EFFECT OF HEAD ORIENTATION ON HUMAN POSTURAL STABILITY FOLLOWING UNILATERAL VESTIBULAR ABLATION

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Abstract — Effective interpretation of vestibular inputs to postural control requires that orientation of head on body is known. Postural stability might deteriorate when vestibular information and neck information are not properly coupled, as might occur with vestibular pathology. Postural sway was assessed in unilateral vestibulopathic patients before and acutely, 1, 4, and 18+ months after unilateral vestibular ablation (UVA) as well as in normal subjects. Postural equilibrium with eyes closed was quantified as scaled pk-pk sway during 20 s trials in which the support surface was modulated proportionally with sway. Subjects were tested with the head upright and facing forward, turned 45° right, and 45° left. Equilibrium was uninfluenced by head orientation in normal subjects. In contrast, patients after UVA showed both a general reduction in stability and a right/left head orientation-dependent asymmetry. These abnormalities adaptively recovered with time. It is concluded that vestibular inputs to postural control are interpreted within a sensory-motor context of head-on-body orientation.

Keywords — posture; equilibrium; vestibular compensation.

Introduction

Maintenance of postural stability and orientation in space requires accurate information about the configuration of the body, its parts, and their relationships to external space. This information is available through various sensory systems including visual (1–3), vestibular (4,5), somatosensory (3,6), and presumably efferent motor signals (3). However, another equally important but largely neglected aspect of equilibrium control is CNS processing that integrates these various sensory inputs into a coherent “model” of internal and external spatial relations used to regulate postural sway. The accuracy of this processing is crucial for a variety of equilibrium functions including the control of posture.

The importance of CNS processing is clear from an analysis of “raw” sensory information available during postural sway, which is inherently ambiguous. For example, assume an inverted pendulum model of postural sway. If an individual with head erect and facing forward sways so that the head pitches 10° forward, the vestibular endorgans signal a 10° forward body pitch. If the same body motion occurs with the head turned 90° to the right, the vestibular endorgans now signal a 10° forward body roll. A particular body sway can thus produce very different vestibular feedback signals depending upon head and, therefore, vestibular endorgan orientation. If vestibular information is to be effectively used to maintain postural stability, then the orientation of the head on the body must be available to the postural control system. This sensory-motor context is required to properly encode the vestibular signals resulting from body sway. This head-on-body information is presumably provided by neck proprioceptors that must be properly calibrated to convey true head turn.

In principle, postural stability should dete-
iorate when the relationship between vestibular input and information about head on body is disrupted. This might occur in subjects with vestibular lesions. Vestibular lesions result in marked asymmetry of resting activity and responses in neurons in the vestibular nuclei (13,15). This altered function influences motor responses through convergence with somatomotor functions (see reference 8 for review) and may underlie the decrements in postural stability observed in chronic vestibulopathic subjects (5,9). This argument is supported by work that simulated vestibular dysfunction and asymmetry through unilateral galvanic stimulation. Cathodal current across one labyrinth produces abnormal postural sway that is directionally sensitive to head position (10-12). The directional sensitivity implies a relationship between vestibular and head-on-body information.

The effects of vestibulo-somatosensory dysfunction might best be appreciated in patients with unilateral vestibular ablation (UVA). With acute UVA, the vestibular system is unbalanced, resulting in patterns of stimulation that are similar to multiplanar head rotation to the deficient side and head tilt. In both UVA and galvanic stimulation, the vestibular system inaccurately transduces tonic gravito-inertial information and generates an erroneous asymmetry, even though the head is stationary and upright. This error likely underlies the accompanying postural instability.

It is known that acute UVA causes postural disorders that disappear over time (5,13-15). It is not clear whether the instability resulting from UVA is modulated by head-on-body position and, if so, whether adaptive recovery of function occurs. Two possible mechanisms might govern recovery of postural stability after UVA. The first is a reestablishment of normal balance in the central vestibular system, as is known to be the case (7,14-17). The second mechanism would reestablish accurate calibration between vestibular and neck information, thereby providing an appropriate sensory-motor context to interpret vestibular feedback during postural sway. Recalibration could be accomplished through, for example, the known convergence of vestibular and somatosensory information in vestibular nuclei (see reference 8 for review).

