

A multisensory posture control model of human upright stance

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Abstract: We present a multisensory postural control model based on experiments where the balance in normal subjects and vestibular loss patients was perturbed by application of external torque produced by force-controlled pull stimuli. The stimuli were applied while subjects stood on a stationary or body-sway-referenced motion platform with eyes closed and auditory cues masked. Excursions of the center of mass (COM) and the center of pressure (COP) were analyzed using a systems analysis approach. The results were compared to an ‘inverted pendulum’ model of posture control. The model receives input from four sensors: ankle proprioceptors, semicircular canals, otoliths, and plantar pressure sensors (somatosensory graviceptors). Sensor fusion mechanisms are used to yield separate internal representations of foot support motion, gravity, and external torque (pull). These representations are fed as global set point signals into a local control loop based on ankle proprioceptive negative feedback. This set point control upgrades the proprioceptive body-on-foot (support) stabilization into a body-in-space control which compensates for support tilt, gravity, and contact forces. This compensation occurs even when the stimuli are combined or a voluntary lean is superimposed. Model simulations paralleled our experimental findings.

Introduction

It is still not understood how humans control their upright stance and how they embed their voluntary actions into postural mechanisms. Research in this field faces the problem of a high complexity of the mechanisms involved (multi-segment dynamics, biomechanics of the human body, multisensory integration, volition, cognition, etc.). The complexity of the system requires that researchers develop conceptual hypotheses concerning the involved sensors, the internal ‘reconstruction’ and representation of the external stimuli by means of sensor fusion mecha-

nisms (multisensory integration), and the implementation of these internal stimulus representations into the postural control mechanism (for the clinical approach to multisensory integration of space see also Kerkhoff, 2003, this volume, and Rode et al., 2003, this volume). Conceivably, such an approach has to build upon a number of simplifications, such as the assumption that the control is based to a large extent on a sensory negative feedback mechanism (e.g. Johansson and Magnusson, 1991; Fitzpatrick et al., 1996).

We conceptualized the *physical stimuli*, which challenge body equilibrium of an upright standing human, as consisting of the following.

- (1) Force field due to gravity.
- (2) Motion of the body’s support surface. We consider here only support surface tilt about an axis through the ankle joints of both feet, i.e. tilt in the anterior–posterior (a–p) direction.

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- (3) External contact forces, such as a pull on, or a push against the body.

Humans use several sensors to obtain internal estimates of these stimuli. The most relevant sensory cues are derived from visual input (Berthoz et al., 1979; Amblard et al., 1985; Bronstein, 1986; Van Asten et al., 1988; Peterka and Benolken, 1995), proprioceptive input (Kavounoudias et al., 2001), the vestibular system (Hajos and Kirchner, 1984; Britton et al., 1993), and the somatosensory system (Mittelstaedt, 1996; Jeka et al., 1998).

To simplify, we consider here subjects who have no visual or auditory spatial orientation cues (i.e. eyes are closed and no relevant sound source). In this situation, we assume that the aforementioned three physical stimuli are perceived predominantly with the help of the following four *sensory signals*.

- (1) *An otolith-derived signal of the body lean in space.* It is known from clinical and experimental work that imbalance between the vestibular sensors in the two labyrinths, resulting from unilateral lesion or stimulation, give rise to postural reactions (see Massion, 1994; Horak and Macpherson, 1996; Massion and Woollacott, 1996). It is generally believed that a relevant part of the vestibular information stems from the otolith system. This is thought to provide a signal related to the body orientation with respect to the earth vertical. After shaping it according to current body geometry, it yields an appropriate 'antigravity' extensor muscle tone, such that the gravitational reaction forces are neutralized.
- (2) *A canal-derived signal of support surface motion in space.* There is experimental evidence that humans perceive rotation of the body support with the help of vestibular semicircular canal signals, and it has been suggested that the corresponding internal signals are also used for postural control (Mergner et al., 1997).
- (3) *A somatosensory graviception from deep plantar mechanoreceptors.* There is clinical evidence that posture control is impaired following loss of plantar somatosensory afferents as well as experimental evidence that stimulation of these afferents evokes postural reactions (see Maurer et al., 2000, 2001).
- (4) *A proprioceptive signal of body angular posi-*

tion with respect to the foot. Normally, postural reactions take place at several joints along the body axis. However, here we assume for simplification that the proprioceptive signal stems from one joint only (in this view the body is conceptualized as an 'inverted pendulum' with a single head-trunk-leg segment rotating about the ankle joint).

It is still an enigma how these four sensors interact and what their functional relevance is during posture control. For an analytical approach (such as a systems analysis approach), it would be desirable to further reduce the number of sensors and to compare the system's behavior in the absence versus the presence of a given sensor. We conceived of two possibilities. One would be to compare the postural responses to external perturbations of patients with bilateral loss of vestibular function with those of normal controls. In the above adopted view, the patients would have only the ankle proprioceptive cue and the somatosensory graviceptive cue for postural control, while normal subjects would have all four sensors.

The second possibility would be to 'freeze' the proprioceptive signal by a procedure called 'body-sway referencing of the platform' (BSRP). BSRP is performed by making a motion-controlled platform rotate 1:1 (i.e. in fixed register) with the body sway angle. This procedure has been commonly used in posture control studies to 'open' the ankle joint proprioceptive feedback loop by keeping the ankle angle constant during body sway while the subject is still able to apply torque about the ankle joint. In the envisaged comparison between patients and normal subjects, the patients now would have to maintain balance relying solely on somatosensory graviception, while the normal subjects would have, in addition, the otolith and canal signals.

In the present study we performed these comparisons using a contact force as an external perturbation. The stimulus was created by applying a force-controlled pull on a subject's body around the ankle joints in the a-p direction. We measured center of mass (COM) and center of pressure (COP) excursions to sinusoidal pull stimuli at different frequencies and with various force magnitudes. Gain and phase curves obtained from the responses were compared to simulated data using a dynamic postural control model into which we implemented the four

sensors, sensor fusion mechanisms, a sensorimotor transformation, and the corresponding physics and biomechanics.

