excluded using magnetic resonance imaging of the brain. The age range of the normal subjects was 20–35 years and 32–46 years for the vestibular-loss subjects. All subjects gave their informed consent to participate in the experiments after viewing movements of the support surface.

Stimulus parameters

Subjects stood on a moving support surface which could servo-rotate about the ankle joints and translate horizontally. In response to each of the three types of balance perturbation presented, the subjects were asked to return to upright as quickly as possible. Handrails were available for the subjects to grasp if they felt in danger of falling and two laboratory assistants stood at either the side of the vestibular-loss subjects to impede a possible fall. The randomly presented stimuli consisted of either a 4° dorsiflexion rotation ("normal-ankle" input protocol, as in Fig. 2), or a simultaneous 4-cm rearward translation and a 4° dorsiflexion to yield a total of 6° of ankle dorsiflexion (enhanced-ankle input protocol, as in Fig. 3), or a simultaneous 4-cm rearward translation and a 4° plantar flexion rotation of the support surface to yield negligible ankle-angle changes (nulled-ankle angle protocol, as in Fig. 1). Stimulus durations for these support-surface movements were always 150 ms. However, each profile of ankle dorsiflexion for the enhanced- and nulled-ankle input protocols was controlled for the first 250 ms from stimulus onset by a separate microprocessor which provided an additional support-surface rotation signal. The feedback signals to this microprocessor were potentiometer signals representing the rotation of the support-surface and the rotation of the lower leg. The difference between these two signals yielded the angle of ankle dorsi-flexion. The reference command signal for the microprocessor was the average ankle-angle profile of a different group of ten prior-tested normal subjects to the enhanced-ankle input protocol, and zero ankle angle for the nulled-ankle input protocol. Microprocessor control was tapered in over the first 25 ms from stimulus onset and tapered out over 30 ms at 200 ms. Interstimulus intervals were varied randomly between 5 and 20 s. Interstimulus intervals were automatically started once the subject was again in his preferred vertical position after the support surface returned to its original pre-stimulus position. Each subject received ten stimuli of each stimulus type in a random order for a total of 30 stimuli in a series. One series was presented under eyes-open conditions, and, after a 5 to 10-min pause, a second series was presented with eyes closed. An ordering effect, though probably small (see Keshner et al. 1987), was considered unavoidable for the safety of vestibular-loss subjects.

Biomechanical and EMG recordings

Previous publications (Keshner et al. 1987; Allum et al. 1993) have detailed many of the techniques employed to record the biomechanical and electromyographic (EMG) traces shown in Figs. 1–3. The support-surface reaction forces were measured separately for each foot using four strain gauges imbedded in the support surface, one at each corner of the metal plate supporting each foot. The vertical forces measured by the pairs of strain groups under the toes and heels were added together and multiplied by the distance to the ankle joint to yield a measure of ankle torque. The ankle torques of the left and right foot were added together, low pass filtered at 5 Hz and presented to the subject on a visual display mounted at eye level 1 m away. Prior to each series of stimuli, subjects were requested to stand with knees locked and their arms by their sides in a preferred upright position. The display was then zeroed and subjects were then required to maintain the display within 1 Nm of this reset position during the interstimulus wait period. The subject's display was set inactive at the onset of each stimulus and was reactivated after 1 s of data collection. Under eyes-closed conditions, two auditory tones were used instead of a visual display to feedback pitch sway variations to the subject prior to the balance perturbation.

Angular variations of several body links were measured in the pitch plane. The shank pitch angle of right leg with respect to vertical was measured with a goniometer system whose potentiometer slider was attached to a lightweight metal rod strapped to the lower leg just below the knee, 4 cm below the lateral condyle of the tibia. The upper-leg pitch angular velocity was measured with a Watson Industries transducer (±100°/s range, 0–50 Hz bandwidth). This transducer was mounted on a 20-cm-long metal plate moulded to the curvature of the upper leg. The plate was held firmly attached to the upper leg by means of an elasticated bandage. Knee angular velocity was computed off-line from the difference of the upper- and lower-leg angular velocities after differentiating the lower-leg angle and low-pass filtering both velocity signals digitally at 25 Hz with a zero phase-shift, tenth-order Butterworth filter. Pitch angular velocity of the trunk was also measured with a Watson Industries transducer. The range of this transducer was ±300°/s. It was mounted on a metal plate strapped to the chest at the level of the caudal end of the sternum. The breast plate was supported by mouldable metal arms extending over the shoulders and held tight to the chest with straps. The signal from this transducer was also filtered off-line at 25 Hz, as described above. Head pitch angular accelerations and anterior-posterior (A-P) linear accelerations were measured from normal subjects (see inserts to Figs. 1–3), using techniques described previously (Allum and Pfaltz 1985; Keshner et al. 1987). Briefly the transducers were mounted on a tight-fitting inflatable helmet and low-pass filtered at 10 Hz off-line (the bandwidth of the angular acceleration transducer was 0–10 Hz). All biomechanical signals were sampled at 500 Hz.

Surface EMG recordings were taken from the left and right tibialis anterior (TA) and SOL muscles, and from the right GASTROC, QUAD, paraspinals (PARAS) and upper TRAP muscles, using pairs of surface electrodes placed 2.5 cm apart along the muscle belly. The EMG amplifier gains were kept constant and the pairs of electrodes and lead lengths used to record each muscle were not changed throughout the complete series of experiments. EMG recordings were band-pass filtered between 60 and 600 Hz, full-wave rectified, and low-pass filtered at 100 Hz, as recommended by Gottlieb and Agarwal (1979), prior to sampling at 1 Hz simultaneously with the biomechanical signals.

Data analysis

The analog-to-digital converted and sampled EMG and biomechanical signals of each subject were first averaged off-line after a zero-latency had been defined. The population means comprising Figs. 1–3 were computed from mean subject responses. The first 3 respones of 30 in a series were ignored to reduce the possibility of adaptation effects entering the data (see Keshner et al. 1987). The remaining 9 responses from each of 3 rotation/translational protocols were averaged together. Zero latency (the vertical line at 0 ms in Figs. 1–3) or stimulus onset was defined separately for each subject. To do this, the first inflexion in the trace of the computed velocity of ankle dorsi-flexion was used if an early dorsi-flexion rotation of the support surface occurred (Figs. 2, 3). Alternatively, if this trace could not be used, as in the case of the nulled-ankle angle protocol (Fig. 1), the first inflexion in the velocity of knee flexion was used after checking that this onset did not exceed the subject's maximum delay measured between the onset of the command signal for rearward translation and the onset of ankle dorsi-flexion when the enhanced-ankle angle protocol was used. The knee angular velocity traces in Figs. 1 and 3 indicate that this procedure yielded identical onsets for knee flexion and ankle dorsi-flexion.

The areas under traces of EMG activity were calculated for individual trials across intervals: (1) spanning early stretch reflex activity, that is, between 40 and 100 ms (for triceps surae muscles) between 80 and 120 ms (for QUAD and PARAS); (2) spanning balance-correcting responses, that is, between 100 and 200 ms (for triceps surae) or between 120 and 220 ms (for all other recorded muscles); (3) spanning the final stabilizing stage of the responses, that is, over the interval 240 to 500 ms. These integration periods were selected on the basis of previous experimental results (Allum et al. 1993, 1994) and confirmed by onsets and durations of bursts of activity in these experiments. Areas across these intervals were calculated using trapezoid integration after the area due to background activity in each single-response EMG trace was subtracted from each response area. Background activity level was set as the mean EMG level in the trial over the 100-ms period immediately prior to stimulus onset. Data from EMG response areas were combined in the same way as mean responses. First responses of an individual subject to all identical stimuli were combined, then the data were averaged across subjects in the same population.

Onsets of bursts of muscle activity were marked by the operator semi-automatically. All traces of a single muscle for one protocol were displayed to the operator simultaneously so that the repetitive occurrence of any burst of activity could be identified. These traces only displayed activity that exceeded the mean plus one standard deviation of activity during the 100 ms prior to stimulus onset. Onsets were marked if a burst of activity lasted at least 40 ms. Two types of onsets were marked: short-latency stretch reflex activity if the onset was between 35 and 90 ms, and balance-correcting activity if the onset was between 90 and 160 ms. The end of each activity burst was also marked in order to yield an estimate of burst duration.

Amplitudes of biomechanical traces were measured from the mean recordings of each subject at sample intervals 25 ms apart for upper-leg and trunk-angular velocity. The change in ankle torque was also calculated over the interval 160–260 ms, when the steepest change occurs in normals (Keshner at al 1987). After establishing that a significant population difference occurred for each stimulus protocol with a MANOVA analysis, significant differences between population means of EMG response area, onsets and biomechanical measures were tested with a *t*-test for pooled variances.