The present study investigated the role of sensory-motor context underlying postural control. Vestibular-proprioceptive influences on postural stability were quantified while the head was upright and facing forward, leftward, and rightward. The effects of UVA on postural sway measures in these different contexts, as well as the time course of recovery, were measured.

**Methods**

**Subjects**

Thirty normal and 19 unilateral vestibulopathic human subjects (age range, 18 to 54 years) participated in these experiments. Clinical history and examination (by GDP) as well as audiologic and electronystagmographic (ENG) tests were obtained on all subjects. Evaluation of normal subjects revealed no evidence of ocular, auditory, vestibular, somatosensory, or central nervous system disease.

Evaluation of vestibulopathic patients revealed normal functions, except those specific to unilateral otologic function. Of the 14 patients who took part in the main experiment, 4 patients had unilateral Meniere's disease and 10 had small eighth nerve tumors with no evidence of CNS compromise. All showed normal caloric responses (standard 30°C and 44°C irrigations) on the unaffected side and either normal (n = 6), depressed (n = 6), or absent (n = 2) responses on the vestibulopathic side. Five additional patients who had undergone UVA for small eighth nerve tumors were studied 18 or more months postoperatively (range, 18 to 72 months).

**Equilibrium Measures**

Anterior-posterior (A/P) postural sway was quantified using the NeuroCom “Equi-test” device whose basic operating principles have been described elsewhere (4,18). Subjects stood on a surface instrumented with
force transducers fore and aft. The surface was secured to an infrastructure that translated in the A/P direction or rotated about an axis roughly collinear with the ankle joint. During 20 s “static” trials, subjects stood with eyes closed on the support surface, which either was fixed or was pitched proportionally to body sway (“sway-referenced”). Sway-referencing served to minimize the influences of relevant somatosensory inputs of the lower extremities in postural control. This, together with the absence of visual input (eyes closed), enhanced the sensitivity of postural sway as a coarse measure of vestibular influences in postural control (4,6,18).

Experimental manipulations. Each subject was asked to align head and eyes with a target that was either central, 45° right, or 45° left, in the visual surround. Forward sway would stimulate the left anterior/right posterior canal pair for the 45° head right position (L-side forward), but the left posterior/right anterior canals for the 45° head left position (R-side forward). The pattern of otolith stimulation would also change. For patients, head turn had the effect of reorienting the pathologic (ipsi) or normal (contra) vestibular end-organ forward. For all conditions, 2 trials were performed and the results were averaged.

Data Recording and Analysis

Dynamic force acting upon the support surface was transduced and analog signals digitized and stored at 100 Hz. Subjects’ A/P center of gravity (COG) in the horizontal plane was internally calculated based on a nonlinear model that used an internal table of arthrokinetic data (19).

COG was further reduced to an equilibrium score (ES). ES is calculated as pk-pk COG excursion for the entire trial scaled by the arthropometric base of support, calculated from subject height (18). An ES of 100 indicates no sway and 0 indicates a “fall”; that is, a shift in bipedal stance. When averaging the two ESs for a given test condition, a 0 was treated numerically. Thus, if a subject scored 0 and 50 on the two trials, the average score was taken as 25.

Additionally, head position asymmetry scores (AS) were calculated as percent difference in ES between right (R) and left (L) side forward positions in normal subjects (AS_n) (equation 1), and between ipsi-(I) and contra-(C)-side forward positions in the vestibulopathic (AS_v) subjects (equation 2):

$$\text{AS}_n = ((R - L) / R + L) \times 100; \quad [1]$$

$$\text{AS}_v = ((I - C) / I + C) \times 100. \quad [2]$$

0% represents perfect symmetry, +100% represents a fall only with left or contra forward head position and −100% represents a fall only with right or ipsi forward head position.

Experimental Protocol

Normal subjects and the 5 patients who were 18+ months postoperative were measured in the 3 head positions during a single session. The 14 remaining patients were measured in the 3 head positions at each of 4 separate time periods: a) Preoperatively (within 1 week prior to surgery), and b) 1 week (wk), c) 1 month (mo), and d) 4 months postoperatively (n = 14, 7, 10, 9, respectively).

