

The Role of Somatosensory Input for the Perception of Verticality

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INTRODUCTION

In the absence of a visual frame of reference, a normal subject is able to set a line to the vertical accurately, within 1 or 2 deg, when sitting upright (subjective visual vertical, SVV)¹. He perceives uprightness of his body within an inverted cone with a base of about 5 deg diameter, when passively tilted in the roll and pitch planes at low velocity.² When normal subjects sitting in a motor-driven gimbal are asked to actively set themselves to vertical by means of a joystick control, their judgments deviate on average 1.7 deg from the true gravitational vertical.³

Aubert was the first to describe an apparent systematic tilt of the SVV depending on the orientation of the head.⁴ When the head (or whole body) is tilted to one side, a vertical line appears to the observer tilted to the opposite side, so that, when asked to set it vertical, he rotates its upper edge toward the side he is tilted (A-effect). This effect has been reported to be absent after complete proprioceptive sensory loss below the neck due to polyneuropathy.⁵ While patients with acute unilateral vestibular lesions show strong deviations of the SVV toward the lesion side, their postural vertical judgments remain veridical.^{2,3} Thus, conflicting orientation responses to the gravity vector can coexist, depending on the multiple sensory inputs and processing structures involved. In fact, the contribution of each system (visual, vestibular, proprioceptive) is not yet fully understood.

In the following we assessed the contribution of the somatosensory system to the perception of verticality by asking patients with various degrees of sensory disturbances to set the SVV in upright (sitting) position and when lying sideways. The perception of the subjective postural vertical (SPV) was also evaluated.

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METHODS

Apparatus

Subjects sat upright in a chair located inside a spherical cabin, with the head positioned in the center of the sphere and the torso restrained in vacuum cushions. The cabin was supported by the inner ring of a four-axis gimbal system.⁶ Appropriate positioning of the middle ring, which could be rotated around an axis perpendicular to that of the outer ring, allowed tilts of the subjects about a naso-occipital axis (i.e., in the roll plane). A 10-cm-long luminous line, generated by a laser, could be projected at eye level on the cabin wall in front of the subject. Both the cabin and the target line could be rotated in the roll plane either by the subject by means of separate joysticks or by the experimenter.

Procedures

The SVV was determined by means of six adjustments from a random angular offset of the target line (variable up to ± 40 deg from earth vertical) in darkness. The subject was required to set the line "so that it appears to be vertical." The angular deviation from the true vertical in degrees was measured by a potentiometer and read by the experimenter. SVV estimates were repeated after tilting the chair 80 deg in the roll plane randomly to the right and left. The SPV was determined by 12–16 adjustments of the position of the cabin by the subject so that he "feels his body is vertical" after a random offset in the roll plane of ± 5 , 15, or 25 deg. Acceleration and deceleration of the stimuli used to offset the subjects was 10 deg/s², thus resulting in triangular velocity profiles with maximal velocity of 7.0, 12.1, and 15.5 deg/s respectively. Subjects responses were self-paced, with an upper velocity limit of either 2 deg/s or 3 deg/s randomly set when subjects were adjusting themselves to earth vertical. This procedure guaranteed the absence of time cues for repositioning to upright. Measurements were determined manually from the computer-stored displacement traces.

Subjects

Eighteen patients with acute and chronic somatosensory disturbances (3 with brain and spinal cord tumors, 2 with cerebral haemorrhage, 6 with cerebral and spinal ischaemia, 4 with a first episode of demyelination, and 3 with polyneuropathy) were compared with 20 age-matched normal subjects (mean age 50.2, SD 10.8 years). Not all patients could be tested in both paradigms. In two patients the sensory loss was extreme, including complete unilateral loss of position sense. The cause was an acute cerebral hemisphere infarction (FIG. 1A and 1B); only SVV estimates were obtained from these two patients. Their tilt estimates were obtained lying in bed and adjusting a luminous line presented on a computer screen by means of a hand-held trackball.