Results

The six muscles we recorded from could be divided into three groups based on the influence of vestibular loss on their stretch reflex, balance-correcting and balance-stabilizing responses. One group consisted of the triceps surae muscles, SOL and GASTROC. These muscles had responses which were generally, with one exception, uninfluenced by vestibular-loss. The exception was the response in SOL coactivated between 120 and 220 ms with the balance-correcting response in TA when the support surface was rotated toe-up (see Fig. 2). QUAD was placed in a separate group, because it was the only muscle observed with stretch reflex response amplitudes influenced by vestibular-loss. Furthermore, balance-correcting responses in QUAD showed complex pattern changes following vestibular loss. The third group of muscles comprised TA, PARAS and TRAP, which were characterized by excessive activity post 240 ms in vestibular-loss subjects, presumably in an attempt by these subjects to stabilize upright posture. Activity in this third group of muscles was preceded by balance-correcting responses which were greater or less than normal depending on the direction of initial head accelerations.

Triceps surae muscles – early stretch reflexes

As the left half of Fig. 4 shows, the amplitude of triceps surae muscles (SOL and GASTROC) in the period 40–100 ms after the onset of ankle dorsi-flexion, when early stretch-reflex evoked responses can be observed, was related to the velocity of ankle dorsi-flexion. As expected, the nulled-ankle angle protocol of Fig. 1 yielded no significant early reflex activity in triceps surae muscles. Figure 1 shows the absence of activity in the SOL and GASTROC muscles of normal and vestibular-loss subjects during this period when ankle inputs were reduced during a rearward translation of the support surface using a simultaneous servo-controlled plantar flexion.

The very small response areas measured over the stretch reflex averaging period of 40–100 ms for triceps surae were not significantly different from baseline activity prior to the stimulus. The amplitudes shown in the left half of Fig. 4 (columns labelled BT and PF) were equal to the standard deviation of baseline activity for SOL and an even lower ratio for GASTROC. The ankle inputs were thereby nulled to a bound of less then 1° over the first 250 ms with a mean velocity of ankle dorsi-flexion of 7°/s (SD 3°/s) over the first 150 ms. The lack of activity in both triceps surae muscles between 40 and 100 ms confirms the efficacy of the nulled ankle input protocol in Fig. 1. Increasing the triceps surae stretch velocity using a pure dorsi-flexion of the support surface with mean ankle-flexion velocities of 25°/s (SD 1.5°/s) and mean peak of 40°/s, over the 150 ms of stimulus duration, caused a vigorous stretch reflex response in SOL and a weaker response in GASTROC (Fig. 2). A further increase in mean dorsi-flexion velocity to 40°/s (SD 2°/s) and mean peak of 75°/s obtained by combining dorsi-flexion rotation with backward translation of the support surface (see Fig. 3) caused a further increase in SOL and GASTROC response areas between 40 and 100 ms (see also Fig. 4). The difference in SOL and GASTROC response amplitudes illustrated in Figs. 2, 3, and the left half of Fig. 4 is consistent with the work of Gottlieb and Agarwal (1979).

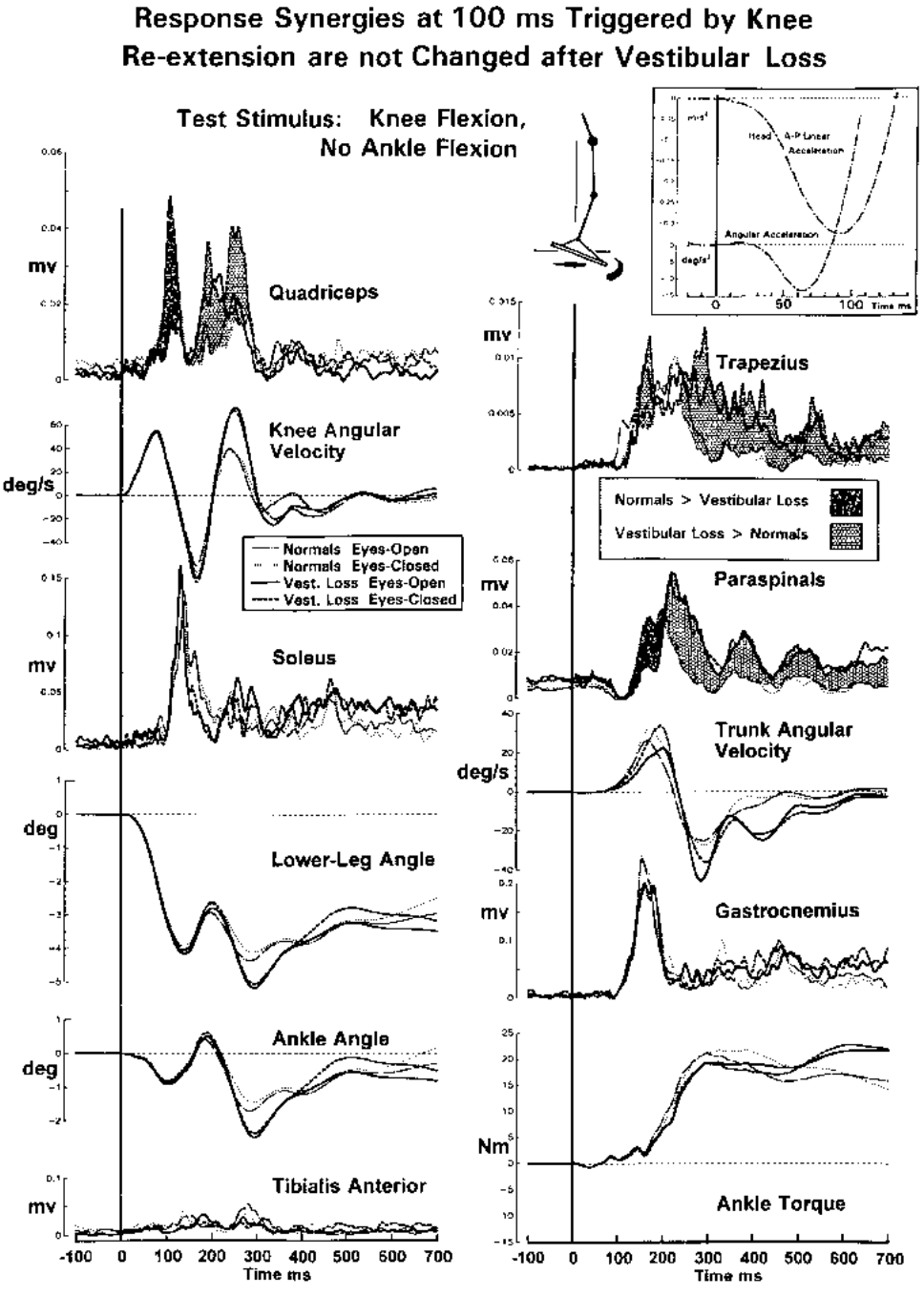

Fig. 1 Muscle activation patterns of normal and vestibular-loss subjects for a nulled ankle-input protocol using a rearward translation and a servo-controlled plantar-flexion rotation of the support surface. The traces have been aligned in time (zero latency is shown as a *thick vertical line*) according to, first, the deflection of knee angular velocity because neither ankle angular velocity nor ankle torque are useable for this purpose. Dorsi-flexion of the ankle and lower-leg angle is plotted as negative data. Knee flexion is plotted as positive data as is rearward rotation of the trunk and increased dorsi-flexion torque imposed by the ankle joint torque on the support surface. The mean population traces of 14 normal subjects are shown by *thin lines* and of five bilateral vestibular-loss subjects by *thick lines*. Each subject's contribution to the population trace was the mean of nine responses. The *insert at the top right* shows the downward head angular acceleration and forward linear acceleration of the head occurring in the first 100 ms of the perturbation. Notice the absence of stretch reflex responses in the triceps surae muscle soleus and medial gastrocnemius at 50 ms and the similarity of the balance-correcting responses in these muscles for both populations

Differences in the triceps surae stretch reflex responses were not observed between vestibular-loss and normal subjects with the possible exception that vestibular-loss subjects had SOL responses slightly larger than those of normals when knee inputs were small (see Figs. 2 and columns labelled DF in Fig. 4). This difference has been noted in previous studies (Keshner et al. 1987) and may well be related to the tendency of vestibular-loss subjects to lean further forward than normals in their preferred "upright" position, thereby activating SOL more than normal. A larger background activity in SOL can be observed in Fig. 2 prior to stimulus onset. Such an increase in baseline activity yields larger stretch reflex responses in SOL (Allum and Mauritz 1984).

