Posturography does not test vestibulospinal function

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The clinical usefulness of posturography is unknown, despite its costing more than $500 per test in some areas of the United States, including Boston. We cross-sectionally and prospectively studied blinded vestibulo-ocular and vestibulospinal tests from 29 stable patients with chronic vestibular hypofunction; 22 patients were affected bilaterally (BVH), and 7 were affected unilaterally (UVH). Vestibulo-ocular function was assessed by electronystagmographic caloric stimulation and sinusoidal vertical axis rotation gains at 0.05 Hz. Vestibulospinal function was assessed by moving-platform and visual-surround posturography sensory organization tests (SOTs), paced and free gait in a gait laboratory, and clinical tests of timed gait and standing. Posturography SOT moving-platform tests 4 through 6, designed to assess vestibular function, correlated significantly ($r \leq 0.72$, $P \geq 0.01$) with vestibulo-ocular tests in 5 of 6 comparisons among BVH patients. Posturography SOT results, however, correlated poorly with other vestibulospinal measures: correlations were statistically significant for only 7 of 18 comparisons with clinical balance and gait function ($r \leq 0.69$, $P \geq 0.01$) and with 2 of 12 comparisons for gait laboratory dynamic stability measures ($r \leq 0.55$, $P \geq 0.01$) among the BVH patients. When both the platform and visual surround moved (SOT 6), however, correlations were statistically significant with static standing clinical measures ($r = 0.51$ to 0.69, $P < 0.01$) and with whole-body maximum moment arm during paced gait ($r = 0.55$, $P < 0.01$). Posturography scores for the UVH patients did not significantly correlate with any vestibulo-ocular or other vestibulospinal measures. These data indicate that among patients with BVH posturography SOT scores relate at best modestly with accepted measures of vestibulo-ocular function, less well with clinical measures of balance control, and poorly with dynamic gait-performance measures. We conclude that posturography SOT does not assess vestibulospinal function. (Otolaryngol Head Neck Surg 1999;120:164-73.)

Posturography is intended to characterize postural control in general and vestibulospinal abnormalities in particular.[1-5] There is no "Barany chair" for vestibulospinal reflex (VSR) assessment: no single test evaluates vestibular deficits.[1,6-8] Several authors suggest, however, that posturography characterizes VSRs, and electronystagmography (ENG) measures vestibulo-ocular responses (VORs).[5,6]

There are 2 main types of posturography. Static posturography measures "standing still" body sway on a stationary force platform[5,7] where the subject is not perturbed; dynamic posturography (EquiTest; NeuroCom International Inc, Clackamas, OR) uses a moveable platform and visual surround to estimate body sway during sensory or mechanical perturbations.[2,7,9] Vestibulo-ocular tests, including ENG calorics and Barany chair vertical axis rotation, are the gold standard used to diagnose vestibulo-ocular function.[7,10] Posturography reportedly enhances vestibular patient classifications and can monitor change after treatments such as rehabilitation.[4,5]

Posturography is intended to measure the ability to maintain the body's center of gravity (CG) within the base of support[3,9] during alterations of ankle and visual sensory input. In functional activities such as gait, by contrast, the CG is outside the base of support except during the brief double-support phase.[11] Thus on both practical and theoretical grounds it is important to assess whether measures of postural control during gait are associated with postural control on a moving platform. We therefore hypothesized a direct correlation between posturography and vestibulo-ocular and gait-function scores.

The belief that posturography test results relate to gait performance in subjects with vestibulopathy is intuitively appealing but almost entirely untested.[7] To date, no research correlates dynamic posturography scores with calorics and sinusoidal vertical axis rotation (SVAR), nor with other vestibulospinal markers derived from locomotion tests or timed bedside clinical tests, although 2 studies note a good relationship between vestibulo-ocular and static posturography and ataxia

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tests.[5,12] Nashner[13] reported that leg electromyogram response patterns of healthy subjects during perturbed locomotion were similar to those seen during perturbed standing. Nashner found the latency and spatial organization of automatic postural reactions during the single-support phase of gait closely resembled those occurring during platform perturbations. He noted that the rate of body-forward sway during ambulation must continually be readjusted to coordinate timing and placement of the swing foot. Nashner therefore concluded that the "automatic" subtests of the EquiTest were similar to functional gait responses. Sensory organization tests (SOTs) evaluate postural reactions to sway-referenced platform or visual-surround motions. This SOT evaluates subjects' ability to select appropriate postural reactions during diminished or conflicting sensory information about their spatial orientation. Both posturography and gait involve center of gravity (CG) control; we therefore hypothesized a direct correlation between gait performance measures and SOT equilibrium score.