Results

Head Centered Orientation

ES means and standard errors for both the normal and vestibulopathic groups for the head centered position are illustrated in Figure 1. ES in normal subjects (Figure 1, mean = 69.1, SE = 1.7) is in accord with comparative data described by NeuroCom (19).

In unilaterally vestibulopathic patients, ES is generally lower than normal both before and after UVA (Figure 1; P < 0.01, 0.01, 0.05, 0.05 for preoperative, 1 wk, 1 mo, and 4 mo, respectively; 2-tailed t tests). Postural stability was poorest immediately after vestibular ablation, as demonstrated by a near 50%
drop in ES compared to preoperative values (Figure 1). Paired t tests on the subjects who had measurable scores at both time periods \((n = 7)\) revealed this drop to be statistically significant \((P < 0.05)\). ES recovered to better than preoperative levels by 1 month \((P < 0.05)\). However, postural stability in the patient group never reached the performance level of the normal group even after 4 or more months (Figure 1). The low ES in the 18+ mo group is primarily due to one subject who was unable to stand under the test conditions employed.

**Effects of Head Turn**

ES means and standard errors for both the normal and vestibulopathic groups for both head-turned positions are illustrated in Figure 2. Postural stability in normal subjects for the left-forward and right-forward head orientations were statistically indistinguishable (paired t test). Further, ES for both head-turned positions were statistically indistinguishable from ES for the head-centered position (compare Figures 1 and 2). Results imply that postural stability in normal subjects is uninfluenced by horizontal head position.

Preoperatively, vestibulopathic subjects were generally less stable than normal subjects, resulting in lower ESs (Figure 2). During the acute postoperative period, ES scores dropped further for all head positions, and were on average indistinguishable between orientations, both before and after UVA (Figures 1 and 2). However, considering only those subjects who had measurable (nonzero) responses both before and acutely after UVA \((n = 5;\) the other 2 fell repeatedly), the drop in ES for the contra-forward position was not statistically significant, while that for the ipsi-forward position was \((P < 0.01;\) paired t test). Note the recovery in postural stability for all head orientations after 1 or more months (Figures 1 and 2).

Asymmetries in postural stability due to changes in horizontal head orientation were also analyzed in terms of right–left asymmetry scores (AS, see Methods), which are presented in Figure 3. Mean AS for normal subjects were indistinguishable from 0 (mean = −1.85, SE = 4.87). AS between the ipsi- and contra-forward head positions for all subjects at each time period (Figure 3) demonstrate many abnormally large asymmetries. However, no systematic preference for either head orientation was observed.

Since head position affects postural stability in unilateral vestibulopathic subjects, but not in a clearly systematic way, asymmetry scores were analyzed as absolute values \(|AS|\) to investigate overall asymmetries and the time course for recovery of normal func-
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Figure 3. Asymmetries in postural stability (AS) for each patient preoperatively (pre-op), acutely postoperative (1 wk), and 1, 4, and 18+ months postoperatively (1 mo, 4 mo, 18+ mo, respectively). Means for the normal subjects are also plotted.

Figure 4. Absolute asymmetry in postural stability (|AS|) in vestibulopathic subjects at each time period. Means for the normal subjects are also plotted. A + or −100 indicates a “fall.”

Head Orientation and Falls

Figure 5 illustrates the “Fall” rate; the mean number of fallers for each condition and head orientation. A faller is here defined as an individual whose ES was 0 on at least one test. Normal subjects never fell (Figure 5). In contrast, vestibulopathic patients showed the same general pattern for “Fall” rate as they did for ES (compare Figures 1 and 2 with Figure 5). That is, a relatively low level of falls occurred preoperatively. “Fall” rate increased acutely postoperatively, but then decreased over time. Where ipsi/contra asymmetries exist, there were more falls with the head in the ipsi- than in the contra-forward position. This corroborates the differential drop in ES where the contra-position showed less acute effects of surgery than the ipsi-position did (see above). Patients 18+ months postoperatively (Figure 5) showed either no falls in any horizontal head position (n = 2), or no falls in the contra-forward position but falls in the head-centered or in the ipsi-forward position (n = 3). It is worth noting that the 2 subjects that did not fall had normal |AS| scores.