Triceps surae muscles – balance-correcting responses

Figure 4 summarises the findings in triceps surae across all three perturbation conditions for the two populations of this study. Response areas are plotted for the period 40–100 ms on the left and for the area from 100 to 200 ms on the right of Fig. 4. The latter response area corresponds to balance-correcting activity (Allum et al.

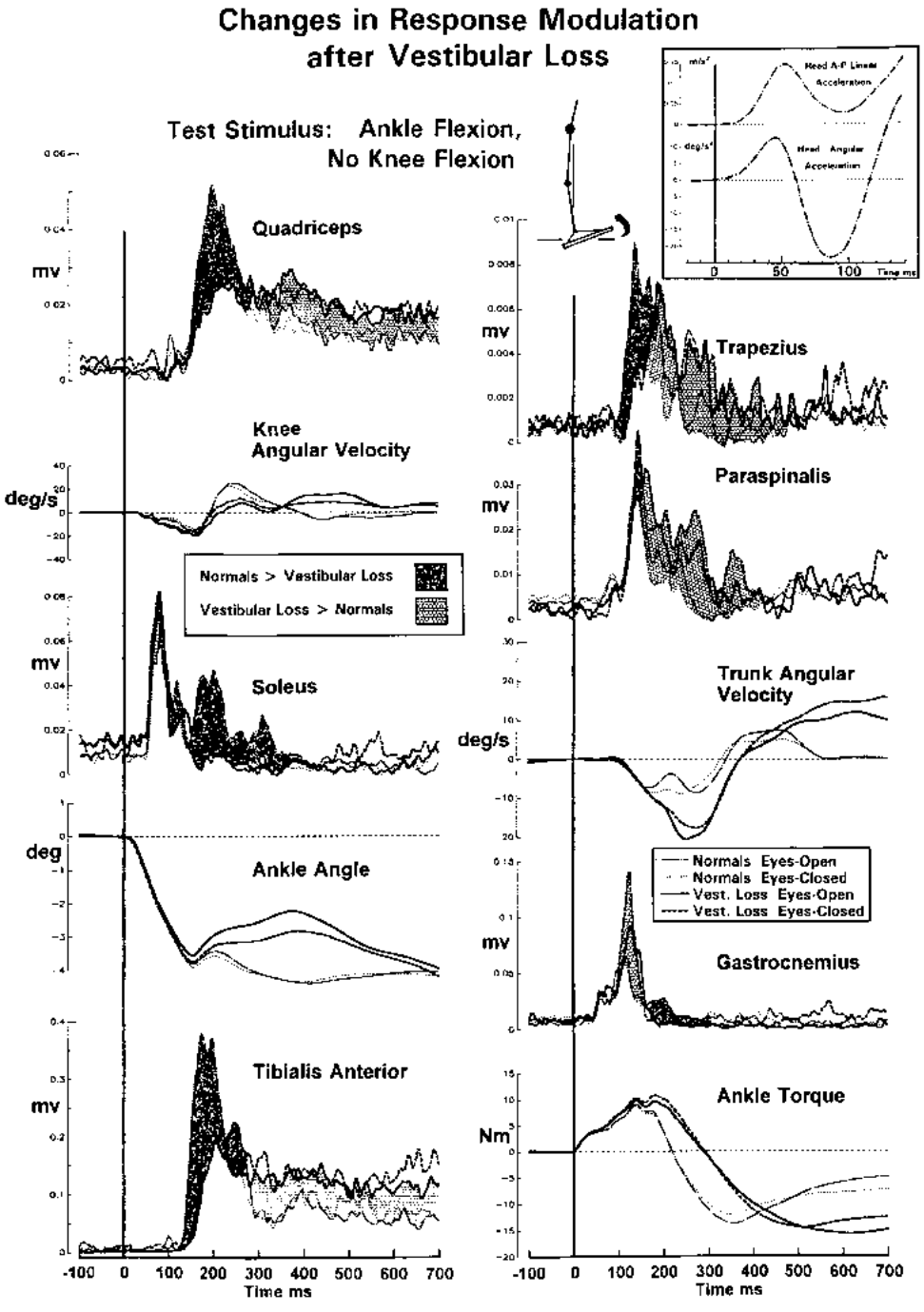

Fig. 2 Responses to dorsi-flexion of the support surface obtained from populations of normal and vestibular-loss subjects. Note the small velocities of knee flexion compared with those of Fig. 1. The traces have been aligned with the first inflexion of ankle angular velocity (*thick vertical line* at 0 ms). The *insert at the top right* shows the initial backward rotation and rearward linear acceleration of the head. Other details are described in the legend to Fig. 1

1993) and includes the second burst of SOL and GASTROC activity in Figs. 2 and 3 as well as the first large, prominent burst of SOL and GASTROC activity shown in Fig. 1. The GASTROC burst in Fig. 1 had a mean onset latency of 119 ms (SD 12 ms) for normals under eyes-closed conditions and 115 (SD 10 ms) for vestibular-loss subjects under the same conditions. By comparing amplitudes of SOL and GASTROC responses between 100 and 200 ms across the three perturbation protocols shown in Fig. 4, it is quite apparent that triceps surae activity over this period is not primarily related to the velocity of ankle dorsi-flexion. The columns in Fig. 4 are arranged from left to right in order of increasing ankle angular velocity as the numbers below the columns representing mean and peak velocities in the upper left of Fig. 4 indicate. Both the backward-translation perturbations of Figs. 1 and 3 (BT+PF and BT+DF columns in Fig. 4) produced larger response areas between 100 and 200 ms compared with the responses for dorsi-flexion in Fig. 2. As the set of numbers for peak-knee angular velocity at 175 ms below the columns in the lower right of Fig. 4 indicate, the response areas between 100 and 200 ms in triceps surae muscle were primarily related to a second stretch of triceps surae as the upper leg rotates forward. Previous studies of responses to rearward translation (Allum et al. 1993) have documented that, some 30 ms after the onset

