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A cognitive intersensory interaction mechanism in human postural control

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Abstract Human control of upright body posture involves inputs from several senses (visual, vestibular, proprioceptive, somatosensory) and their central interactions. We recently studied visual effects on posture control and their intersensory interactions and found evidence for the existence of an indirect and presumably cognitive mode of interaction, in addition to a direct interaction (we found, e.g., that a ‘virtual reality’ visual stimulus has a weaker postural effect than a ‘real world’ scene, because of its illusory character). Here we focus on the presumed cognitive interaction mechanism. We report experiments in healthy subjects and vestibular loss patients. We investigated to what extent a postural response to lateral platform tilt is modulated by tilt of a visual scene in an orthogonal rotational plane (anterior–posterior, a–p, direction). The a–p visual stimulus did not evoke a lateral postural response on its own. But it enhanced the response to the lateral platform tilt (i.e., it increased the evoked body excursion). The effect was related to the velocity of the visual stimulus, showed a threshold at $0.31^\circ/\text{s}$, and increased monotonically with increasing velocity. These characteristics were similar in normals and patients, but body excursions were larger in patients. In conclusion, the orthogonal stimulus arrangement in our experiments allowed us to selectively assess a cognitive intersensory interaction that upon coplanar stimulation tends to be merged with direct interaction. The observed threshold corresponds to the conscious perceptual detection threshold of the visual motion, which is clearly higher than the visual postural

response threshold. This finding is in line with our notion of a cognitive phenomenon. We postulate that the cognitive mechanism in normals interferes with a central visual–vestibular interaction mechanism. This appears to be similar in vestibular loss patients, but patients use less effective somatosensory instead of vestibular anti-gravity mechanisms.

Keywords Intersensory interaction · Postural control · Human · Vestibular loss

Introduction

Control of human upright stance is known to involve several sensory systems (vestibular, visual, proprioceptive, somatosensory; for overview, see Horak and Macpherson 1996). These inputs are known to interact with each other, but the way in which this is performed is still not fully understood to date. Previous work showed that, in behavioral situations which involve two or more sensory feedback signals, the postural responses can be described as resulting from summation of these signals (Peterka 2002). Summation of the signals in combination with central threshold-like mechanisms, furthermore, is thought to yield sensory re-weightings by which the system can cope with changing behavioral situations (Mergner et al. 2003, 2005; Maurer et al. 2005). In these concepts of intersensory interaction, there is a central representation of a given sensory input that, on its own, can elicit a postural response and, when combined with other inputs, directly affects their internal representations and thus their responses. We will refer to this in the following as ‘direct’ intersensory interaction and distinguish it from an ‘indirect’ interaction mode. With the indirect mode, information is derived from a sensory input, which does not evoke a postural response on its own, but modulates the responses to other sensory inputs (in terms of a parametric gain change, e.g., evoked by general information extracted from the stimulus and other factors such as high order cognitive mechanisms). In the

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following we explain this point by giving examples of these two interaction modes.

In a previous study (Mergner et al. 2005) we investigated visually evoked postural responses. Subjects were standing upright and viewed a visual scene that was sinusoidally rotated in the anterior–posterior (a–p) direction about an axis through their ankle joints. This visual stimulus evoked a body excursion in the direction of the stimulus. The excursion, in turn, activated vestibular, proprioceptive, and somatosensory responses which counteracted and thus limited the visually evoked excursion. This we take to represent a direct interaction of visual versus vestibular, proprioceptive, and somatosensory effects. However, we observed in these experiments also an indirect interaction. When we superimposed on the a–p visual stimulus a lateral tilt of the support surface (platform), the lateral tilt evoked a postural response in the lateral direction and no direct response in the a–p direction. But its presence led to an increase of the a–p visual response. We attributed this effect to a cognitive mechanism by which subjects shifted sensory weight such that they used information from the visual scene more than from the support surface for their postural stabilization. In another experiment, we evaluated the effect of the visual response after replacing the visual stimulus, which consisted of a real visual scene, by a ‘virtual reality’ scene. Then the visual response became reduced in magnitude. We attributed this effect again to a cognitive mechanism by which subjects weight the impact of the stimulus based on an estimate of its quality (e.g., its reality character). Parameter identification with the help of a dynamic postural control model suggested that the modifications of the visual response were due to a central strengthening and weakening of the visual signal, respectively.