Discussion

Postural Control and Head Orientation

Sensory information for postural control acquires significance only when interpreted within a sensory-motor context specified by the location and orientation of the different sensing organs in relation to the controlled motor system. The postural control system must have access to this context through independent sources (for example, somatosensory information about head orientation) that are accurately cross-calibrated with other sensory inputs (for example, from vestibular) if postural control is to operate properly in different head positions.

In this study, postural stability in normal subjects was unaffected by static horizontal head position. This implies that vestibular inputs are indeed interpreted properly within a context of head-on-body orientation in con-
trolling posture. Findings are consistent with other studies that quantified postural stability during head tilt, which reorients the head relative to gravity, instead of head turn, which does not (21,22).

**Effects of Vestibulopathy**

An interesting demonstration of the influence of sensory-motor context on vestibular control of posture is provided by studies using unilateral galvanic stimulation of the labyrinth (10–12). When the head is upright and facing forward, cathodal current across the right labyrinth produces predominantly leftward body sway. However, when the head is turned to the right or left, the same stimulus induces predominantly forward or backward sway, respectively (10). That head turn directly alters the direction of sway patterns resulting from identical vestibular stimulation suggests that the interpretation of vestibular inputs shifts appropriately within the context of changes in head-on-body orientation. Perhaps vestibulopathy provides a set of conditions (vestibular imbalance) that are similar to galvanic stimulation, and results in orientation-dependent postural asymmetry.

Vestibulopathic patients showed generally subnormal postural stability, more so acutely after than before unilateral vestibular ablation. Results with the head centered agree with other data on postural stability with unilateral labyrinthine lesions (8). More interestingly, after UVA they showed head-orientation-dependent asymmetry.

There are two possible explanations for the asymmetry. First, the vestibular inputs representing head movement might be correctly interpreted within the sensory-motor context provided by neck input, but asymmetric due to the direct tonic effects of UVA. The vestibular nuclei exert, through vestibulo-spinal reflexes, a tonic influence on posture that is symmetric between the two sides. Acute unilateral vestibular loss asymmetrically changes the resting activity of neurons in the vestibular nuclei, which in turn produces postural asymmetry (7,13,15). The resulting disequilib-

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**Figure 5.** Mean occurrence of “falls” (# Fallers/n) in vestibulopathic subjects at each time period and head orientation. Means for the normal subjects are also plotted.
rium should be most evident in increased, primarily lateral, sway (9). Since the Equitest is designed to assess A/P sway, some of the effects of UVA may be unmeasured. However, to the extent that UVA is similar to the effects of unilateral galvanic stimulation (see above), turning the head should bring the instability into the A/P plane, and any asymmetry might be expected to show postural differences between head orientations.

The second possibility is that the effects of UVA are not properly calibrated and integrated with somatosensory cues regarding head/neck orientation. UVA results in decreased resting activity in the ipsilateral vestibular nuclei (13); it is known that vestibular and somatosensory information converge in these structures (see reference 8 for review). This may result in misinterpretation of vestibular information concerning body movement and, in turn, abnormal (and asymmetric) postural responses. The loss of vestibular inputs disrupts other central integrative mechanisms underlying spatial orientation, as is the case with visual-vestibular interactions. For example, vestibular lesion eliminates optokinetic afternystagmus, presumably through disruption of a “velocity storage” mechanism (23). Similarly, UVA may disrupt central integrative functions that provide the sensory-motor context underlying postural stability. This sensory-motor miscalibration explanation is consistent with data presented here and in other reports (10-12,21,22) concerning head position effects on vestibular inputs to postural control.

The two explanations are not mutually exclusive. The important point from these experiments is that postural control mechanisms use information about head orientation to interpret vestibular inputs for regulating postural sway.

Adaptive Recovery

Adaptive recovery of postural control after UVA is well-established (5,9,13-15). The results of the present study support these findings and illustrate a novel adaptive plastic mechanism in human postural control. The findings suggest that the head-dependent asymmetries evident after UVA also adaptively recover over time. This is most likely due to either a) recovery of resting activity in the vestibular nucleus ipsilateral to the ablation (13), and/or b) recalibration of the mechanisms that integrate vestibular inputs with head/neck proprioceptive information.

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