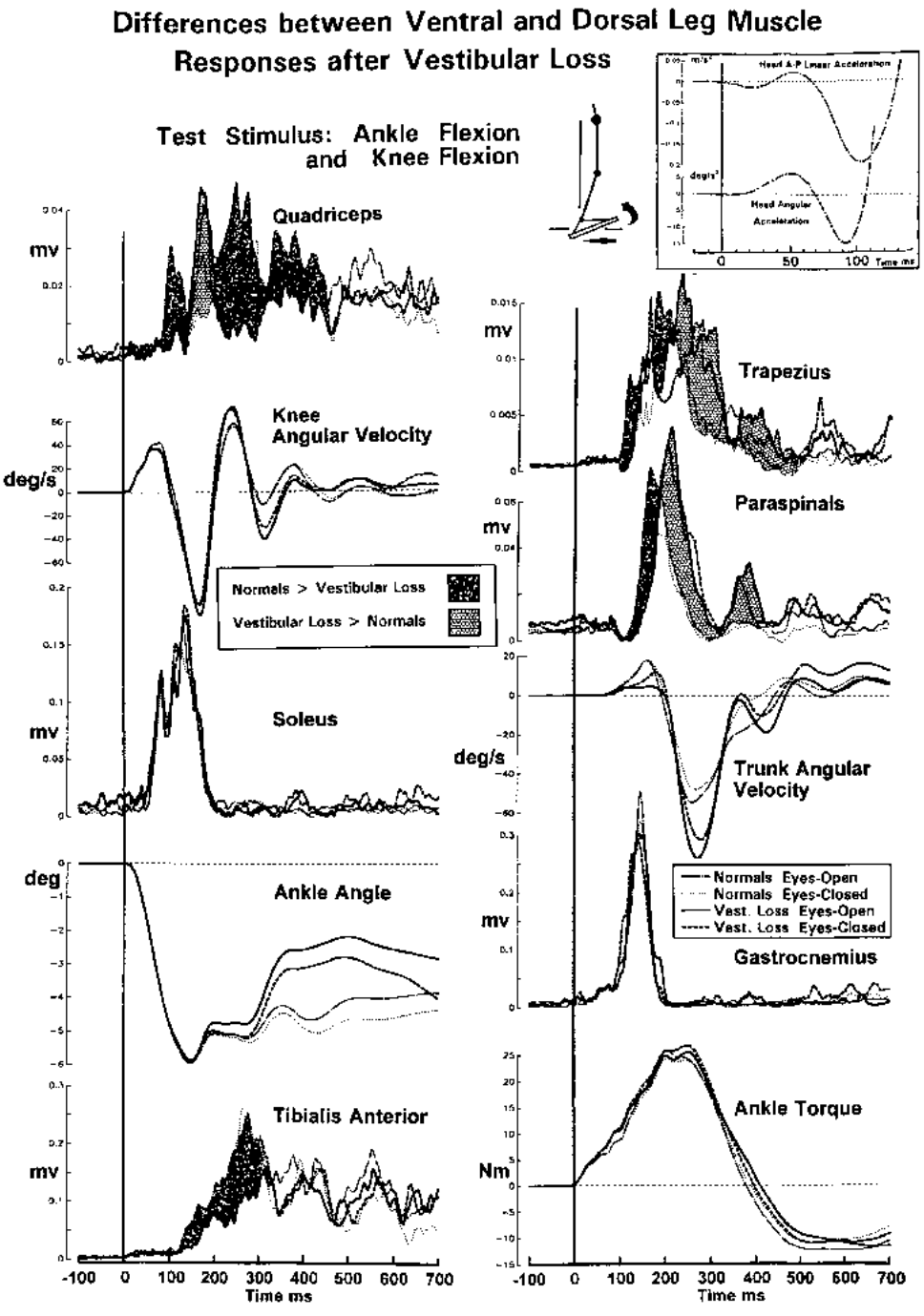

Fig. 3 EMG and biomechanical recordings of normal and vestibular-loss subjects (population means) to a combined rearward translation and dorsi-flexion of the support surface. The traces have been aligned with the first inflexion of ankle angular velocity. The *insert at the top right* shows the small head accelerations over the first 70 ms of the balance perturbation. Note the presence of stretch reflexes in soleus, gastrocnemius and quadriceps muscles and the unloading response in paraspinals all within the first 100 ms

of knee flexion (which commences with support-surface motion), the upper leg begins to pitch forward re-extending the knee. This leads to a peak-knee flexion velocity, as shown in Figs. 1 and 3, at 60 ms. Thereafter, as the traces in Figs. 1 and 3 indicate, a continuous increase in knee extension velocity occurred until a maximum was reached at ca. 175 ms. The mean onset of the 100- to 200-ms phase of GASTROC activity ranged from 100 to 120 ms (shorter onsets for faster knee extension velocities; compare Fig. 3 with Fig. 1). The first stretch reflex responses in triceps surae had a mean latency of 56 ms (SD 6 ms) for the protocol of Fig. 3. Thus it is noteworthy that three factors were consistent with the triceps surae response at 100 ms resulting from a second stretch to this group of muscles when the kneee is re-extended during backwards translation. Firstly that the difference in timing between knee re-extension (60 ms) and the timing of the response onsets at 100–120 ms is similar to expected stretch reflex latencies of 40 ms, secondly that there is continuous knee extension which would cause stretch over most of the 100- to 200-ms response duration, and third that there is a greater response amplitude with increased knee extension.

Neither the nulled ankle input condition in Fig. 1 nor the enhanced ankle input condition in Fig. 3 produced differences between normal and vestibular-loss subjects in

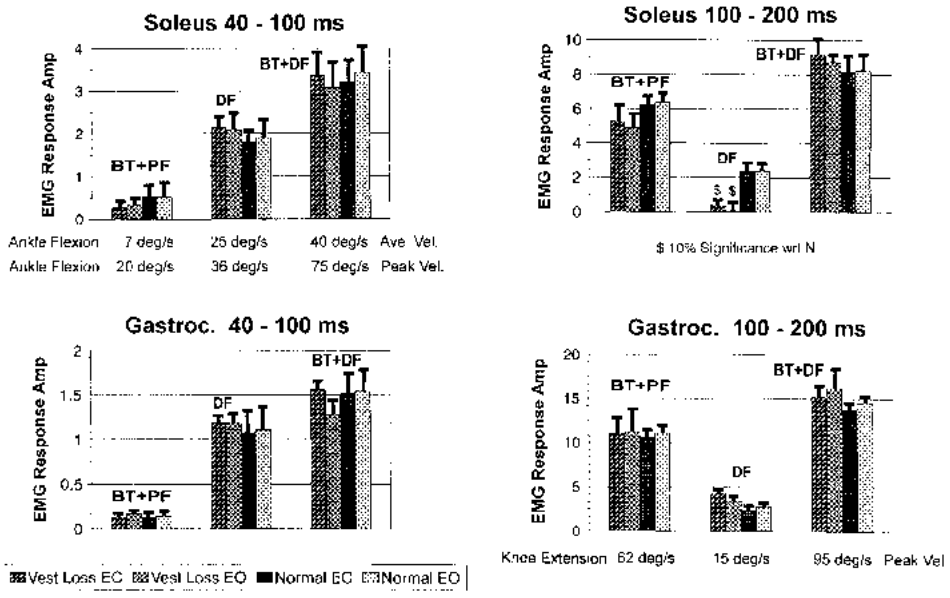

Fig. 4 Response areas of triceps surae muscles (soleus and gastrocnemius) measured over 40–100 ms and 100–200 ms from onset of the balance perturbation. The mean area in microvolt-seconds for each stimulus protocol (*BT+PF* backward translation plus plantar flexion of the support surface as depicted in Fig. 1, *DF* dorsiflexion, as in Fig. 2, *BT+DF* backward translation plus dorsiflexion, as in Fig. 3), condition (eyes open or closed),and population (normal or vestibular-loss subjects) is the height of the column**.** The *vertical bars on each column* represent the SEM. The symbols @, #, $ indicate 1%, 5% and 10% levels, respectively, of significant differences of the vestibular-loss subjects' population mean values with respect to those of normal subjects (*wrt N*) for the same stimulus protocol and condition in this and the following figures. Values for mean velocity (between onset and first zero crossing) and peak velocity of ankle flexion are listed *below the upper left* graph and peak knee extension velocities at ca. 175 ms are listed *below the lower right* graph

SOL or GASTROC responses between 100 and 200 ms (see Fig. 4, right, columns labelled BT+PF and BT+DF, respectively). If, as postulated by Nashner and co-workers (Nashner et al. 1982; Horak et al. 1990, 1994), normal subjects switched to vestibular inputs to create the responses in triceps surae muscles after 100 ms when ankle inputs are nulled a population difference should be observed in Figs. 1 and 4. Furthermore, a biomechanical difference should result from differences in triceps surae and other related muscle responses. The amplitude plots in the lower left of Fig. 6 (see columns labelled BT+PF) show only a minor decrease in ankle torque for vestibular-loss subjects at time periods when a changed triceps surae response would have affected this biomechanical variable. In fact, the only change observed was for the onsets of balance-correcting responses at 100 ms in GASTROC for vestibular-loss subjects which were some 4 ms earlier on average than those of normal subjects for the conditions of Figs. 1 and 3 for both eyes-closed and eyes-open test conditions. This change is to be expected if the vestibular-loss subjects lean forward more increasing triceps surae background activity prior to the balance perturbations. A small reduction in the amplitude of SOL responses of vestibular-loss subjects was observed as a shorter response duration for the nulled-ankle protocol. This difference was not significant (see BT+PT columns in the upper right of Fig. 4). More significant differences between normals and vestibular-loss subjects were observed in triceps surae muscles over the period 100–200 ms, when knee inputs were small (see Fig. 2 and columns labelled DF in Fig. 4). Previously this decrease in the responses of vestibular-loss subjects has been described as part of a coactivation mechanism underlying vestibular modulation of TA responses (Keshner et al. 1987; Allum et al. 1994). (This coactivation of SOL with TA is not the result of "cross-talk" between electrodes, as may be confirmed by observing the lack of possible "cross-talk" activity in SOL in Fig. 3 when TA was active.) A coactivation effect was not observed for SOL when vestibular modulation occurred in TA but knee inputs were large (see Fig. 3). Thus, the switching theory proposed by Nashner and co-workers may be applicable to triceps surae muscles when knee proprioceptive inputs rather than ankle inputs are absent.