In the present study we analyzed so far unpublished material of the previous study, focusing on the presumed cognitive contribution to the intersensory interaction. Being fascinated by the discovery that the platform tilt in a plane perpendicular to the a–p visual stimulus modulated the a–p visual response, we asked whether, vice versa, the a–p visual stimulus in these experiments would have a modulating effect on the lateral platform tilt response as well. Furthermore, we aimed to find support for our notion that the interaction effect is cognitive. We therefore analyzed the lateral tilt response, for which we had used only one stimulus parameter set (0.25 Hz sinusoid, $\pm 0.2^\circ$), as a function of the a–p visual stimulus, for which we had used a number of different stimulus magnitudes and frequencies. The findings obtained were compatible with our notion of a cognitive contribution to the intersensory interaction and gave us some insight into the underlying mechanism.

Methods

A detailed description of the methods and the set up is given in Mergner et al. (2005) and Blümle (2004). Eight normal subjects and three patients with bilateral loss of

vestibular functions participated (mean ages \pm SD, 34.4 ± 9.9 and 36.0 ± 1.4 years, respectively). Patients’ vestibular loss was assessed by clinical examination, electronystagmography, and case histories.

Set-up

Subjects stood upright on a motion platform inside a movable cabin that was used for presenting the visual motion stimuli (Fig. 1a). This set-up allowed us to apply platform tilt stimuli independently of the visual motion stimuli. The platform rested on six extendable motorized ‘legs’ by which it could be moved in the three rotational and three translational planes of space (hexapod with Stewart principle; see Stewart 1965). In the present experiments, we used it to present our subjects with a lateral platform tilt (frontal plane; axis of rotation at foot sole level). The stimulus was always sinusoidal at a frequency of 0.25 Hz and showed an amplitude of $\pm 0.2^\circ$.

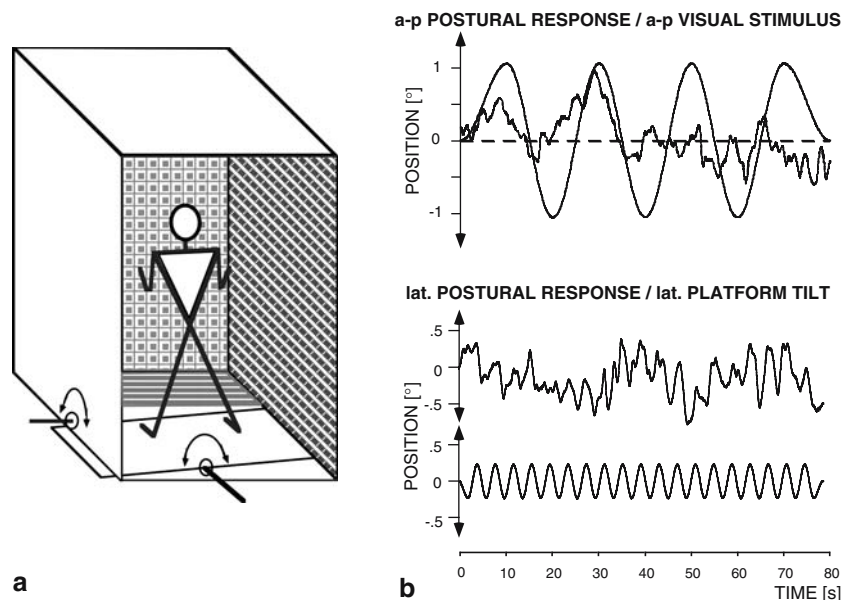
The cabin used for the visual motion stimuli measured $100 \times 80 \times 190$ cm³ (depth \times width \times height) and was illuminated on the inside. It had styrofoam walls that were covered by a wallpaper showing an optokinetic-like pattern with ornaments of various sizes, orientations, colors and brightness contrasts (subjects’ eye-to-pattern distance in primary position, 60 cm). The cabin was rotated in a–p direction by means of two computer-controlled cable winches (position error, $< 3\%$). The axis of rotation was at the level of the subjects’ ankle joints (so that the visual input which subjects received from the stimulus was essentially the same as that which normally arose during spontaneous a–p body sway about the ankle joints). The cabin rotations also were sinusoidal, but differed in frequency from the orthogonal platform tilt. The frequency was varied ($f = 0.05, 0.1, 0.2,$ and 0.4 Hz), as was the amplitude ($a = 0^\circ, \pm 0.25^\circ, \pm 0.5^\circ, \pm 1^\circ, \pm 2^\circ,$ and $\pm 4^\circ$).