Methods

Subjects

Experiments were performed in six normal subjects (age 34–53 years) and four patients with loss of vestibular function (age 33–42 years). Loss of vestibular function in patients was assumed on the basis of routine clinical examinations (e.g. balancing problems when standing on foam rubber with eyes closed) and electronystagmographic criteria (absence of caloric nystagmus and of rotation-evoked VOR) as well as on case histories (meningitis and ototoxic medication in childhood). Apart from hearing problems and slight vertigo during rapid head movements, patients showed no neurological symptoms. In compliance with the Helsinki declaration (1964) all subjects gave their informed consent to the study which was approved by the local Ethics Committee of the Freiburg University Clinics.

Experimental setup and procedures

The experiments were conducted with a setup which allowed us to apply a pull on the subjects' body in the sagittal plane and thereby to exert a defined torque about their ankle joints. The stimuli were applied via a body harness by means of two force-controlled cable winches (servo motors) under computer control, as shown schematically in Fig. 1. Sinusoidal stimulus profiles were applied at five different frequencies (0.05, 0.1, 0.2, 0.4, and 0.8 Hz) and three different amplitudes of torque about the ankle joint (± 1 , ± 4 , and ± 16 Nm). During these stimuli subjects were standing upright, heels 5 cm apart, on a custom-built motion platform. The platform rested on six 'legs', each incorporating a motor capable of producing a change in the length of the leg (hexapod with Stuart principle). The motors were controlled by a computer to generate platform tilts in the sagittal plane with the axis through subjects' ankle joints during some of the stimuli.

Mounted on the motion platform was a force-transducing platform (Kistler[®], platform type 9286,

Winterthur, Switzerland) that registered subjects' COP shift in the a–p direction. We also measured subjects' hip-to-ankle and shoulder-to-hip angular excursions in the sagittal plane by means of an optoelectronic device with active markers (Optotrak 3020[®], Waterloo, Canada). These measures were used to obtain estimates of subjects' COM angular excursion in the sagittal plane.

Two experiments were performed. In one experiment, the pull stimuli were applied while the platform was kept stationary ('pull alone' experiment). In the other, the pull stimuli were applied while platform position was coupled 1 : 1 to subjects' COM angular position, by which their ankle angle essentially was held constant ('pull with BSRP' experiment). In the BSRP condition, any spontaneous, actively produced, or stimulus-evoked angular excursion of the body off its primary position causes the platform to rotate with the body with a small time delay (<40 ms) and error (<2%).

Subjects were instructed to always remain upright and to maintain balance. The stimuli with the longest duration (0.05 and 0.1 Hz) were presented as two separate trials with 4 stimulus cycles each, while those with shorter duration were presented within one trial which comprised either 8 stimulus cycles (0.2 and 0.4 Hz) or 16 cycles (0.8 Hz). The order of these presentations was varied, starting always with a stimulus in the mid-frequency range with 4 Nm, but varying the remaining ones in random order. The pull stimuli on stable support were always performed prior to those in the BSRP condition, in order to allow subjects to become accustomed to the stimuli. No attempt was made to hide from the subjects which of the two conditions was applied. The patients and three of the normal subjects were completely naive as concerns the postural tests used, the remaining three normal subjects were naive as to the stimulus frequency and amplitude presented. During the stimulus presentations, we instructed the subjects to perform simple mental arithmetic, in order to draw their attention away from the postural stimuli.

Data analysis

Optotrak and Kistler output signals as well as platform rotation and pull stimulus signals were recorded

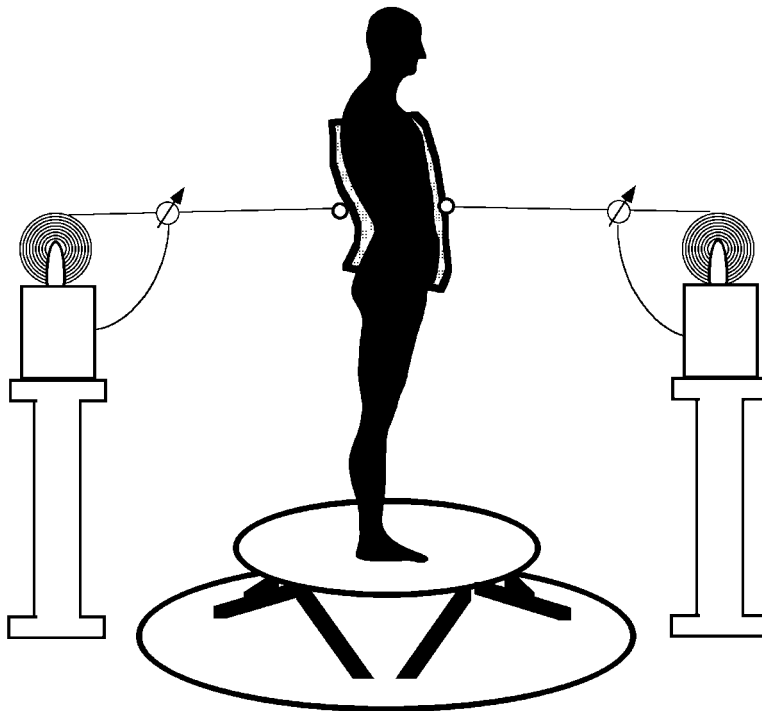


Fig. 1. Schematic presentation of stimulus conditions. Sinusoidal pull stimuli were applied by means of two force-controlled cable winches via a padded body harness to exert defined torques about the subject's ankle joints in the a-p direction. The subject stood on a motion platform which was either stationary or was tilted in fixed register with spontaneous and stimulus-evoked body excursions (body-sway-referenced platform, BSRP).

on a computer system (IBM compatible Pentium[®]) via analog-digital converter with a 100 Hz sampling rate. The data were recorded with software programmed in LabView[®] (National Instruments, Austin, TX, USA) and analyzed off-line with custom-made software programmed in MATLAB[®] (The MathWorks Inc., Natick, MA, USA). COM angular displacement was calculated from the shoulder-to-hip and hip-to-ankle angular displacements according to anthropometric data of Winter (1990). Within-subject averages were obtained for six cycles of each stimulus. Further analysis was performed using a spreadsheet program (Microsoft Excel[®]) and a statistics program (StatView[®], SAS Inc., Cary, NC, USA).