Posturography SOTs are intended to be useful for establishing which sensory system—proprioceptive, visual, or vestibular—the subject relies on most for maintaining upright standing.[9] The locomotor laboratory assesses postural stability during naturalistic, self-induced voluntary movement, particularly the effect of vestibulopathy on gait, when the CG is outside the base of support.[11,14] Clinical tests of balance and gait are quick and efficient measures of stability under differing sensory and biomechanical conditions during voluntary movements.[14]

Patients with peripheral vestibular hypofunction have difficulty walking down a busy street, getting out of bed at night, carrying bundles, or performing simple activities of daily living such as taking a shower.[14-16] Patients with peripheral vestibular dysfunction have difficulty with both static and dynamic disturbances in the VOR and vestibulospinal reflexes (VSR).[6] Patients with peripheral vestibular hypofunction have difficulty orienting to gravity when visual inputs and somatosensory inputs are decreased.[16] Numerous studies conclude that rehabilitation increases these individuals' mobility at home and in the community,[4,6,14,16,17] but to date none has focused on how the SOT portion of posturography relates to traditional measures of balance and gait performance.

METHODS AND MATERIAL

Subjects

Twenty-two subjects with bilateral vestibular hypofunction (BVH) and 7 subjects with unilateral vestibular hypofunction (UVH) participated. Twenty subjects were female; subjects' ages ranged from 31 to 90 years (Table 1). All subjects exhibited vestibulo-ocular test abnormalities and abnormal scores on EquiTest posturography SOTs 5 and/or 6. Subjects were included on the basis of history, physical examination, and vestibular diagnostic test results: ENG, SVAR, and visual-vestibular interaction rotation testing.[1,6,16] Patients classified as having BVH had bilaterally decreased caloric responses (total slow-phase velocity of ≤10 degrees/second for the sum of AD27 + AS27 + AD44 + AS44 (ie, right and left ear caloric stimulation with 27°C and 44°C warm water) and ≤8 degree/second slow-phase velocity for the sum of ADO + ASO (ie, 35 ml of ice water stimulation in each ear) and decreased gains (at least 2.5 SD below normal mean values) at all frequencies of SVAR testing.[18] Subjects with UVH had at least 1 of the following: 30% unilaterally reduced caloric response, positional nystagmus while lying with the damaged ear down, and confirmatory abnormalities on rotational testing (mildly decreased low-frequency gains, increased phase leads, and/or asymmetric rotation-induced nystagmus).[7] In addition, all subjects reported a locomotor balance impairment of at least 2 months' duration and had requested vestibular rehabilitation. No subjects had vestibular rehabilitation before these experiments. All subjects were free of musculoskeletal disorders and central nervous system deficits, as determined by a board-certified neurologist's examination. The UVH and BVH patients exhibited significant impairments in gait, most notably in double-support time and average gait velocity (Table 2). Not all subjects were able to complete all of the tests because of postural instability, vertigo, nausea or other physical symptoms, or technical problems. This research was reviewed and approved by the institutional review board of MGH. All subjects gave informed, written consent before the study.

Table 1. Subject characteristics

	BVH subjects		UVH subjects	
	Mean	**SD**	**Mean**	**SD**
Age (yr)	63.0	18.0	56.4	19.1
Height (m)	1.7	0.1	1.4	0.7
Weight (kg)	68.4	16.1	60.7	14.8
Number	22		7	

Vestibulo-ocular Testing

Calorics. A closed-loop irrigator circulates 27°C or 44°C water for 40 seconds through a distensible balloon placed in the external auditory canal. The stimulation and initial recording are performed with the patient's eyes open in the dark (light-proof mask). Approximately 110 seconds after onset of stimulation, the mask is removed, and the eyes are fixed on a light-emitting diode to examine fixation suppression of nystagmus. At least 5 minutes is required between the end of an

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Table 2. Descriptive data from all tests for all patients

	BVH subjects			UVH subjects		
	Mean	SD	n	Mean	SD	n
Vestibulo-ocular function						
Calorics*	0.32	0.42	22	0.86	0.24	7
SVAR gain	0.18	0.16	22	0.66	0.09	7
SVAR phase†				16.15	9.3	7
Posturography						
SOT 1	91.4	4.3	22	94.1	1.7	7
SOT 2	81	20	22	89	6.3	7
SOT 3	82.6	20	22	90	8.7	7
SOT 4	62	28	21	82.4	8	7
SOT 5	7	19	21	46.8	24.7	7
SOT 6	5.7	18.2	21	45.8	23.8	7
Clinical						
TSEO (s)	54.6	79.1	22	117.3	76.5	7
USEO (s)	20.9	33.2	22	30.9	34.2	7
USEC (s)	2.6	7.2	22	18	34.2	7
TGEO (no. of steps)	11.6	11.8	22	21.8	8.7	7
WHRTC	1.8	1.4	22	2.4	1.2	7
WHRTI (s)‡	22.9	21.3	10	27.3	12.2	7
Locomotion laboratory§						
DST (%) (Nl = 10)	15.9	3.3	20	17.1	1.6	7
ST (%) (Nl = 60)	66.8	9.6	20	70.7	5.5	7
MOMAX (cm) (Nl = 20)	17.2	5.3	19	18.4	3	7
GAV (cm/s) (Nl = 143)	94.8	29	21	100.5	20.2	7