The stimuli were presented in trials that lasted 80 s, with the number of sine wave cycles in each trial depending on stimulus frequency. Beginning and end of the visual and platform tilt stimuli were smoothed to avoid transients. The trials were presented in four different runs, one for each of the four frequencies of the visual stimulus (each run contained all stimulus amplitudes in randomized order). Subjects were instructed to always remain upright, keeping gaze straight ahead. With the hands held up at shoulder levels, they held ropes that hung loosely from room-fixed beams. They used the ropes as a safety set-up; when fearing to fall, they lowered their hands and put tension on the ropes.

Procedures and recordings

Six-D position measures of subjects’ hips, shoulders and heads, and of the platform and cabin were obtained using an optoelectronic device (Optotrak 3020,

Fig. 1 a Schematic presentation of stimulus condition. Subject is standing on a motion platform that is laterally tilted (i.e., in the frontal rotational plane) while a visual motion stimulus is presented in the anterior–posterior (a–p) direction (sagittal rotational plane). The visual stimulus is generated by rotating a cabin with an optokinetic pattern on the inside walls about an axis through subject’s ankle joints. **b** Examples of a subject’s postural responses (hip angular excursion) in the a–p direction evoked by the a–p visual stimulus ($f=0.05$ Hz; $a=\pm 1^\circ$) and the concurrent lateral responses evoked by the platform tilt ($f=0.25$ Hz; $a=\pm 0.2^\circ$)



Waterloo, Canada; spatial resolution, 0.2 mm). The data were analog–digital converted at a sampling rate of 100 Hz and stored on a computer hard disk for off-line analyses of a–p and lateral angular position values. Examples of an original trace of a–p hip and a–p visual scene angular excursions as well as lateral hip and lateral platform excursions from a normal subject are shown in Fig. 1b. Subjects’ a–p and lateral postural responses were quantified by calculating the amplitude (peak angular displacement) of the fundamental waves of the stimuli and the body excursions using fast Fourier transformation (FFT; we ascertained that the power of the response fundamentals were always larger than the first and second harmonics by a factor of 3 and 6, respectively). Corresponding phase values were taken from the temporal stimulus–response relationship between fundamentals (in degrees; positive values, lead). The first and the last cycle of each trial were not evaluated. Two cycles were evaluated for the 0.05 Hz trial, 6 for 0.1 Hz, 14 for 0.2 Hz, and 30 for 0.4 Hz.