RESULTS

Subjective Visual Vertical

In the upright position, the SVV estimates of the normal and patient groups were similar (1.9 SD 1.4 deg; 1.9 SD 1.7 deg, respectively; unsigned values). Only one patient with

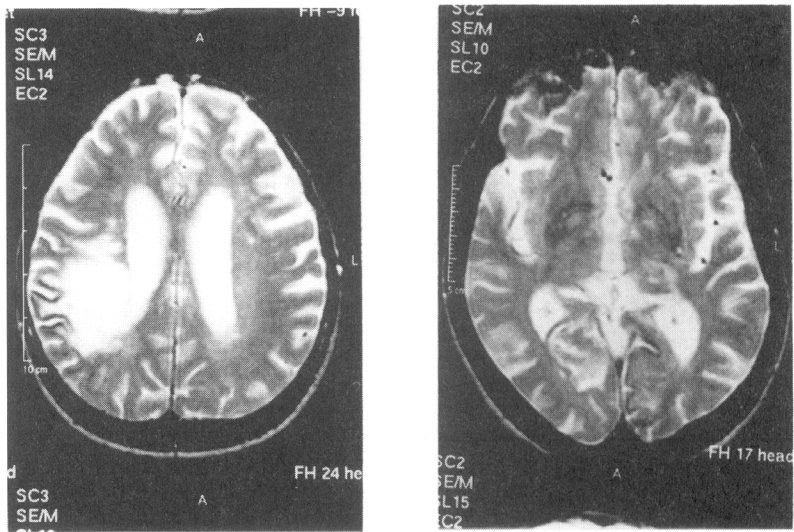


FIGURE 1. MRIs of the two patients lacking A-effect on the SVV when lying on the hypoesthetic side, showing ischemic infarctions in the right parietal lobe (A) and left thalamic region (B). The visual vertical data of these patients is shown in FIGURE 2, with symbols ▲ and ★, respectively.

complete hemisensory loss due to a parietal infarct (FIG. 1A) showed a marginal ipsiversive tilt of 4.9 deg (FIG. 2A). In the lateral body position, SVV settings of normal subjects deviated in the direction of body tilt (normal values—16.5 SD 10.4 deg and 14.2 SD 7.8 deg for left- and right-tilted position, respectively). In the two patients with almost complete hemisensory loss the A-effect was essentially abolished when lying on the hypoesthetic side (FIG. 2B). The remaining had SVV settings deviated in the same direction as body tilt by a normal amount (i.e., normal A-effect).

Subjective Postural Vertical

Only in 2 patients with moderate hypesthesia was there a bias of the SPV, toward the hypoesthetic side. One of them had an acute infarct of the right internal capsule (SPV–5.3 deg) and the other a right temporoparietal glioblastoma (SPV–4.6 deg) (FIG. 2A, showing unsigned data). They were the two most severely hypoesthetic patients tested in the SPV task. As group data, SPV setting from the normal subjects (1.6 SD 1 deg) and patients (2.1 SD 1.5 deg) were not different (unsigned values).

DISCUSSION

Our patients with sensory loss did not show significant tilts of the SVV in the upright, seated position. In contrast, while lying sideways, unilateral sensory loss completely abolished the apparent displacement of the SVV in the opposite direction of body tilt in our