A spectral analysis was performed on the pull stimulus and response (COP and COM) waveforms using a discrete Fourier transformation. Fourier coefficients at the frequency corresponding to the stimulus frequency were used to calculate gain (peak response amplitude divided by peak stimulus amplitude) and phase (relative timing of the response

compared to the stimulus). Gain measures have units of cm/Nm for measures related to COP displacements caused by the pull stimulus, and $^{\circ}$ /Nm for COM angular displacements caused by the pull stimulus. Gain values of zero indicate that the pull stimulus had no effect on the subject's COM angle or COP displacement. Gain and phase change patterns as a function of stimulus frequency characterize the dynamic behavior of the postural control system.

By developing models of postural control that explain these patterns, we hope to understand how sensorimotor integration is achieved in the postural control system. The model was implemented in Simulink/MATLAB[®].

Results

Pull alone

Fig. 2A shows the results of normal subjects to pull stimuli on a stationary support for the different stim-

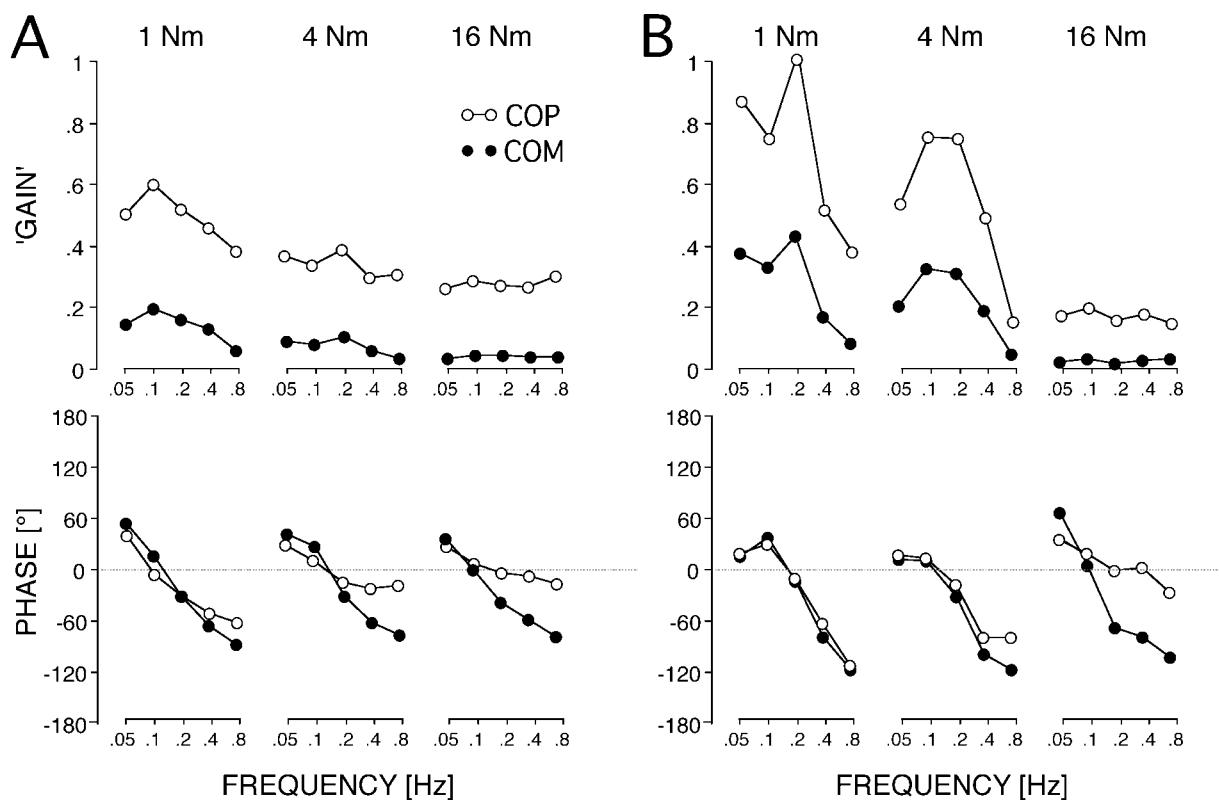


Fig. 2. Responses of normal subjects (A) and vestibular loss patients (B) to sinusoidal pull stimuli on a stationary platform. The angular excursion of COM and the linear shift of COP are given in terms of mean gain (in $^{\circ}/\text{Nm}$ and cm/Nm , respectively) and phase values as a function of peak ankle torque (1, 4, 16 Nm) and of stimulus frequency, as indicated. COM and COP gains show a clear dependency on stimulus amplitude (reflecting that excursions were relatively larger, the smaller the stimulus was), unlike the corresponding phase values. Furthermore, gain and phase of COM and COP change as a function of stimulus frequency (exception: gains with the 16 Nm stimulus). The responses of patients in (B) are similar to those of normal subjects, apart from larger gain values in the mid- to low-frequency range with the 1 Nm and 4 Nm stimuli.

ulus frequencies and the three peak ankle torques. In the upper panel we give the gain measures for the COM (in $^{\circ}/\text{Nm}$) and the COP (in cm/Nm). In the lower panel the corresponding phase values are given.