Analyses were performed four times for each trial: Subjects’ a–p postural responses were related to both the a–p visual stimuli (1) and the lateral platform stimulus (2), as were their lateral postural responses, i.e., to the a–p visual stimuli (3), and the lateral platform stimulus (4). Gain and phase values of the a–p postural responses to the a–p visual stimuli and their modulation by the lateral platform tilts have been described previously (Mergner et al. 2005). Also reported in the previous study were the findings that gain of the a–p responses to the lateral platform stimulus and, vice versa, gain of the lateral responses to the a–p visual stimuli were essentially zero, which indicates that there was no considerable direct sensory or mechanical ‘cross-talk’ between the two orthogonal rotational plane systems. The present study solely presents findings on the lateral postural responses to the lateral platform tilt and their

modulation by the a–p visual stimulus. The data is presented in the form of ‘grand averages’ across subjects, with the error bars representing inter-individual variability (1SD).

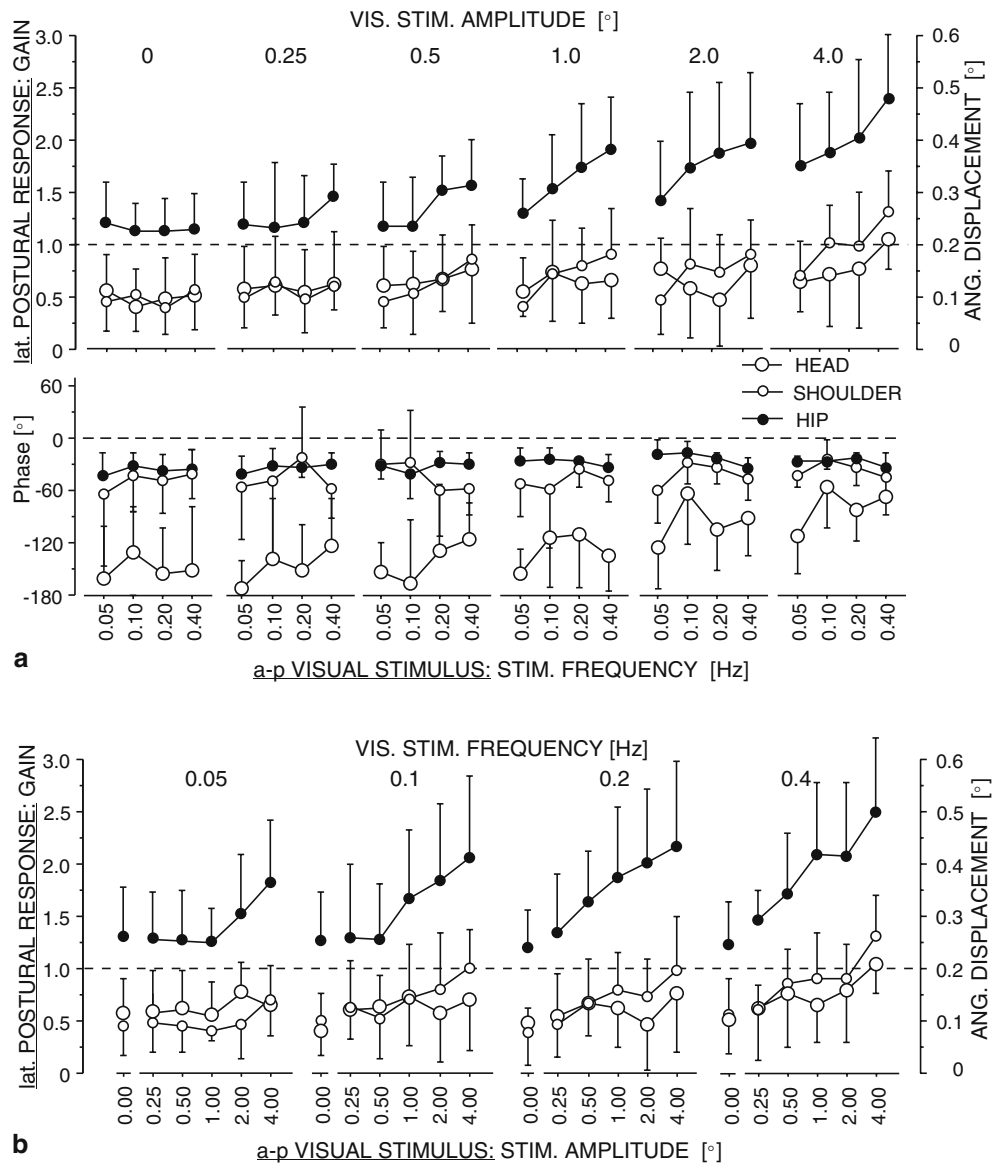
Results

Normal subjects

Figure 2a shows the results for the lateral postural responses to the lateral platform tilt (0.25 Hz, $\pm 0.2^\circ$) as a function of stimulus frequency of the a–p visual motion, separately for the five different amplitudes of the visual stimulus (0° to $\pm 4^\circ$). In the upper panels, gain can be read from the left ordinate and angular excursion from the right ordinate, while the lower panels give response phase. In each panel we superimposed the frequency response curves for the hip, the shoulders, and the head. We first consider the situation with stationary visual scene (0° stimulus; left most panel), which we then use for comparison when assessing the question how the a–p visual motion affects posture control in the lateral rotational plane in which it does not evoke a direct response.

Consider first the gain values taken from the 0° trial of the 0.05 Hz visual stimulus run. Hip excursion slightly exceeds platform excursion (gain ≈ 1). This response reflects the net result of the passive mechanical excursion, plus a gravitational effect that tries to accelerate the body further in the direction of its tilt, plus passive and active counter-torques produced in the leg and hip joints. The active counter-torque is not enough to keep the lower body segment upright in space (note that a perfect compensatory response, i.e., a full righting, would yield zero gain, i.e., no hip excursion in space during the platform tilt; pronounced under-compensation is typical