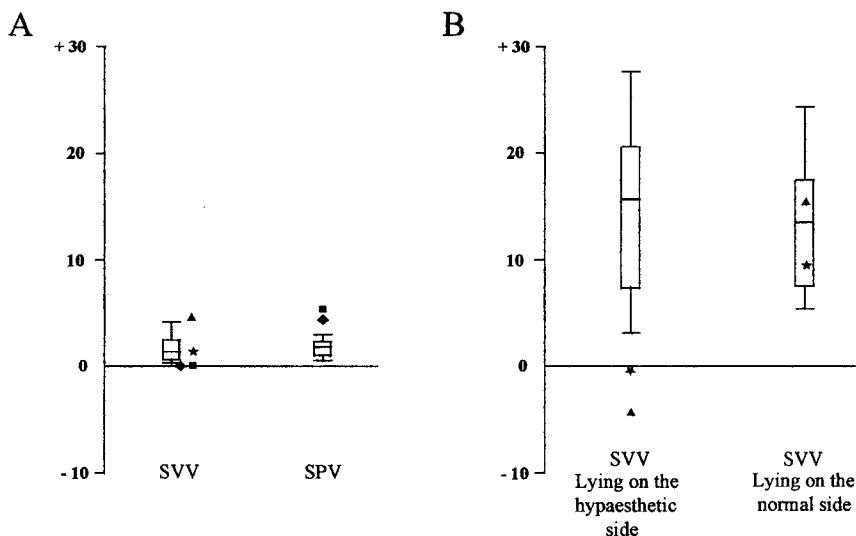


FIGURE 2. (A) SVV in the upright position in 4 patients with moderate to severe hemihypoesthesia, and SPV in 2 of these patients in whom it could be tested. Normal control data shown are medians plus 75th and 90th percentiles. All data points are unsigned. (B) Visual vertical settings in two patients with severe hemianesthesia when lying on the normal and anesthetic side. Median, 75th and 90th percentiles of the normal group are shown, arbitrarily taking left side down for control of the hypoesthetic side. These latter values for normal controls and patients have been inverted to facilitate comparison with the unaffected side. Note absence of the A-effect in the patients when lying on the hemianesthetic side.

two patients with parietal and thalamic infarcts. It appears that the origin of tilt-mediated effects on the visual vertical (A-effect) is primarily somatosensory, not otolithic. Consistent with this hypothesis are the findings of symmetrical A-effect in patients with unilateral peripheral vestibular disease⁷ and the abnormally large A-effect in patients with bilateral absence of vestibular function.⁸ An additional patient with a long-standing thalamic infarct previously tested also showed an absence of A-effect when lying on the hypoesthetic side (Anastasopoulos and Bronstein, submitted manuscript), as well as a patient with a severe polyneuropathy.⁵ The findings in patients with hypoesthesia have the general implication that somatosensory input is capable of influencing the visual perception of verticality. However, this influence seems to come into play only during body tilt, as documented by the finding that even patients with acute hemihypoesthesia do not show significant tilts of the visual vertical while upright. The functional significance, if any, of the displacement of the SVV during lateral tilt (A-effect) is unknown. Mittelstaedt explains its occurrence as secondary to an internal drive that tends to rotate the SVV in the subject's main body axis (the idiotropic vector; Mittelstaedt, this issue). It would be reasonable to assume that such a body-centered vector relies on symmetric somatosensory input and that severe anesthetic lesions could have a profound influence on its magnitude or orientation.

It has been concluded that the SPV, at least while sitting with the body strapped to the seat, is overwhelmingly determined by somatosensory input, so that even acute vestibular imbalance does not bias it.^{2,3} Only two patients with moderate to severe hemihypoesthesia showed a bias towards the side of the sensory loss. Our results are also of significance in

showing that mild to moderate unilateral sensory losses are not sufficient to produce a bias of the SPV. It is likely that the remaining somatosensory, and possibly otolithic, information is able to counteract any bias induced by unilaterally reduced contact cues. It could be argued that the lesions in these patients may have interfered with central vestibular pathways or cortex leading to abnormal perception of the SPV, as it is known to occur with the SVV.^{9,10} Similarly, the loss of the A-effect on the SVV might have arisen from interference with central vestibular processing by the lesions. However, the absence of vestibular symptoms and signs as well as the normal visual vertical settings while upright strongly argue against this possibility. On a neurological basis, the findings reported here for both the SVV and the SPV can only be attributed to severe asymmetry in the somatosensory system.

For a comment on our article, see the paper by Mittelstaedt on page 334 of this volume.

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