Quadriceps muscles – early stretch reflexes

Backward translation of the support surface caused an initial flexion of the knee, which stretched the QUAD muscles. This flexion velocity was faster when the support surface was simultaneously rotated into ankle plantar flexion (compare the amplitudes of the first peak in knee angular velocity in Figs. 1 and 3 and values of mean peak velocity listed in the upper left of Fig. 5). Consistent with this increased velocity, a larger amplitude of QUAD activity between 80 and 120 ms was seen in normal subjects in Fig. 1 compared with Fig. 3 (see upper left part of Fig. 5, columns labelled BT+PF and BT+DF, respectively). In Fig. 1 there is even a small increase in QUAD activity at ca. 40 ms before the main burst of activity at

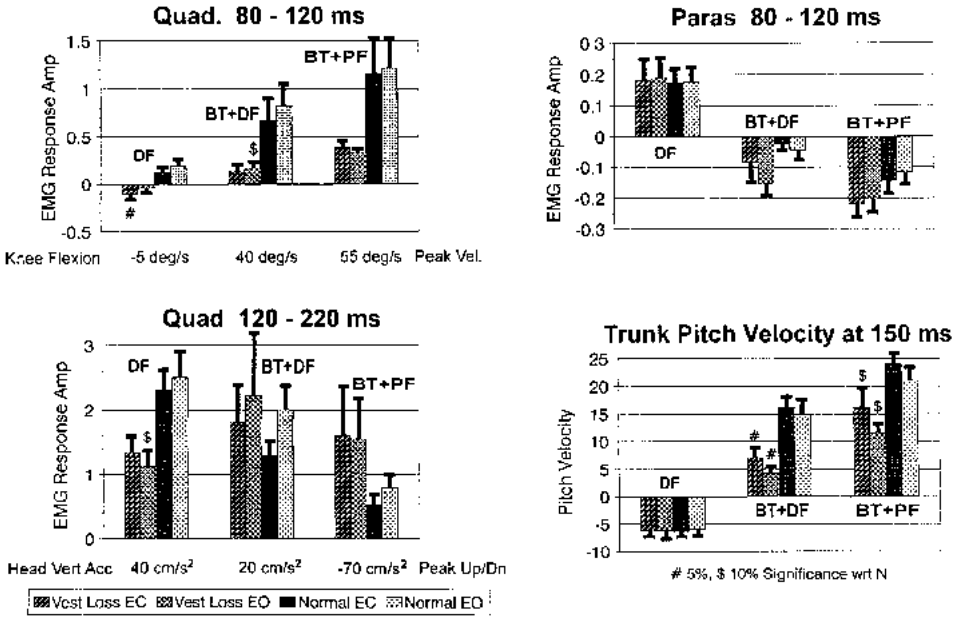

Fig. 5 Response areas of quadriceps and paraspinal muscles measured over 80–120 ms as well as quadriceps responses measured over 120–220 ms from onset of the balance perturbation. For comparison the values for the peak velocity of knee flexion are listed below the 80–120 ms quadriceps responses (*upper left*) and peak initial head vertical acceleration below the 120–220 ms quadriceps responses (*lower left*). The mean angular velocity of the trunk at 150 ms for the two populations is displayed at the *lower right* (for DF the trunk velocity is forward-pitching, for BT+PF and BT+DF, backward-pitching). Other details of the figure are described in the legend to Fig. 4

80 ms. When knee flexion is initially absent, as during pure dorsi-flexion of the support surface, then QUAD activity between 80 and 120 ms was significantly less than when early large velocities of knee flexion occurred (upper left, Fig. 5). Thus, as the upper left part of Fig. 5 shows, across the three different velocities of the protocols of Figs. 1, 2 and 3, the area of the QUAD activity between 80 and 120 ms in normal subjects was strongly related to the velocity of knee flexion. Surprisingly, the amplitude of the same response was significantly reduced in vestibular-loss subjects even though the profiles of knee angular velocity were identical to those of normals for more than the first 150 ms (see Figs. 1 and 3). Figure 5 shows that the 80- to 120-ms QUAD responses of vestibular-loss subjects also increased with knee flexion velocity but at a lower overall rate than the responses of normal subjects. Because the baseline activity in QUAD was not markedly different between vestibular-loss and normal subjects (compare in Fig. 3 the overlap of QUAD baseline activity prior to stimulus onset with the increased baseline activity in the SOL muscles of vestibular-loss subjects), this population difference in stretch reflex responsiveness cannot be ascribed to a different level of background QUAD motoneuron excitability as a result of an initial posture adopted by vestibular-loss subjects.

Quadriceps muscles – balance-correcting responses

The amplitude of the balance-correcting responses in QUAD measured over the period 120 to 220 ms differed from prior stretch-reflex responses. That is, QUAD 120–220 ms responses were not modulated in a manner consistent with the QUAD stretch reflex modulation. As the lower left part of Fig. 5 shows, for normals the largest balance-correcting responses were preceded by the smallest stretch-reflex responses (compare DF response, amplitudes in Fig. 2 and the lower left part of Fig. 5) and the smallest 120- to 220-ms responses followed the largest stretch-reflex responses (Figs. 1 and 5, left columns labelled BT+PF). The relationship of the QUAD balance-correcting responses to biomechanical variables was therefore quite complex but appeared to be related to the initial trunk velocity (see lower right part of Fig. 5) and to the direction of head accelerations as the values below the amplitude columns in the lower left part of Fig. 5 indicate. In Fig. 2 the initial trunk movement is forward-pitching and a QUAD response would correct this forward motion by pulling the upper leg forward with respect to the trunk. In Fig. 1 initial trunk motion is backward. A hamstring response (from which we did not record) rather than a QUAD response is required to correct the trunk motion. QUAD activity should consist of, as in Fig. 1 for normals, a weak co-activation. The initial trunk motion in Fig. 3 is intermediary in amplitude to that of Figs. 1 and 2. In addition to this trunk-related QUAD activity, differences between normal and vestibular-loss QUAD balance-correcting responses were consistent with the direction and amplitudes of head accelerations shown in the upper right inserts of Figs. 1, 2 and 3, and by the values of vertical linear accelerations in Fig. 5. Prior to the forward pitching of the trunk in Fig. 2, the head was accelerated backwards and upwards. Vestibular-loss responses between 120 and 220 ms in QUAD were less than those of normals (see Fig. 5, lower left columns labelled DF), suggesting that vestibular inputs associated with the head accelerations caused an increased QUAD response in normals. Conversely prior to the backwards pitching of the trunk in Fig. 1, the head accelerated forwards and downwards. Vestibular-loss responses for this perturbation were greater than those of normals, suggesting with a change in the direction of head accelerations, that vestibular inputs act to decrease the balance-correcting response of normals (see Fig. 5, lower left, columns labelled

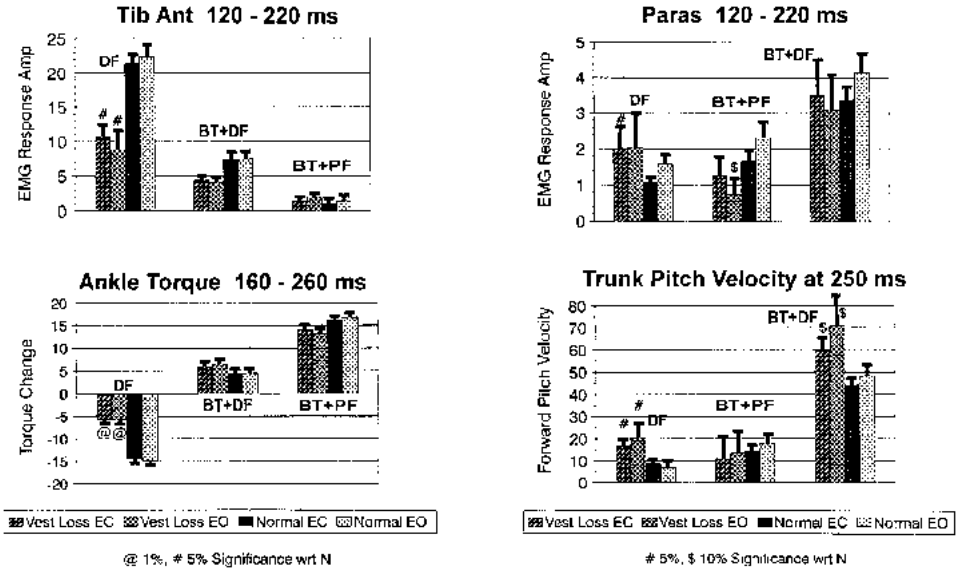

Fig. 6 Mean response areas of balance-correcting responses in tibialis anterior and paraspinal muscles (area measured over 120–220 ms) for normal and vestibular-loss subjects compared with the absolute mean trunk velocities at 250 ms (all trunk velocities at 250 ms are forward pitching) and the torque change between 160 and 260 ms. Notice on the *left* the columns are ordered based on the amplitude of the tibialis anterior and torque responses in normals, whereas on the *right* the ordering is based on the amplitude of paraspinal responses and trunk-pitching velocities in normals

BT+PF). In Fig. 3, the initial head accelerations were small compared with those of Figs. 1 and 3, and the overall response levels between 120 and 220 ms in QUAD muscles of vestibular-loss patients were not different from those of normals (see Fig. 5, lower left columns labelled BT+DF). Thus the weaker modulation of responses by head accelerations for the protocol of Fig. 3 was consistent with a similar QUAD response between 120 and 220 ms for normal and vestibular-loss subjects.