Fig. 2 Lateral sway response to lateral platform tilt as a function of stimulus frequency (a) and amplitude (b) of the a-p visual scene motion in normal subjects. Presented are mean gain and phase curves of hip, shoulder, and head angular excursions (additional ordinates on right hand side give absolute values of the excursions; vertical lines, 1SD values). Phase curves in b are omitted for simplification. Dashed horizontal line in upper panels give unity gain, representing an evoked excursion equal to platform tilt



with the very small platform tilt used, see Maurer et al. 2005). Shoulder and head excursions are approximately half of the hip excursion. Shoulder excursions are almost in phase with hip excursions, showing similar phase lags. Thus, there is a compensatory upper body righting by about half the platform tilt amplitude. Head excursion is almost in counter-phase to the tilt stimulus and to the hip and shoulder excursions, indicating some over-compensation. The effects obtained from the other three trials (those for the 0.1, 0.2, and 0.4 Hz runs) were essentially the same as with the 0.05 Hz run.

In the trials in which the a-p visual motion stimulus had an amplitude of $\pm 0.25^\circ$ (next rightward panel), the responses were essentially the same as before, indicating that the stimulus yielded no considerable effect. This applied to the 0.05, 0.1, and 0.2 Hz visual stimuli, whereas a slight gain increase occurred with the 0.4 Hz stimulus. The response characteristics were similar with the $\pm 0.5^\circ$ stimulus amplitude, but the gain increase

occurred already at 0.2 Hz and rose further at 0.4 Hz. This tendency continued with the $\pm 1^\circ$ stimulus amplitude (gain increase starting now at 0.1 Hz), with the $\pm 2^\circ$ amplitude (gain increasing already at 0.05 Hz and rising further with increasing frequency), and with the $\pm 4^\circ$ amplitude (as before, but gain increase starting at a higher level). These effects were quite prominent with the hip excursion and less prominent with the shoulder and head excursions. In contrast, the phase of the lateral platform tilt response was not affected to a considerable degree by the a-p visual motion stimulus (exception, being that the phase of the head response became similar to the phase of the shoulder response with increasing visual stimulus frequency and amplitude).