Peak COM excursion by the pull stimulus in normal subjects did not exceed 1° in absolute values, even with the largest stimulus magnitude (16 Nm). This is reflected in the rather low COM gain values. Noticeably, the gain values decrease with increasing stimulus magnitude (thus, the small stimuli were counteracted relatively less than the large ones). For instance, gain was about 0.2 with the 1 Nm/0.1 Hz stimulus and decreased to 0.02 with the 16 Nm/0.1 Hz stimulus. In contrast to this clear gain

nonlinearity, COM phase is essentially independent of stimulus magnitude.

COM gain also depends on stimulus frequency. With the smallest stimulus magnitude (1 Nm), increase of frequency leads to an increase in COM excursion, with a peak at 0.1 to 0.2 Hz, after which it decreases at higher frequencies. This frequency-dependent modulation levels off when gain becomes small with the larger stimuli. COM phase is approximately in phase with the stimulus at 0.1 and 0.2 Hz and develops some lead at 0.05 Hz and some lag at 0.4 and 0.8 Hz.

Normal subjects' COP shows qualitatively a very similar gain behavior in terms of both the nonlinearity related to stimulus magnitude and the modulation

in relation to stimulus frequency. Also COP phase is similar to COM phase, especially with the smallest stimulus magnitude (± 1 N m). At higher magnitudes, however, the COP phase tended to level off at about 0° , losing the frequency modulation almost completely with the 16 N m stimulus, similar to the COP gain curve.

The results of vestibular loss patients are shown in Fig. 2B. Patients exhibit qualitatively similar COM and COP responses as normals during the pull stimuli on the stationary platform. This applies to the gain nonlinearity, the gain modulation with frequency, and the phase modulation with frequency. A clear difference, however, concerns the gain of COM and COP at low frequencies and small stimulus magnitudes. Gain is then almost double compared to that in normal subjects. At the higher frequencies and the highest stimulus magnitude (16 N m), gain is essentially in the normal range (thus, patients behave like normal subjects and limit their COM and COP excursions to values $<1^\circ$ and <3 cm, respectively).

Pull with body-sway-referenced platform

Normal subjects have no difficulty maintaining stance on a sway-referenced platform with their eyes closed. However, stance in this condition is associated with a relatively large and irregular body sway. The effect of this variability was largely removed, however, from the experimental data by the averaging process. Subjectively the platform is experienced as unstable in this situation, since any body lean is associated with a corresponding tilt of the platform. Subjects tend to avoid this and normally try to keep the body orientation vertical.

Fig. 3 shows the COM and COP gain and phase curves of normal subjects obtained with the pull stimuli in the sway-referenced condition. The COM gain curves are qualitatively similar to those obtained with the stationary platform, showing again the nonlinearity related to stimulus magnitude and the modulation in relation to frequency. However, overall gain is higher by a factor of approximately 2 (i.e. pull-evoked body excursions were essentially doubled). Also COM phase is qualitatively similar as before (lead at low and lag at high frequency, no major change related to stimulus magnitude). But

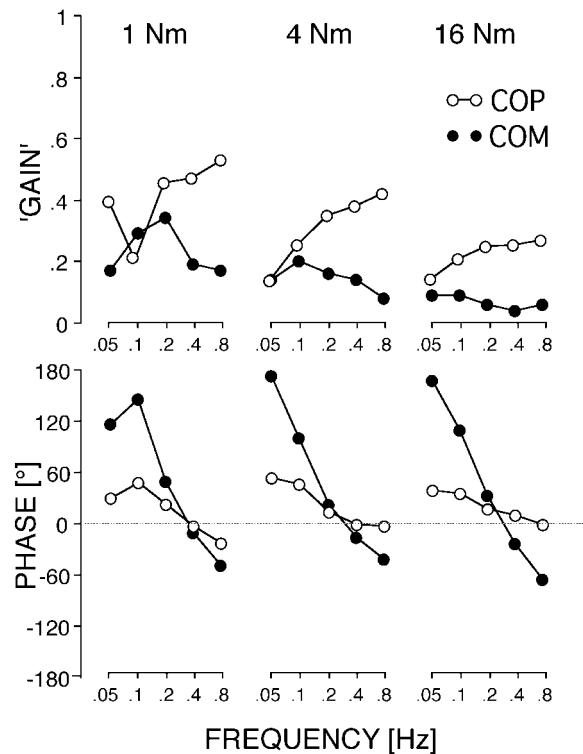


Fig. 3. Responses of normal subjects to sinusoidal pull stimuli on body-sway-referenced platform (BSRP). Presentation as in Fig. 2. Compared to the normals' responses on the stationary platform (Fig. 2A) the gain and phase curves of COM are similar, apart from slightly higher gain values and a shift of the phase towards lead. The latter also applies to COP phase. In contrast, the COP gain curves now start with relatively small values at low frequency and increase with increasing frequency, unlike on the stationary platform. Vestibular loss patients were unable to maintain balance in the BSRP condition (not shown).

overall there is a shift towards lead as compared to the stationary condition; already at 0.2 Hz the phase shows a lead, which further increases at the lower frequencies and reaches approximately counter-phase (180°) at 0.05 Hz. Thus it appears that normal subjects produce a counter-leaning body position when compensating for the pull at this low frequency. This counter-leaning position allows gravity to act on the body to counteract the pull.

COP gain in the sway-referenced condition shows a nonlinearity related to stimulus magnitude as before, while its modulation by frequency is clearly different compared to the stable-platform experiment. The largest gain is reached at the highest frequency,

and shows a monotonic decrease with decreasing frequency. COP phase is slightly shifted towards a lead as compared to the platform stationary condition, but otherwise it remains very similar.

Patients are not able to maintain balance on the sway-referenced surface with eyes closed. When standing on the body-sway-referenced platform with eyes open, they can maintain balance even with the pull stimulus. However, as soon as they close their eyes, they fall, even without the pull stimulus.