Tibialis anterior, paraspinals and trapezius

Muscles in this group were characterized by two phases of vestibularly modulated activity, one between 120 and 220 ms and the other after 240 ms, mostly without preceding stretch- reflex activity. If preceding stretch-reflex activity occurred, as in paraspinals, the amplitude of the stretch reflex activity was not altered by vestibular-loss. These muscle responses acting at the ankle, trunk and neck at 120 ms had many of the properties of preprogrammed, triggered automatic balance-corrections because response amplitudes over 120–220 ms appeared to be correlated with the velocity of the peak forward-pitching trunk velocity that followed these responses at 250 ms.

The initial trunk motion caused either a stretch or unloading response in PARAS at ca. 80 ms. The right-hand side of Fig. 5 illustrates how the amplitude of paraspinal activity between 80 and 120 ms changed with the direction of early trunk motion and the fact that the amplitude of this early muscle activity is not influenced by vestibular-loss. When the trunk extended backwards first, prior to trunk flexion,, as in Fig. 1 an unloading response was observed in PARAS. In contrast, early flexion of the trunk caused a small stretch-reflex response at ca. 80 ms as shown in Fig. 2 and the columns labelled DF in the upper right-hand part of Fig. 5. The metrics of the subsequent PARAS balance-correcting response with an onset that occurred at 120–150 ms depending on the protocol used, was, however, identical for all three conditions shown in Figs. 1, 2 and 3. The amplitude of the burst of PARAS activity measured between 120 and 220 ms increased in normal subjects with the velocity of trunk forward pitching (see Fig. 6, right, for which the columns are arranged in order of increasing trunk velocity at 250 ms in normal subjects). The amplitude and onset of this PARAS activity changed with vestibular-loss so that the relationship to trunk velocity also changed (see Fig. 6). For initial backward and upward acceleration of the head induced by dorsi-flexion of the support surface, the activity was larger in vestibular-loss subjects (Figs. 2 and 6, upper right columns labelled DF); whereas for the opposite initial direction of head movement the PARAS activity was less or delayed in the vestibular-loss subjects (see Figs. 1 and 3 and the upper right part of Fig. 6, columns labelled BT+PF).

The amplitude of initial, rearward pitching motion of the trunk (shown in the lower right part of Fig. 5) may have also influenced the differences in the amount of 120- to 220-ms PARAS activity observed between normals and vestibular-loss subjects as changes in response onsets in these two populations. Such an effect was apparent between the protocols of Figs. 1 and 3 within each of the two populations tested. Onsets of the PARAS balance-correcting response in normals were a mean 15 ms later (SD of onsets 17 ms) for the nulled-ankle-input protocol of Fig. 1, than the PARAS onsets for the protocol of Fig. 3. Figure 1 has the largest initial rearward velocity of the trunk and therefore longer unloading reflex in PARAS, whereas Fig. 5, with enhanced ankle-inputs, has the smaller initial rearward velocity of the trunk and therefore shorter unloading reflex in PARAS. The directions of initial head accelerations are also different: forward and downward rotation in Fig. 1, backward and upward rotation in Fig. 3. Notice that this 15-ms difference in PARAS onset latencies is too short to be consistent with a paraspinal response triggered by an ascending signal first triggered at the ankle joint. A 50-ms difference would be ex-

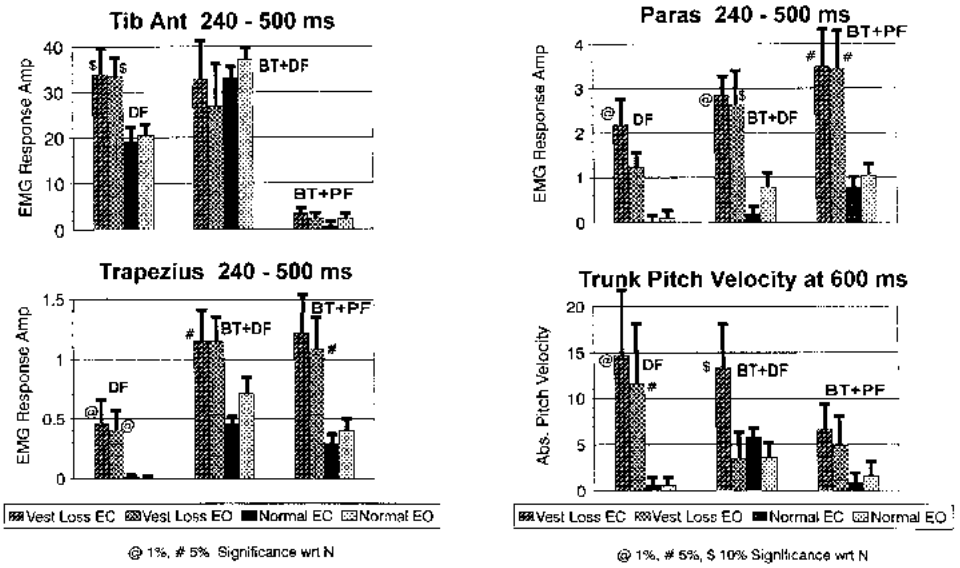

Fig. 7 Mean response areas of stabilizing responses in tibialis anterior, paraspinal and trapezius muscles (area measured over 240–500 ms) for normal and vestibular-loss subjects compared with the absolute value of mean trunk angular velocity at 600 ms. For BT+PF, trunk velocities at 600 ms were, on average, forwards pitching; for the other stimulus protocols, backwards pitching. The sets of columns are arranged from right to left in order of increasing trunk pitch velocity. Note the tendency for eyes-closed responses of vestibular-loss subjects over the 250- to 500-ms period to be greater than those of normals

pected based on the difference in SOL response latencies to initial ankle stretch. The 15-ms delay would be consistent with a triggering by knee extension because the delay of GASTROC onsets in normals for the approximately 100-ms response was 12 ms on average between the protocols of Figs. 1 and 3 (later in Fig. 3). Differences between GASTROC and PARAS latencies in normals and vestibular-loss subjects, however, do not support this knee trigger mode either.

The PARAS balance-correcting responses of vestibular-loss subjects were delayed at onset with respect to the onsets of normal subjects across all three protocols of Figs. 1–3. The greatest delay occurred for the protocol of Fig. 3 (difference in mean onsets 20 ms, with SD of 12 ms for normals and 20 ms for vestibular-loss subjects). Interestingly, the delay in onsets in PARAS responses in Fig. 3, which the dark-shaded area at the balance-correcting response onset in Fig. 3 clearly indicates, was accompanied by a change in the profile of trunk angular velocity. The initial trunk velocity of vestibular-loss subjects in Fig. 3 was significantly less than for normals (mean 7.1°/s at 150 ms compared with 16.2°/s under eyes-closed conditions; see columns labelled BT+DF in the lower right of Fig. 5) and had a longer duration of initial trunk extension (see trunk velocity traces in Fig. 3). The *delayed* PARAS responses in vestibular-loss subjects are not consistent with the notion that these responses are triggered in an ascending sequential order after the onset of balance-correcting responses in GASTROC because GASTROC responses for the same "enhanced ankle input" protocol of Fig. 3 are *earlier* in vestibular-loss subjects. In summary, the differences in PARAS activity between normal and vestibular-loss subjects over the period 120 and 220 ms appeared to be due to two effects: absent vestibular modulation and changes in onset latencies brought about by early changes in trunk velocity profiles. We cannot distinguish between these two effects because unlike knee and ankle velocities over the first 200 ms, initial trunk velocities were not servo-controlled to be identical for normal and vestibular-loss subjects. Presumably the changes in initial rearward trunk velocity result from preceding changed vestibular modulation from, for example, stretch reflexes in QUAD muscles or other leg and trunk muscles from which we did not record.