Thus, lower and upper body stabilizations upon small lateral platform tilts were less effective in the presence of a moving visual scene as compared to a stationary visual scene. The destabilizing effect appeared to increase not only with increasing frequency, but also with increasing

amplitude of the visual motion stimulus. This notion was confirmed when we re-plotted the data as a function of stimulus amplitude, separately for the four frequencies of the visual stimulus (Fig. 2b; phase curves omitted for simplification).

The finding of a combined dependency of the lateral response on frequency and amplitude of the a-p visual stimulus suggested that the effect is mainly related to stimulus velocity (because with sinusoidal stimuli, velocity co-varies with both frequency and amplitude). We therefore re-plotted the data once more after averaging the gain (and amplitude) values across all subjects and across all stimuli that had the same peak velocity. The results are given in Fig. 3 which shows gain (amplitude) and phase of the lateral tilt response as a function of peak angular velocity of the a-p visual stimulus (only hip data are shown for simplification). The figure shows that gain increases with increasing peak velocity of the a-p visual stimulus. This applies to peak velocity values $> 0.31^\circ/\text{s}$, whereas at and below this value the lateral postural tilt response is essentially the same as with a stationary visual scene ($0^\circ/\text{s}$). The phase of the lateral response shows hardly any dependency on the a-p visual stimulus.

Vestibular loss patients

The characteristics of patients' responses were similar to those of normal subjects, apart from higher gain values. The lateral platform response of the hip is shown in

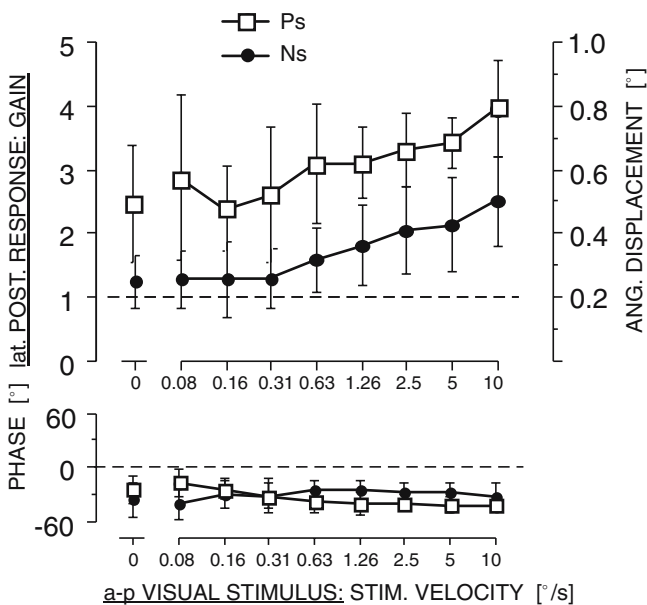


Fig. 3 Lateral sway response to lateral platform tilt as a function of peak velocity of the a-p visual scene motion in both, normal subjects (*Ns*) and vestibular loss patients (*Ps*). Replot of mean gain and phase values shown in Fig. 2 (vertical lines, 1SD values). Note that the gain and phase curves, which were presented in the different panels of Fig. 2a, b for stimulus frequency and amplitude, here are collapsed into one panel where the variable of the sinusoidal visual stimulus is velocity

Fig. 3 as a function of peak velocity of the visual stimulus, superimposing the gain and phase curves on those of the normal subjects. Note that gain is approximately double that of the normal subjects in the situation with stationary scene ($0^\circ/\text{s}$ stimulus). This suggests that patients' counter-action to the gravitational effect was abnormally weak. In contrast, the phase of the response was essentially normal. Similar responses were obtained in the presence of the a-p visual scene motion at and below a peak velocity of $0.31^\circ/\text{s}$. Above this value, the gain curve increased essentially in parallel to that of normals, while the phase still showed no considerable change (apart from a slightly larger lag than in normals).