Calorics, ENG response to thermal stimulation; *DST,* paced-gait double-support time; *ST,* paced-gait stance time; *MOMAX,* paced-gait maximum moment arm distance between CG and CP; *GAV,* preferred speed gait average forward velocity of the body's CG.
*Median score for BVH and UVH subjects was 0 and 1.0, respectively.
†Phase at 0.05 Hz, 50 degrees/second.
‡In WHRTI only 10 of the 21 subjects completed the timed portion; 11 subjects stepped over 1 of the margin lines and scored an incomplete.
§References 14 and 17; momax normal value *(Nl)* from unpublished MGH locomotion laboratory data.

observed response and the beginning of the next irrigation. Each ear is stimulated with warm and cool water. The mean peak slow-phase velocity of nystagmus is calculated for each stimulation from the electronystagmogram. Data for calorics were trichotomized for scoring and analysis into normal response, no response, or partial response, and scored 1, 0, or 0.5, respectively.

SVAR. Rotational testing is performed with individuals seated in a chair attached to a motorized platform that rotates left and right. The head is strapped to the top of the chair. The sinusoidal rotational stimuli at 7 frequencies from 0.01 to 1.0 Hz have a peak velocity of 50 degrees/second. During rotation, induced eye movements are recorded with conventional ENG while the individual's eyes are open in the dark. The parameter analyzed in this study is the gain of the VOR (ratio of peak slow-phase eye movement velocity to peak stimulus velocity), at a chair rotation stimulus frequency of 0.05 Hz.[7]

Vestibulospinal Tests

Posturography. A commercially available EquiTest posturography device was used to examine SOTs 1 through 6. The subjects stood with a standardized foot placement on 2 strain gauge platforms that measured horizontal and vertical forces. The platform and visual surround were computer controlled. The computer program allowed measurement of body sway as well as control of the platform or visual surround. Movement of the CG was estimated from the horizontal projection of the center of pressure (CP). The visual surround and platform sway were then referenced to this estimation.[2,3] Subjects wore a parachute harness secured to the frame of the platform to prevent falling. Six subtests comprise SOT: SOTs 1 through 3 use a fixed platform and SOT 4 through 6 use a sway-referenced platform. There are 3 visual conditions: (1) eyes open and fixed on a stable visual surround (SOTs 1 and 4), (2) eyes closed (SOTs 2 and 5), and (3) eyes fixed on a sway-referenced visual surround (SOTs 3 and 6).[2] Each sensory subtest is 20 seconds long and is repeated 3 times. The best of 3 trials was used for this study; this equilibrium score is based on the assumption that a normal individual can exhibit 12.5 degrees of anteroposterior sway without falling.[2] The score is determined by comparing an angular difference between the patient's calculated CG maximum displacement during a trial and the patient's theoretical maximum.[2] The score is then expressed as a percentage between 0 and 100. A