Discussion

To reduce the high complexity of the human postural control system during upright stance, we restricted access to visual and auditory orientation sources, thus limiting the postural control system to information from mainly four sensors (otolith, canal, ankle proprioceptive, and somatosensory graviceptive sources). The external physical events, which may endanger body equilibrium, consist mainly of three stimuli (gravitational pull on the body, external pull produced by a contact force, and tilt of the body support surface). The interactions of these physical factors were investigated in normal and vestibular loss subjects using a system analysis approach by measuring COM and COP excursions during force-controlled pull stimuli. In vestibular loss subjects, it was assumed that equilibrium control is achieved only by means of ankle proprioceptive and plantar somatosensory cues. We further challenged the postural control system by introducing tilts of the platform that tracked COM body sway (the BSRP condition). This 'opened the proprioceptive loop' and limited sensory information solely to somatosensory cues in vestibular loss subjects, and to somatosensory and vestibular (otolith and canal) cues in normals. To better understand the interactions of sensory information required for postural control, we developed a computer model that allowed us to compare experimental results with model predictions based on plausible sensory integration schemes.

We found that the pull-evoked COM and COP excursions on stable support are very small in normal subjects, indicating a very efficient control mechanism for the effects of this stimulus. At 0.1–0.2 Hz (approximately the *eigen*-frequency of body

sway) the pull-evoked excursions are maximal and in phase with the stimulus (with a phase lead/lag when frequency was decreased/increased). The responses showed a clear nonlinearity in that the gain was largest (the pull-evoked excursions became relatively larger) with the smallest stimulus magnitudes. Since this nonlinearity was also observed in patients and was a consistent finding in normal subjects in the BSRP condition, it appears to represent a property of the postural sensorimotor control mechanism.

Patients' pull responses on stable support were similar to those of normals, apart from a higher gain in the mid- to low-frequency range (larger body excursions). In terms of our concept, the gain difference would be explained mainly by the absence of the otolith signal in patients. This requires patients to produce responses solely using ankle proprioceptive and somatosensory graviceptive cues. Admittedly, one could conceive, as an alternative, that patients used solely a proprioceptive negative feedback loop. However, our previous work showed that patients have at least some sensory information on gravity from plantar pressure receptors during platform tilt (Maurer et al., 2000), and we postulate that they use this cue to control balance during the pull stimulus. But we assume that somatosensory graviceptive cues cannot be used to distinguish between the pull evoked by an external contact force and the pull arising from gravity during body leans away from a vertical orientation. In contrast, normal subjects, using the 'force field meter' provided by the otolith system in addition to the 'contact force meter' in the plantar sole, should be able to make this distinction. Patients indeed failed to keep balance with the pull stimuli in the BSRP condition, while normal subjects were able to do so.

Normal subjects' COM response to the pull stimuli in the BSRP condition showed a pronounced phase lead at low frequency. Extrapolating from this observation to a static situation, this is consistent with subjects producing a counter lean of the body in space such that the gravitational pull almost counterbalances the stimulus pull, with some control error remaining, as indicated by the small COP shift in the direction of the stimulus. In the dynamic situation with increasing stimulus frequency, body inertia resists more and more the force that evokes body excursion, and responses become more and more

similar to the stable-platform condition where subjects produced an appropriate counter force scaled to the stimulus.

Since there is platform tilt associated with the pull-evoked body lean in the BSRP condition, canal-derived estimates of platform tilt can contribute to postural responses. Because the responses in the BSRP condition became similar to those in the stable-platform condition at high frequency and with large stimulus magnitude, one can conclude that normal subjects were able to use canal information to compensate for the platform tilt to a large degree (as they do for body tilt produced by their spontaneous sway in the BSRP condition; see the section Results).

When implementing our assumptions into a postural control model for comparison with the experimental data, we proceeded from the concept (Mergner et al., 1997; Mergner and Rosemeier, 1998) that the control is built around an ankle proprioceptive negative feedback loop, which stabilizes the body with respect to the foot and its support base. During support tilt, a canal-derived internal estimate of the tilt is fed into this 'local' loop as a set point signal, by which the body-on-support control becomes upgraded into a body-on-support-*in-space* control. This concept originally was based on psychophysical data obtained in the earth-horizontal rotational plane (i.e. perpendicular to the gravity vector). Since body sway occurs in the vertical planes, we have to consider additional mechanisms that can also cope with the effects of gravity. Furthermore, postural control mechanisms also need to cope with external contact forces, such as the pull stimuli we used in our experiments.

We developed a model, shown in Fig. 4, based on the concepts described above. The model includes a 'physics' part and a 'subject' part. The physics part includes a body composed of two mutually coupled pendulums, one being the body segment, the other the foot-platform segment. Both segments are interconnected by a joint (see inset), an actuator produces torque at this joint, and only rotations around this joint are considered. We assume that the body is always oriented approximately upward and the foot-platform segment downwards, so that gravity presses the foot firmly on the support, as long as only small angular excursions about the upright

position occur. With the foot-platform segment held stationary in space (foot-in-space angle, $FS = 0^\circ$), the body-to-foot angle (BF; i.e. the reverse of the foot-to-body angle, FB; $FB = -BF$) is coupled 1:1 to the body-in-space angle (BS). This coupling is eliminated ($FB = -BF = 0$) when FS is made to exactly follow BS (mimicked by opening the switch BSRP). Our subjects' anthropometric data were implemented in the box 'body inertia, gravity'. The input signals to the physics part consist of an external torque (the pull stimulus), a muscle torque produced by the subject in the ankle joint, and FS (platform tilt). As outputs of this part and as inputs into the 'subject', BF is sensed by ankle proprioception (PROP), BS by the vestibular system (VEST), and a shift of COP by plantar somatosensory receptors (SOMAT; determined by the shift of the COM's gravitational vector, the actively produced muscle torque, and the external torque evoked by the contact force, i.e. the pull stimulus).

