How the eyes move the body

Abstract—The increased postural sway of patients with disorders of the vestibular system improves with vision. The suppression of pathologic nystagmus also reduces sway. Because the latter effect cannot be attributed to retinal slip as a relevant feedback for postural control, the authors investigated how eye movements rather than retinal slip affect balance. They found that slow eye movements increase sway, possibly by an efference copy, which explains why spontaneous nystagmus causes postural imbalance.

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Balance control depends on the integrity of various sensory and motor systems. Increased body sway is found in patients with vestibular, proprioceptive, and cerebellar disorders. However, if visual input is sufficient, the postural stability of most patients is restored to normal levels. This can easily be demonstrated in healthy subjects by having them close their eyes while assuming an unstable posture, e.g., with feet in the heel-to-toe position.

What is the visual cue for postural stabilization? Comparing posture in darkness and posture with vision of stable space-fixed targets or with full-field vision revealed a significant increase in stability with vision.¹ Large-field moving visual scenes were found to cause body sway in the same direction as stimulus motion.² Only a few studies^{3–5} have tried to determine which visually derived signal may be used for the motor control of body sway. On the basis of the available experimental data, it is commonly believed that retinal slip, i.e., target or background motion on the retina, is the afferent signal that determines visually evoked postural responses. However, during fixation of a space-fixed target, retinal slip is minimized by the vestibulo-ocular reflex and visual smooth pursuit. Therefore, eye movement rather than retinal slip reflects head motion in space. Is it conceivable that eye movement signals, efferent or reafferent, are used for the motor control of postural sway? Here we provide evidence that slow eye movements in the absence of substantial retinal slip can significantly affect postural stability.

Methods. Healthy subjects (six women, nine men; aged 22 to 45 years) standing heel-to-toe on a force-measuring platform (Kistler 9284) were asked to fixate a target spot on a translucent screen 0.7 m in front of them (lateral field of view 94°). Eye and head movements were monitored by video-oculography (lateral field of view of goggles 74°) and head position tracking (Intersense IS-600). After giving their informed consent, the subjects were asked to stand as stable as possible during nine randomized conditions. Fixation conditions included the following: standing in complete darkness and looking straight ahead (F1); fixation of a stationary target in darkness (F2) and fixation of a stationary target on a moving background (F3). Eye movement conditions were as follows: pursuing a moving target in darkness (E1); pursuing a moving target on a stationary background (E2); and pursuing a target and background moving together (E3). Eye-head movement conditions (H1 through H3) were the same as E1 through E3 except that subjects were instructed to follow the target with combined eye-head movements. Background (vertical black and white stripes, each 3° wide) and target motion were sinusoidal (12° amplitude, 0.33-Hz frequency) and computer controlled. Each trial lasted 15 seconds. It was preceded by a minimum of 5 seconds of rest, during which the stable target spot was presented, and was followed by 5 seconds without a visual stimulus.

Recorded data (100-Hz sampling rate) were analyzed off-line (MATLAB, The Mathworks, Natick, MA) to yield the root-mean-square of the center of pressure of the feet (RMS) for lateral sway. One subject had to be excluded as an outlier for the subsequent statistical analysis, because he almost fell during condition E1. For statistical analysis (repeated-measures analysis of variance, Statistica 6.1), conditions were grouped in a 3 × 3 design with the two factors gaze (fixation, eye movement, eye-head movement) and visual reference (no visual reference, space-fixed visual reference, moving visual reference).

Results. Figure 1 shows individual eye movement and lateral sway data for condition E1 from one subject (see figure 1, A and B) and the median over all subjects (see figure 1, C and D). Analysis of the RMS sway showed significant effects of gaze [$F(2,26) = 20.4$, $p < 0.0001$] and of reference [$F(2,26) = 15.8$, $p < 0.0001$] without interaction (figure 2). The effect of gaze showed that subjects swayed significantly more when performing smooth pursuit eye or eye-head movements (mean $\pm$ SEM: eye, 0.96 ± 0.04 cm; eye-head, 1.08 ± 0.05 cm) than when fixating a stationary target (0.76 ± 0.04 cm). In addition, a space-fixed reference (target or background; sway 0.82 ± 0.04 cm) provided sufficient visual input to reduce sway as compared with no such reference (1.02 ± 0.04 cm) or a moving reference (background; 0.96 ± 0.04 cm).