A strong signature of paraspinal responses in vestibular-loss subjects was that these were always larger than normal after 240 ms for at least a further 300 ms. Figure 7 (upper right) shows that these differences were significant across all three protocols regardless of whether the trunk of these subjects was pitching more forwards or backwards at 600 ms than the trunk of normal subjects (see lower right of Fig. 7). Thus it appeared that vestibular-loss subjects had excessive activity after 240 ms for two reasons. Firstly, trunk velocity at 250 ms was larger than normal (see lower right of Fig. 6) and therefore more paraspinal activity was required to right the trunk. Secondly, the metrics (amplitude and timing) of this righting activity were still insufficient to stabilize the trunk by 400–500 ms as observed in normal subjects (see trunk velocity traces in Figs. 1, 2 and 3). Vestibular-loss trunk-velocity profiles oscillated more and only approached zero-velocities, for the protocol of Fig. 1 i.e. a new stable posture, at 600 ms. Otherwise, as in Figs. 2 and 3, velocity was non-zero and, in the case of eyes-closed responses, continuing to increase at 600 ms. To avoid a fall under eyes-closed conditions, vestibular-loss subjects had to be supported for the protocols of Figs. 2 and 3.

TRAP responses had phases of activity modulated by vestibular-loss which paralleled those of PARAS. The main burst of activity at 120 ms also had characteristics of an automatic balance correction, because it occurred prior to the flexion of the trunk forward and its action appeared to be a preprogrammed response to extend the head countering the effect of trunk flexion. As Figs. 1–3 and the lower left part of Fig. 7 show, after 240 ms the TRAP activity was greater than normal in vestibular-loss subjects.

TA responses were also similar to those of PARAS and TRAP, showing characteristics with a distinct change in

modulation pattern with vestibular-loss. One characteristic of TA responses was dissimilar from those of PARAS and TRAP. When ankle inputs were nulled, as in Fig. 1, TA responses were practically absent (see columns labelled BT+PF in Figs. 6 and 7) in both normal and vestibular-loss subjects. The presence of knee, trunk and vestibular inputs for the nulled-ankle protocol of Fig. 1 coupled with the absent TA response suggests a leading role of ankle inputs in triggering TA responses. This was the only muscle we recorded from that responded in this manner to nulling ankle inputs. Otherwise, the responses of TA illustrated response patterns similar to those of PARAS. Activity between 120 and 220 ms was generally less than normal in vestibular-loss subjects. Specifically for dorsiflexion of the support surface, as shown in Fig. 2, vestibular-loss responses were considerably less than normal (see columns DF in the upper left of Fig. 6). The extent of this abnormality leading to a 50% reduction in TA responses and the ensuing stabilizing ankle torque between 160 and 260 ms (see DF columns labelled in the lower left of Fig. 6) have been documented in detail elsewhere (Allum and Pfaltz 1985; Keshner et al. 1987; Allum et al. 1994). Combining the dorsi-flexion of the support surface with rearward translation caused a reduction of the TA response in normals (see Fig. 3) compared with the responses of Fig. 2 (see columns labelled BT+DF in Fig. 6) and a concurrent reduction in vestibular-loss subjects, leading to response differences between normals and vestibular-loss subjects again of the order of 50%. The reduction in the TA response in both groups of subjects for the protocol of Fig. 3 occurred despite the increased rate of dorsi-flexion. It appeared that once triggered the amplitude TA responses between 120 and 220 ms were inversely related to the velocity of knee flexion, suggesting that TA responses might well be additionally triggered by the presence of knee-locking when the upper and lower leg are extended, rather than ankle inputs alone as Fig. 1 suggests. After 240 ms, TA activity was greater than normal in vestibular-loss subjects for the protocol of Fig. 2 (see upper left of Fig. 7). This increased activity was presumably an attempted stabilizing reaction counteracting the effect of excessive paraspinal activity pulling the trunk backwards. Consistent with this viewpoint, eyes-closed TA activity increased further after 500 ms in Fig. 2 as the trunk showed an increasing backwards-falling velocity.

Discussion

The current experiments were designed to highlight the differences between the contributions of local proprioceptive reflexes and globally acting vestibulo-spinal reflexes when these reflexes interact with triggered balance-correcting responses generating muscle activity to rapidly restabilize a disturbed upright posture. Triggered balance-correcting responses elicited by perturbations to stance are quite complex. The muscle activity appears across a number of joints simultaneously within a narrow time window of 100–120 ms and the phasic activity lasts some 100–200 ms. This phasic activity is followed by later (post-250 ms), slowly varying stabilizing activity, bringing the body to an upright position with near-zero trunk sway. As this report has shown, for the balance-correcting activity pattern to be elicited, muscle stretch at the ankle and knee joints is not required, nor is vestibular input. This report does, however, suggest that the absence of ankle and vestibular inputs can cause delays in the onsets of GASTROC and PARAS balance-correcting responses. The greatest delays of up to 20 ms in PARAS occurred with absent vestibular inputs. In general though, our results indicate that the balance-correcting responses are centrally generated using neither ankle, knee nor vestibular trigger signals. However, these signals may help determine the pattern of muscles to activate once a cluster of patterns is centrally triggered. Specifically our observations were able to exclude vestibular inputs and stretch to ankle and knee muscles prior to 100 ms as the primary trigger signals. Clearly, balance responses must be triggered from a sensory source that reliably detects the early onset of the disturbance to stance and serves as a basis for generating every spatial-temporal pattern of muscle activity that must be elicited, depending on how the multi-link structure of the upright body is perturbed. Based on early unloading and stretch reflexes in PARAS muscles, we propose that rotation of the trunk induces the somatosensory signal necessary for triggering balance corrections.

The early changes in PARAS muscles at 80 ms which led us to conclude that trunk rotation signals are crucial for triggering balance corrections may well be co-related with other somatosensory signals at the hip joint, which could equally well serve as a trigger signal. That is, other trunk rotation-sensitive receptor systems may well serve as complementary trigger systems, particularly if the reflex responses of these systems are even earlier than 80 ms. The suggestion that joint receptors in the lumbar vertebral column trigger balance corrections (Gurfinkel et al. 1981; Horstmann and Dietz 1990) would not differ fundamentally from our suggestion concerning trunk rotation signals because changes in the responses of lumbar joint receptors would be expected as the trunk rotates and its loading on hip and trunk joints changes. Likewise, the proposal of Forssberg and Hirschfeld (1994) that pelvis rotation rather than hip rotation is the primary trigger signal is essentially equivalent to our suggestion because pelvis rotation in the absence or presence of hip rotation would induce length changes in the PARAS muscles. Once the primary trigger signal occurs, we propose that other trigger signals, secondarily vestibular (as described in this report) and at the knee (also cited by DiFabio et al. 1992) and perhaps, at a tertiary level, ankle inputs (see Schieppati et al. 1995) and neck inputs (see Horak et al. 1994; Shupert and Horak 1996) provide supplementary trigger signals to establish the final timing of triggered automatic balance corrections. This timing is presumably dependent on where along the body the perturbation occurred and whether the perturbation restricted body movement because a joint became locked. For example, as this report and previous work (Keshner and Allum 1990) have

emphasized, dorsi-flexion of the support surface causes little knee motion and the body moves essentially as two links, legs and head-trunk. For support-surface rearward translation, the body moves as three links, because then knee motion is considerable. These two types of perturbation lead to two distinct muscle-timing patterns (Allum et al. 1993), with differences between the timing patterns appearing in the trunk and neck muscles (abdominals and TRAP). Other timing patterns are presumably triggered by other types (Horak and Nashner 1986) and directions of support-surface motion.

Because there has been much emphasis on the role of ankle inputs on triggering balance corrections (Nashner et al. 1982; Nashner and McCollum 1985; Horak and Nashner 1986; Horak et al. 1990, 1994; Diener et al. 1984), it is important to document why these inputs may not fulfill this role and to examine alternative roles for ankle inputs. The most crucial observation is that nulling ankle inputs and controlling for a lack of 50-ms-onset stretch reflexes in triceps surae muscles yields a set of balance-correcting responses of almost identical timing to those occurring when ankle inputs are enhanced and stretch reflexes at 50 ms to rearward support-surface movement are very prominent. The small 12-ms shifts, which occur for normal subjects, in the well-known distal-to-proximal activation pattern of GASTROC (Horak and Nashner 1986; Allum et al. 1993) at 108 ms and PARAS at 136 ms when ankle inputs are nulled (Fig. 1) compared with when those inputs are enhanced (Fig. 3) are too small to be accounted for by the delay of over 50 ms in the stretch responses of triceps surae muscles if ankle inputs were indeed the triggering signal. This report has called into question two previously cited aspects of the 108-ms response in triceps surae. It is questionable whether this response is part of a centrally arranged muscle synergy calling for an activation first in triceps surae, then hamstrings, then PARAS (Horak and Nashner 1986). Activation metrics, i.e. onset latency and amplitude modulation, described in this report suggest the triceps surae response at 108 ms could well be a stretch-reflex response to knee re-extension rather than a centrally triggered balance correction. Furthermore, the possibility that the 108-ms response, which is the first response observed in triceps surae with nulled ankle inputs, could represent a vestibulo-spinal response which is switched in when ankle inputs are absent (Nashner et al. 1982; Horak et al. 1994) could not be supported by the evidence in this report. Vestibular-loss subjects had similar amplitudes in the triceps surae responses with nulled ankle inputs to those of normals albeit with 5-ms-earlier onsets which could be ascribed to an effect of a forwards-leaning posture in vestibular-loss subjects. Even if the second stretch to triceps surae causing the response at 108 ms is postulated as the proprioceptive trigger mechanism in the lower leg for balance corrections which then ascends in a distal to proximal manner rather than the earliest stretch at 50 ms, the timing of paraspinal responses in vestibular-loss subjects do not support the ankle muscle trigger hypothesis either. Responses in vestibular-loss subjects are earlier in triceps surae muscles. Therefore, PARAS onset latencies should also be earlier in these subjects, not later as we observed. In summary our results appear to exclude lower-leg proprioceptive inputs as the primary trigger signal for balance corrections.