The PROP sensor is considered to have essentially ideal transfer characteristics (broad band pass filter characteristics) and a gain of about unity. The PROP sensor combined with a neural controller in the form of a PID controller (P, proportional; D, differential; I, integrative) yields the local proprioceptive negative feedback loop in the ankle joint. The PID controller was set such that BS follows rather accurately a voluntary lean signal (input VOLUNT. LEAN) in the absence of all external stimuli including gravity. The VEST sensor was considered to contain two parts, one representing the canal system, the other the otolith system. We assumed that by way of a canal-otolith interaction (not shown; see Merfeld, 1995; Mergner and Glasauer, 1999; Zupan et al., 2002) the canal transfer characteristics are improved in the vertical rotational planes such that an essentially veridical internal representation of BS rotation results (signal bs). A combination of canal and proprioceptive cues in terms of a sensor fusion (Mergner et al., 1997; Mergner and Rosemeier, 1998) is taken to yield an internal estimate of foot-in-space motion (fs; $fs = bs - bf$), which is fed into the local loop as a set point signal. A detection threshold, T' , is included in the canal-derived fs signal pathway (T' is about $0.1^\circ/s$ and is expressed as a velocity threshold because the canal signal at early stages of processing codes angular velocity). This threshold, combined

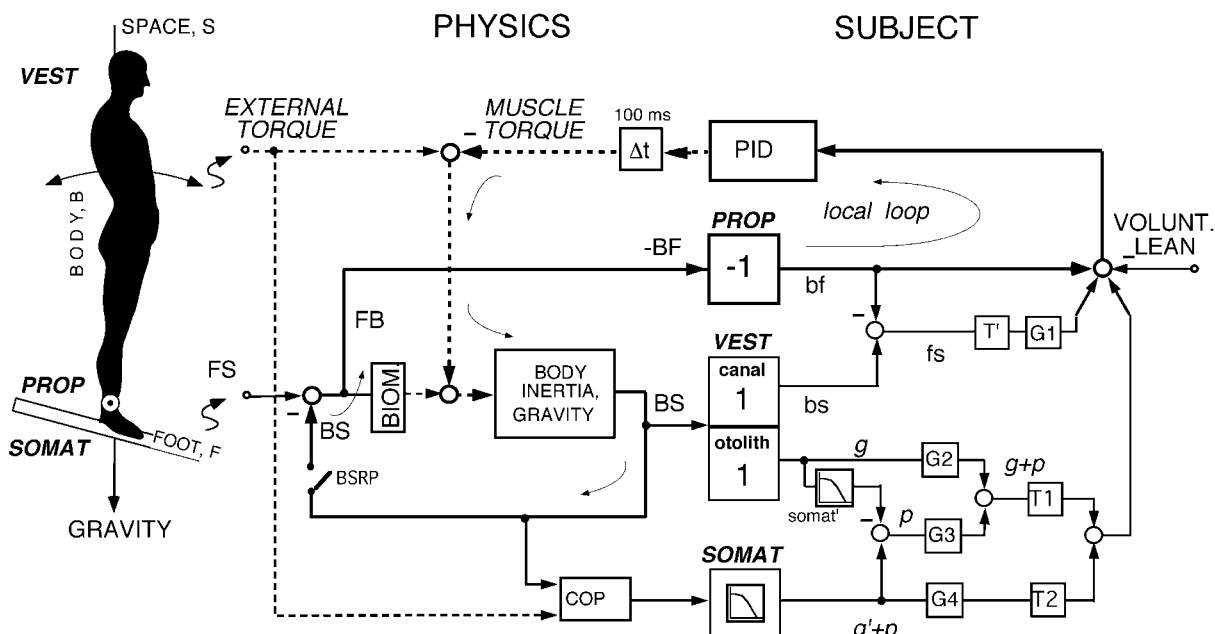


Fig. 4. Multisensory model of posture control. The inset defines the 'PHYSICS' part of the model (left) in terms of an 'inverted pendulum body' (one segment for head, trunk, and legs) that pivots about the ankle joint on a potentially rotating platform (axis through the ankle joint). Pull on the body yields an 'external torque' stimulus acting on the body, together with the 'muscle torque'. FS, foot-in-space angle (resulting from platform tilt); FB, foot-to-body angle (equal to $-BF$); BS, body-in-space angle; BSRP, body-sway-referenced platform (open switch mimics the BSRP condition). Box BIOM (for biomechanics) represents the transformation of FB into ankle torque (in the present case passive viscous-elastic properties are assumed to be zero, since these would be small as compared to external and muscle torques). Subjects' anthropometric parameters are contained in the box 'BODY INERTIA, GRAVITY'. Dashed lines represent torque and solid lines angles. All delays in the system are represented as one dead time (Δt). BS excursion leads to a shift of the COP (box COP; also affected by the external torque stimulus). The 'SUBJECT' part of the model (on the right) establishes internal representations of the external stimuli (torque, stemming from gravitational and external pull on the body, and FS angle), which are fed as set point signals, together with a voluntary signal (VOLUNT. LEAN), into a local proprioceptive negative feedback loop for body-on-support control (loop indicated by thin arrows). PROP, proprioceptive sensor; VEST, vestibular sensors; SOMAT, plantar pressure cue ('somatosensory graviceptor'; it is assumed to have low-pass frequency characteristics with a corner frequency of 0.8 Hz); somat', internal model of SOMAT; bf, bs, and fs, internal representations of BF, BS and FS, respectively; g , otolith-derived internal estimate of gravitational pull (g' , somatosensory derived version of g); p , internal estimate of external pull; T1, T2, and T', detection thresholds; G1-G4, gain of set point signals (on the order of 0.7-0.9; held constant for all simulations of the results of normals).

with other thresholds discussed below, accounts for the observed amplitude nonlinearity of the system.¹

The presumed canal-otolith interaction in the box VEST, in addition, is thought to extract from the