Discussion. The present data show that slow gaze movements, performed by the eyes alone or by combined eye-head movements, can induce significant body sway. Our experiment can easily be duplicated on oneself by trying to balance while standing heel-

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Figure 1. Eye position (black), desaccaded slow phase velocity (gray; A and C), and lateral sway (displacement of the center of pressure; B and D) for the smooth pursuit condition (E1) over time (stimulus presentation: 5 to 20 s). (A and B) Eye movement and sway for one subject. (C) Median eye movement for all subjects. (D) Median sway (gray) and minimum least squares fit (black) of slow phase velocity (5 to 7 s) and eye position (7 to 20 s). After stimulus onset (5 to 7 s), sway was best fitted by slow phase velocity. Multiple regression showed that neither retinal error nor retinal slip contributed significantly to the sway response.

to-toe and pursuing a swinging pendulum with the eyes. Our results cannot be explained by net retinal slip. Pursuit of a target in darkness caused more body sway than fixation of a stable target. In both cases, retinal slip was similarly low. In contrast, pursuit over a stable background caused less body sway than pursuit in darkness, and overall retinal slip was much higher with a stable background. Because the effects of eye and eye-head motion on postural stability were similar, we hypothesize that a gaze signal is of considerable importance for stabilizing balance. Such a signal is a better indicator of head and body motion in space than retinal slip when fixating a space-fixed target. Therefore, it could be used as input for the postural control system.

Several previous findings involving visual influ-

ences on body sway can now be reinterpreted. Smooth pursuit had already been shown to induce body sway, which increased with pursuit amplitude.⁶ This was attributed to increased thresholds for motion detection while moving the eyes. Body sway has been shown to be reduced, the closer a visual space-fixed target is to the eye.¹ This was interpreted by arguing that a nearby target induces greater retinal slip for equal body sway. Body sway increased together with nystagmus intensity in patients with pathologic downbeat nystagmus while the patients tried to fixate an eccentric target.⁷ Because both sway and nystagmus could be driven by the same vestibular input, this result was taken as support for the hypothesis that downbeat nystagmus is a vestibular syndrome, an assumption that has recently been questioned.⁸

All of the above findings can also be explained by the direct influence of eye movements on postural control. An influence of static eye position, even with the eyes closed, on body sway during external perturbation has already been demonstrated.⁹ Further support comes from the finding that visual suppression of pathologic nystagmus by a head-fixed target reduced body sway in patients with acute vestibular neuritis.¹⁰ Because a head-fixed target cannot be used for postural stabilization, the reduction of postural sway must have been caused by the decrease in eye movement amplitude. Thus, in addition to the well-known influence of movement of the visual scene on balance control, we demonstrated that eye movement signals have a direct influence on postural control: the eyes move the body. The impaired balance control of patients with increased body sway and nystagmus may thus be caused solely by ocular drift due to nystagmus, rather than being indicative of additional sensory or motor dysfunction.

Figure 2. Mean lateral root-mean-square (RMS) sway data (in centimeters) for all conditions. The pictograms on top illustrate the experimental conditions (see Methods; F = fixation, E = eye movement, H = eye-head movement). The bars give 95% confidence intervals of means. Regardless of the visual reference, conditions with eye movements (eye pursuit; squares) or combined eye-head movements (head pursuit; diamonds) caused larger body sway than when fixating or attempting to fixate a stationary target (circles). Thus, slow gaze movements increased body sway in comparison with target fixation, even in the presence of a space-fixed background as reference.

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A synovial cyst in the cervical spine causing acute spinal cord compression

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An 86-year-old man, previously independently mobile, presented with weakness in all limbs and fecal incontinence. He had fallen the previous day and 4 hours later developed progressive weakness in his legs. Examination confirmed a flaccid tetraparesis with a sensory level for pinprick sensation to C7 bilaterally and absent anal tone.

MRI scan revealed marked spondylosis and a synovial cyst at the level of C4 to C5, causing severe compression of the spinal cord. A cervical decompression was performed at C4/C5/C6, which confirmed the diagnosis. Postoperatively the patient regained full power in his right arm and leg and minimally reduced power in his left arm, but a moderate power deficit persisted in the left leg.

A presumed flexion–extension injury at the time of the fall has resulted in acute cord compression from a previously asymptomatic synovial cyst (figure).

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Figure. T2-weighted sagittal MRI of the cervical spine, showing a circumscribed, high signal intraspinal but extradural mass (arrow) causing severe compression of the spinal cord at the level of C4 to C5. The appearances are consistent with a synovial cyst arising from a facet joint in the cervical spine.

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