Discussion

In a previous postural control study we investigated the effects of visual motion (Mergner et al. 2005). The visual stimulus evoked body excursions which, in turn, activated vestibular, proprioceptive, and somatosensory cues. We found that the impact of the visual signal was strongly suppressed in this condition (i.e., gain is very low in situations where the visual scene is perceived as moving as compared to situations where the scene is perceived as stationary; see below). We relate this finding mainly to a visual-vestibular interaction mechanism and speculate that the one in posture control is similar to the one which we previously described for human self-motion perception (Mergner et al. 2000). This mechanism brings about that the visual input is used instead of the vestibular input in situations where the visual scene is perceived as stationary (it is then taken as a space reference), while the visual input becomes suppressed and the vestibular input prevails when the scene is perceived as moving. As specified further below, we conceive that cognitive factors contribute to this interaction (cognitive, in the general sense that 'knowing' of scene motion, be it by perception, memory retrieval, etc. is the relevant factor). A possibly related cognitive effect on posture control has recently been reported by Guerraz et al. (2001). These authors reported that explicit prior knowledge of visual motion largely suppressed the postural response which otherwise would be evoked upon unexpected visual motion. This cognitive effect appears to be clearly distinct from more general aspects of cognition such as attention, mental load, etc., which also repeatedly have been investigated in relation to postural control (e.g., Riley et al. 2003; Redfern et al. 2004).

As already mentioned in Introduction, our previous work on visual postural responses (Mergner et al. 2005) led us to conclude that cognitive mechanisms modulate the gain of the central visual signal. Furthermore, it suggested that the effect can consist of either a reduction or an enhancement of the gain of the visual signal. The present findings now allow us further characterizations. They show that not only is lateral platform tilt able to modulate the a-p visual postural response, but, vice versa, also an a-p visual motion stimulus can modulate the lateral

platform tilt response. Furthermore, they led us to identify the effective visual signal as being mainly a velocity signal. Finally, we found that the cognitive effect does not occur unless peak velocity of the visual stimulus exceeds $0.31^\circ/\text{s}$. Above this value the effect increased monotonically with increasing peak velocity. Interestingly, the $0.31^\circ/\text{s}$ value corresponds to the detection threshold at which our subjects consciously perceived the visual motion, in line with the literature (see Mergner et al. 2005). This is different from the direct (a–p) postural responses to the visual stimulus, which were evoked even at the lowest stimulus velocity used ($0.08^\circ/\text{s}$). The finding supports our notion of cognition being involved, in that it allows us to relate the effect to conscious perception and ‘higher mental processing’ levels where knowledge or expectations are drawn from memory, etc. (see above, ‘knowledge’).

The notion of a cognitive effect is further supported by the finding that it occurred in a rotational plane where the visual stimulus did not evoke a direct response. It appears that the brain was generalizing from perceived a–p motion of the visual scene that a moving scene may not represent a reliable space reference for postural stabilization in any plane (therefore placing relatively less weight on the visual information). It is this particular feature of an effect across orthogonal stimulus planes which allowed us to separate the cognitive effect from direct intersensory interaction effects. Interestingly, in the above mentioned study of Guerraz et al. (2001) the cognitive effect also was independent of the direction of the visual stimulus.

In a later study, Guerraz and Day (2005) doubted whether cognitive effects on posture control involve the vestibular signal. The reason is that they found that postural responses to predictable galvanic vestibular stimuli are essentially similar to unpredictable ones. We conceive, however, that the vestibular response here is particular, in the sense that a galvanic vestibular stimulus is not well represented as a predictable external stimulus at higher (cognitive) brain centers. In this respect, one may ask whether the abnormally high gain of the responses of our vestibular loss patients can be taken to indicate a missing cognitive effect due to the missing vestibular input. We think that this is not necessarily so. The reason is that patients’ response characteristics were similar to those of normals. Their high response gain may simply indicate a too weak compensatory torque to platform tilt. The reason could be that they use a somatosensory graviception from plantar sole pressure cues instead of vestibular graviception and/or that they tend to keep their bodies aligned with the platform vertical instead of the space vertical (see Maurer et al. 2000).