¹ Note that the thresholds here are thought to represent central mechanisms, in contradistinction to the thresholds of the sensors in classical sensory physiology. In our previous psychophysical studies, the central thresholds were related to mechanisms that minimise noise in the system and thereby help to achieve perceptual stability (Mergner et al., 2001). The presumed central thresholds in the present context, however, are considerably lower than those in the psychophysical studies. Their functional significance and underlying processes remain to be elucidated.

otolith signal the gravitational component (assumed to show essentially ideal transfer characteristics as well). This yields an internal representation of the body's orientation with respect to the gravitational vertical, g , and thereby a measure of the 'gravitational pull' on the COM (zero with upright orientation of the inverted pendulum). The somatosensory plantar cue (SOMAT) is assumed to show low-pass frequency characteristics, and to yield an internal representation of the sum of (1) the COM's gravitational vector with respect to the foot support base (signal g'), and (2) the additional shift of the COP induced by the pull stimulus (p). (The acceleration

components contained in COP, that are mainly due to active changes in ankle torque, are assumed to be removed by way of an interaction with acceleration signals from shear force receptors in the feet or by an efference-copy-like signal.)

Fusion of an otolith-derived signal (a sign-reversed version of the otolith signal is given the low-pass characteristics of the plantar signal by feeding it through an internal model of the SOMAT cue; *somat'*) with the plantar cues is thought to yield an internal representation of the effect of pull, $p = (g' + p) - g'$. This then yields, in combination with the original otolith-derived signal, a set point signal for the local loop to compensate for the external forces in any adopted body position ($g + p$).² The set point signal is given a detection threshold, T1, on the order of 0.1°.

This form of the model was used for simulation of the results of our normal subjects obtained with the pull stimulus on stationary support (note that the model contains, in addition, a direct set point signal from SOMAT, $g' + p$, which was disabled here; see below). The simulation results, given in Fig. 5A, are consistent with the essential features of the experimental data. Preliminary testing showed that the model also yields similar successful simulations of our previous data obtained with tilt stimuli (Maurer et al., 2000).

In order to describe the data obtained from the patients, we first considered a model that included only the local proprioceptive loop. The simulation results described the experimental results rather well for the 1 N m pull stimulus if the PID values in the neural controller were slightly increased. However, the experimental results obtained with 4 and 16 N m stimuli were clearly better than the predicted ones (gain was smaller, reflecting smaller body excursions than expected from model simulations). This led us to assume that patients use, in addition to the proprioceptive loop, a set point signal derived from the

SOMAT sensor (the $g' + p$ signal). This signal includes a threshold, T2, whose value is about 0.6° (which corresponds to a COP displacement of about 1 cm). The simulations described rather accurately the experimental findings (Fig. 5B). This modeling result suggests that the patients control small body-on-support excursions entirely with the proprioceptive loop, while larger excursions require the help of the plantar somatosensory cues to prevent the center of ground reaction force from shifting beyond the base of support given by the foot. This extended model for patients is also able to describe their tilt responses from the earlier experiments (Maurer et al., 2000). In addition, the model correctly predicts the experimental finding that patients cannot maintain stance in the BSRP condition.

We conceive furthermore that normal subjects also make use of the somatosensory signal, but use it with a wider threshold, so that there is only a small somatosensory contribution for the pull stimulus on stationary support. In fact, for the simulation of the experiment in Fig. 2A (Fig. 5A) the somatosensory derived signal was not included in the model. In contrast, when simulating the results of normals for pull in the BSRP condition, we used the somatosensory signal with a low threshold (and disabled the $g + p$ signal by raising its threshold). This allowed us to simulate the normals' results with the 1 N m stimulus where COP phase and gain curves became clearly dissociated from those of COM (Figs. 3 and 6). The results obtained with the 4 N m and 16 N m stimuli were also well simulated. Therefore, we postulate that normal subjects can make use of two internal estimates of $g + p$, one that is primarily derived from the otolith signal and the other that is based primarily on the somatosensory cue alone. We speculate that normal subjects can shift the weighting to emphasize the use of one or the other (in the extreme, this weighting of the two thresholds, T1 and T2, can be considered to represent a 'switch' between the use of vestibular and somatosensory graviceptive control). From the simulation of the data, we determined that normal subjects balanced the pull stimulus in the BSRP condition mainly by using the COP-like somatosensory cue.

Using sinusoidal stimuli we cannot exclude that our subjects used predictive mechanisms to some extent for their postural responses (this despite the

² Note that during a voluntary body lean, for instance, these signals are used to compensate the external forces that arise with the lean, such as the gravitational pull, by providing the appropriate ankle torque (in the sense of a force or load compensation). They are not used to bring the body to an upright position and therefore do not interfere with the kinematic control (which in the model is provided by the internal signals *bf*, *fs*, and the voluntary signal VOLUNT. LEAN).