Previous observations have established that balance corrections are modulated by proprioceptive and vestibular inputs in a continuous manner so that movement strategies are not discrete but form part of a continuum of strategies dependent on initial link velocities (Allum et al. 1993). Other authors have also emphasized the role of proprioceptive and vestibular systems in modulating balance corrections (Diener et al. 1988; Forssberg and Hirschfeld 1994; Horak et al. 1994; Schieppati et al. 1995). Our current observations build on our previous observations (Allum et al. 1993, 1995) by suggesting that the vestibular modulation of balance corrections arresting forward trunk sway may be classified into three types: essentially no modulation as in triceps surae muscles; modulation of stretch reflexes plus modulation of balance corrections as in quadriceps; and modulation of balance-correcting and later balance-stabilizing responses as observed in TA, PARAS and TRAP muscles. Perhaps the most interesting of the three types of modulation is that observed in QUAD because it includes an early vestibulo-spinal influence consistent with observations of early otolith-triggered responses in leg muscles (Greenwood and Hopkins 1976). The change in stretch-reflex activity in QUAD suggests that early vestibulo-spinal activation during postural control of upright stance in the pitch plane must be functionally coupled to a stretch reflex and be a muscle targetted to receive vestibular modulation. Thus, changes in stretch-reflex activity in triceps surae muscles with vestibular-loss may not occur for two reasons. First, because the onset of stretch-reflex activity at 50 ms and 100 ms is not occurring at the same time as the onset of vestibulo-spinal responses and, second, because, as indicated in this report, triceps surae muscles receive only a weak vestibulo-spinal modulation.

This absence of early vestibulo-spinal influences in SOL during postural disturbances despite the presence of an influence during free-fall (Greenwood and Hopkins 1976; Lacour et al. 1978) is presumably an outcome of the pre-set interaction between proprioceptive and vestibulo-spinal pathways for the direction and mode (two or three link moving body segments) we investigated. Examples of such interactions have been described in cats both at a functional (Prochazka et al. 1988) and at a neurophysiological level (Pompeiano et al. 1995). Specifically, vestibular input via the locus coeruleus is employed to achieve a differential weighting of proprioceptive and vestibulo-spinal modulation at lumbar-spinal motoneurones (Pompeiano et al. 1995). The example of early interaction between these proprioceptive and vestibular inputs we have identified in QUAD is functionally useful because a collapse of the body at the knees so prevalent in the elderly (Sauvage et al. 1992) would be sensed both by stretch receptors in QUAD muscles and otolith systems sensing the vertical linear acceleration of the head.

Interestingly, the largest linear accelerations recorded at the head during postural disturbances are those associated with vertical motion of the body (Allum and Pfaltz 1985). Thus in the case of QUAD the simultaneous arrival of both sensory influences would have an immediate righting effect on the body as the knees bend, but in the case of vestibular-loss would lead to the presence of an early destabilizing influence on the body as observed by the early changes in backward trunk-pitching profiles when the support surface was translated (Figs. 1 and 3) and cumulatively by the excessive forwards-pitching trunk profiles at 250 ms observed in the test protocols of the current experiments.

Whether or not hip flexor and extensor muscles also demonstrate early interactions between proprioceptive stretch reflexes and vestibulo-spinal signals during early stretch reflex activity resulting from a balance perturbation is not known. For the PARAS muscles, such an interaction was not observed, leading us to suggest that trunk angular velocity coding in trunk muscle afferents provides an excellent trigger signal for balance corrections because this trigger would not be susceptible to vestibular-loss based on our observations of stretch and unloading responses in PARAS. Thus it appears, on the basis of early reflex responses in PARAS that the early afferent signals from trunk muscles in response to a balance perturbation emanate from only the proprioceptive sensory system. In contrast, early neck muscle activity is known to depend on complex interactions between vestibular and proprioceptive signals (Kasai and Zee 1978; Horak et al. 1994; Ito et al. 1995; Allum et al. 1997). The plethora of sensory interactions in neck muscles, including the influence of preprogrammed activity, yields a number of differing, early stretch reflex response onsets from which a vestibular or proprioceptive response can only be distinguished with difficulty (Allum et al. 1997; Ito et al. 1995). Thus, from a consideration of possible sites that could provide triggering afferent signals for balance corrections, the knee and neck are less suitable because of the interactions at these sites with vestibular inputs. Our studies also indicate no evidence that proprioceptive inputs from these sites or ankle proprioceptive inputs trigger balance corrections. The trigger site with the least interaction from other sensory inputs yet most important for upright stability appears to be the trunk. If the trunk is not kept near upright a fall will result, as Figs. 2 and 3 demonstrate in vestibular-loss subjects. Once balance corrections are triggered, central mechanisms presumably use both proprioceptive information from a number of joints, vestibular inputs, and prior knowledge of the postural stance and environment to select the thresholds and weighting underlying vestibular and proprioceptive modulation of balance synergies. It is an open question how the proportion of modulated versus the basic non-modulated response amplitude is preset for various postural movements, for example, in preparation for arm and stepping movements (Nashner and Cordo 1982; Burleigh and Horak 1996), and in the presence of an alternative support reference (Jeke and Lackner 1995) compared with the simpler balance corrections described in this report. It is also an open question how the weighting of vestibular and proprioceptive contributions is selected instantaneously based on the way the body is perturbed (Keshner and Allum 1990; Allum et al. 1993).

The selection of appropriate balance-correcting timing patterns for different types of perturbed stance implies a central synthesis of relevant afferent information that could be simplified by relegating some of the tasks to structures of the lower nervous system. One of these task simplifications would be to select the basic timing of postural responses (Forssberg and Hirschfeld 1994) prior to selection of the response scaling at different muscles. Previously we have argued that timing pattern selection is set on the basis of the number of links that the body is forced to move with at the onset of the disturbance (Allum et al. 1993). If the disturbance forces the knees into a locked position so that the legs move as one rather than two elements, it is to be expected that QUAD and triceps surae muscles will have somewhat different timing patterns. Another way our results suggest that the CNS simplifies the computation is to pre-weight groups of muscles to receive more proprioceptive or more vestibular weighting. Presumably the selection of TA, QUAD and PARAS muscles to receive more vestibular weighting and the triceps surae muscles to receive less vestibular and emphasized proprioceptive weighting during the control of trunk forwards-pitching, preventing total body rearwards-falling, is based on a learning process in early infancy in which the growing infant employs these muscles to prevent a backward fall. The fact that subjects who suffer vestibular-loss as adults cannot switch this weighting to prevent a fall, even after years of experience controlling falls, implies that this basic level of pre-processing of balance corrections cannot be modified by switching the modulation weighting between muscles and is pre-set. Thus the final central computation of the metrics of the balance-correcting synergy would be the selection of a base-level amplitude of muscle activation about which proprioceptive and vestibular inputs would interact. We hypothesize that the amplitude of this basic activation level is rapidly selected using prediction of the amplitudes with which the trunk and legs will move as the balance correction occurs. With this organization sequence, the CNS would have the advantage of a rapid reaction to a postural disturbance and have computational freedom to make minor postural adjustments during the following stabilizing period when the body is repositioned in a new upright position with low levels of muscle activity. Our studies indicate that it is in this stabilizing period that the CNS has more flexibility to choose between visual, vestibular and somatosensory inputs in modulating muscle activity rather than during initial rapid balance corrections.

Acknowledgements Support for this work was provided by Swiss National Research Foundation grant 32-41957.94 to J. H. J. Allum. Typographical assistance was provided by Ms. E. Clarke.

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