How does the cognitive effect work? We know very little about it, at present, but would like to refer—as a hypothesis—to our model of visual–vestibular interaction in human self-motion perception (Mergner et al. 2000). There we distinguished a basic interaction mechanism, consisting of a summation of visual and vestibular signals and a central threshold, from a ‘higher level’

mechanism that measures dynamic visual–vestibular conflict. High conflict is taken to result from visual scene motion and to prevent, in a graded way, that the visual cue is taken as a reference for spatial orientation. This is performed by means of a parametric gain modulation (a throttle-like mechanism) which receives the dynamic conflict signal as modulating input, and long-lasting modulating inputs may contribute as well. An important aim, for which the whole interaction mechanism is used, is to reduce the dependency of spatial orientation on the vestibular signal, whenever this is possible (true when visual scene is stationary), because the vestibular signal is very noisy (see Mergner et al. 2001). We conceive that the vestibular noise represents a problem also with posture control and that this may be a reason for involving cognitive effects. Sensory re-weighting by means of a throttle-like mechanism likely is not restricted to visual–vestibular interaction, but appears to be involved in vestibular–proprioceptive interaction as well, a notion that still has to receive experimental support, however. Interestingly, also patients appear to suppress the visual signal when they perceive the scene as moving. They tend to use a somatosensory substitute for the vestibular signal (Maurer et al. 2000), which is prone to lead to positive sensory feedback and thus may lead to postural instability, however (see Maurer et al. 2005), so that its substitution by visual input may be a desired aim, as well.

How does the present findings relate to our previous study on visually evoked postural responses of normal subjects? As mentioned in [Introduction](#) we attributed the lateral tilt effect upon the a–p visual response to a cognitive mechanism by which subjects shifted sensory weight such that they used information from the visual scene more than from the support surface for their a–p postural stabilization (this notion was supported by the finding of a gain increase of the visual a–p signal in the parameter identification procedure we used). Interestingly, the lateral tilt effect on the visual response was largest with small and slow visual stimuli and became relatively smaller with large and fast visual stimuli. In view of the present observations, we attribute this to the presumed cognitive effect by which the weighting of the visual signal becomes reduced with visual stimulus peak velocities $> 0.31^\circ/\text{s}$. This, in turn, affects also the lateral tilt response. The way in which this effect is produced remains to be elucidated. At present, our hypothesis, based on plausibility, would be that subjects derived from the scene also a visual signal in the lateral direction and, when perceiving the scene as stationary, used it to compensate for insufficiencies of the vestibular input.¹ If cognition starts to decrease the visual gain, the insufficiency of the vestibular signal and of its antigravity effect in posture control becomes more and more evident.²

¹Here, with the small tilt stimuli used, the gain of the vestibular signal would be too low due to the fact that this signal has to surpass a detection threshold.

²The proprioceptive body-on-platform stabilization alone is not enough to counteract the gravity effect during support tilt.

Finally, we like to come back to our previous concept according to which postural control is governed by internal estimates of physical stimuli rather than directly by the sensory signals (Mergner et al. 2003; Maurer et al. 2005). By the same token, we assume that the cognitive signals are not identical with the sensory signals, but rather represent sensory-derived and knowledge-derived estimates of the kinematic state of external references such as the visual scene or the body support surface scaled by some measure of their validity. An intuitive understanding of this notion may be obtained by imagining that one intends to enter a room in which the floor is unstable. The information at the cognitive level would be “unstable body support surface” rather than “there is an unreliable or erroneous vestibular and proprioceptive signal”. Generally, the scaling of the cognitive signal may be caused by a broad spectrum of phenomena which also may include an impoverished information content or increased noise due to peripheral or central nervous diseases.

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