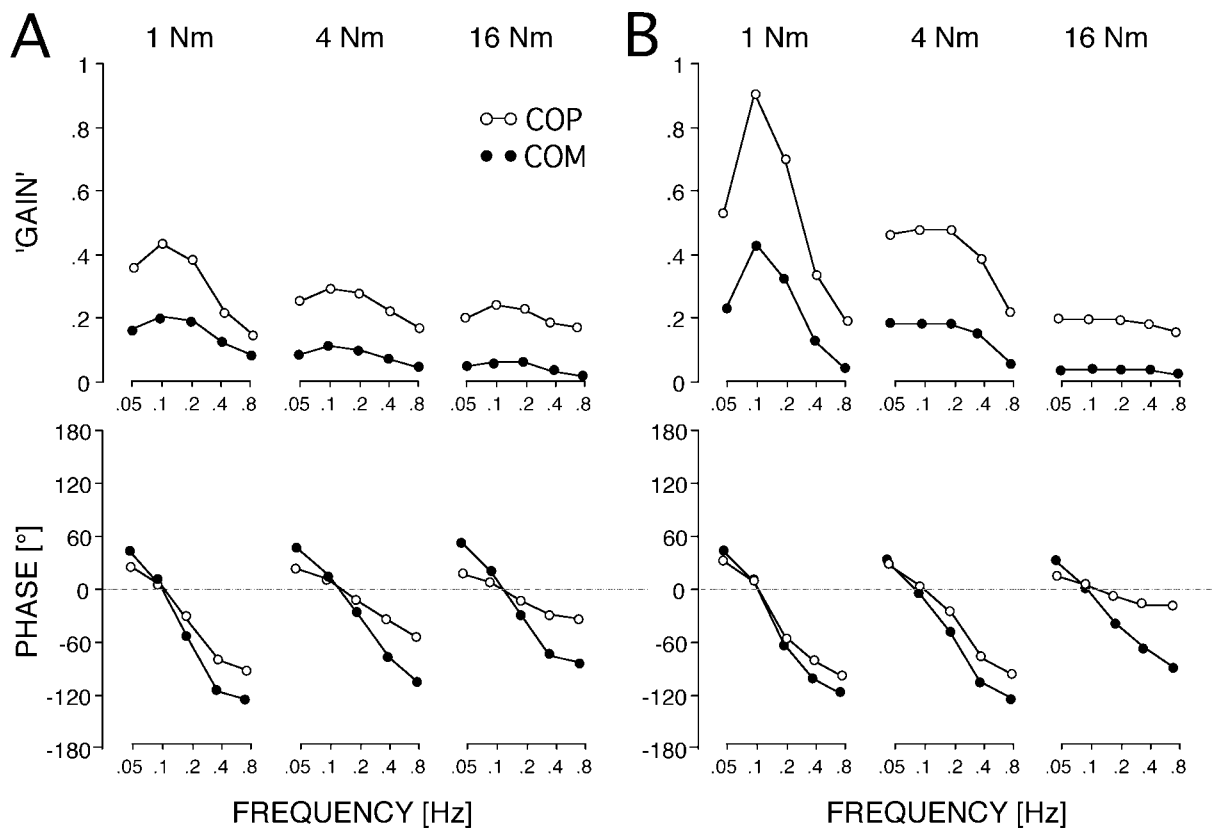


Fig. 5. Simulation results for the pull responses on the stationary platform of normal subjects (A) and vestibular loss patients (B), using the model shown in Fig. 4. Presentation as in Fig. 2 which shows the corresponding experimental results.

fact that they performed mental arithmetic during the trials and often did not perceive consciously the stimuli, especially the 1 N m stimuli). The possibility exists that prediction may have led to a reduction of the effective feedback time delay. Therefore, the choice of the time delay in the model might already be accounting for predictive effects. But this idea still has to be tested in future work. Future models may include a more complete representation of prediction, possibly including feed-forward pathways.

One major aim in our modeling was to establish a parsimonious description of postural control mechanisms which can cope simultaneously with all three external physical stimuli (tilt, pull, gravity) and with voluntary lean, while at the same time minimizing the need for a task-dependent adjustment of parameters (i.e. without explicit sensory reweighting). We were successful for conditions of pull and tilt stimuli on stationary support. In this case, the necessary

'sensory weighting' occurred implicitly because the external stimuli are internally implemented in an appropriate way. However, successful simulation of the condition of the pull stimulus with BSRP required additional parameter adjustments.

Another aim of the modeling was to establish a mechanism which successfully predicts changes that occur with a reduction of sensory information. We were successful at predicting the results for vestibular loss patients by disabling the VEST sensor in the model and the related sensor fusion mechanisms, and making a simple threshold adjustment. This model was also able to predict that vestibular loss patients are able to maintain stability during low-frequency tilt of the support surface (see Maurer et al., 2000). A future experiment is motivated by the prediction that loss of the SOMAT sensor should have a limited effect on stability (e.g. by evaluating patients with distal neuropathy of the legs).

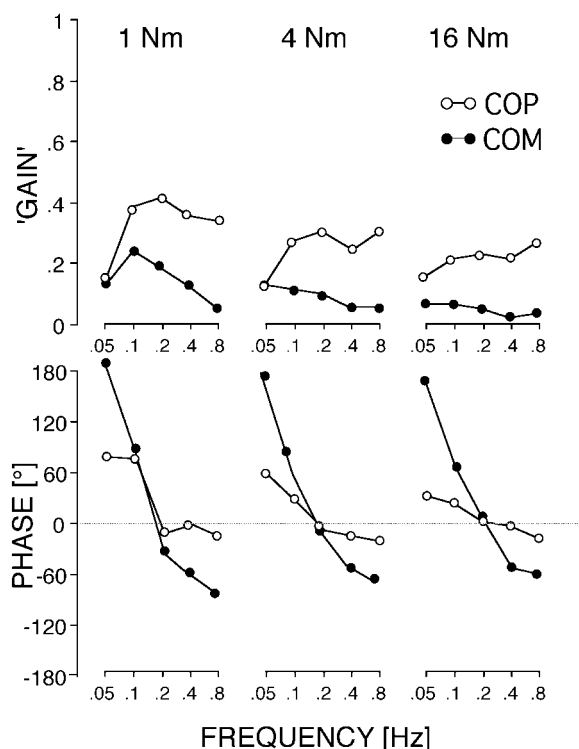


Fig. 6. Simulation results for the pull responses in the BSRP condition of normal subjects. The corresponding experimental results are shown in Fig. 3.

We are aware of the fact that many more receptor systems are involved in postural control, but consider the four sensors considered here as the essential minimum. We also learned from the modeling that many different model topologies yield essentially equivalent results, as long as they are implemented within the same basic framework (internal representations of the external stimuli, their use as set point signals of the local proprioceptive loop). We consider our modeling as a first and preliminary step in a novel approach whose aim is to understand the reasons for the high flexibility and robustness of the human postural control system. This approach clearly distinguishes it from otherwise similar work in the literature (e.g. Nashner, 1972; Johansson and Magnusson, 1991; Van der Kooij et al., 1999; Jeka et al., 2000; Peterka, 2002).

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