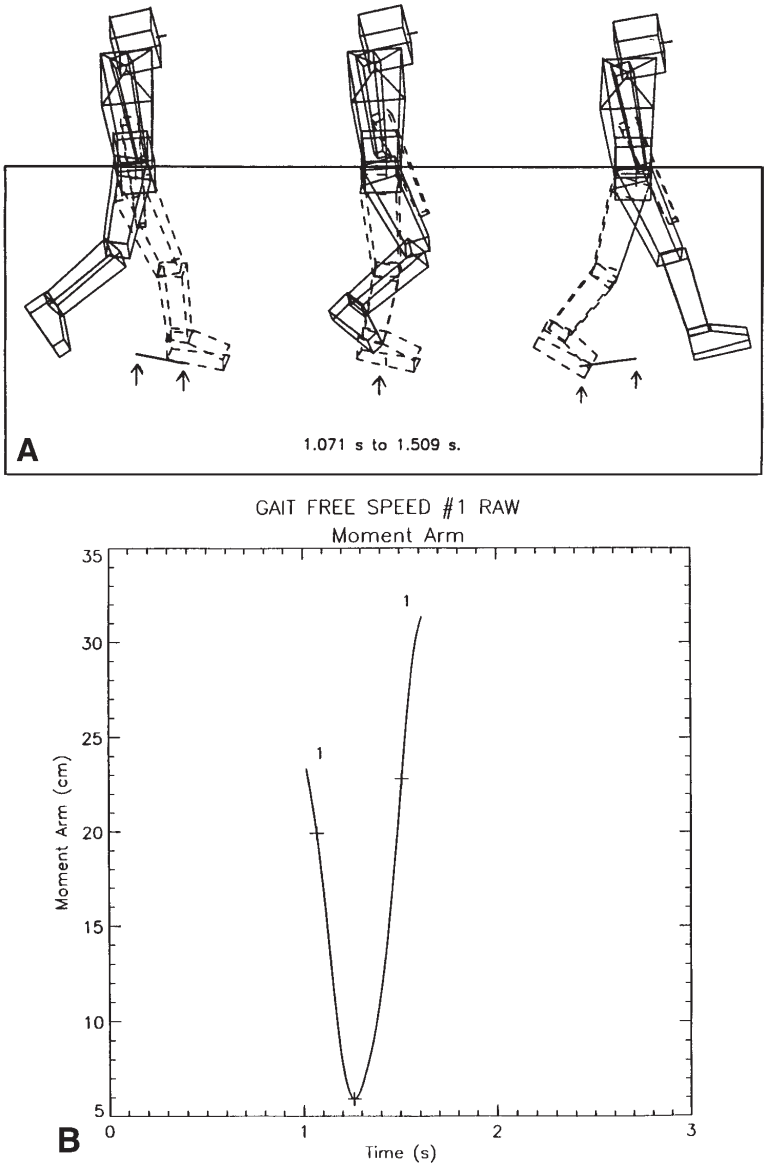

Fig 1. A, Three-dimensional 11-segment data from a subject during gait from a three quarters overhead perspective. Horizontal distance (moment arm) between the whole-body CG projected onto the floor and the CP is shown for the right side *(dashed lines). Left,* Immediately after left toe off, the *first (left-most) arrow* indicates horizontal projection of the whole-body CG relative to the *second arrow,* indicating the CP. The line between the CG and CP is the whole-body moment arm. *Middle,* At right midstance, the *arrow* indicates that CP and CG are nearly coincident in this view although in fact this minimum moment arm is 6 cm (see **B**). *Right,* Just before left heel strike, the *first arrow* indicates CP, the *second arrow* indicates the whole-body CG, and momax is attained near the end of this subject's single-limb stance. **B,** Whole-body moment arm time history. The 3 *plus signs* correspond respectively in time with the 3 events in **A**. The *first plus sign* corresponds to just after left toe off and shows a 19.5 cm moment arm; the *second plus sign* corresponds to right midstance and shows a 6 cm moment arm; and the *third plus sign* corresponds to just before left heel strike and shows a 23 cm moment arm, the momax for this subject.

score of 100% indicates maximum stability and no sway, and a score of 0% indicates maximum sway and/or a step off the platform.

Locomotion laboratory. Full-body kinematics and kinetics were measured during locomotion as described in detail elsewhere.[19] In brief, full-body kinematic data are acquired with a motion-analysis system by use of SELSPOT II hardware and the TRACK kinematic data analysis software pack-

Table 3. Pearson correlation between vestibulo-ocular tests (SVAR and calorics) and posturography scores (SOTs 4 through 6) for BVH and UVH patients

	BVH subjects (n = 16)			UVH subjects (n = 3)		
	SOT 4	SOT 5	SOT 6	SOT 4	SOT 5	SOT 6
SVAR gain	0.38	0.63*	0.68*	–0.99	–0.78	–0.39
Calorics	0.42†	0.72*	0.67*	NV	NV	NV

Note that negative correlations are insignificant because the hypothesized relationship was positive.
NV, No variance in caloric data so correlation cannot be computed.
*$P < 0.01$.
†$P < 0.05$.

age (Massachusetts Institute of Technology, Cambridge, MA). System precision is <1 degree for angular and <1 mm for linear kinematics. Kinetic data were obtained from 2 Kistler platforms and processed in the same computer. All data were sampled at 150 Hz.

Light-emitting diode arrays were mounted on 11 body segments: the right and left feet, shanks, thighs, and arms; the pelvis; the trunk; and the head. These markers did not restrict motion in any way. Subjects walked barefoot; each test was performed twice. Free, and then paced, gait was performed by the subjects walking a 10 m path. Arm swing was not restricted during gait. Subjects were instructed to move forward in a straight line at their preferred speed (free gait) or to keep their foot strikes in time with a metronome set at 120 beats/minute (paced gait). Four measures of dynamic stability were extracted: free gait forward velocity, paced-gait maximum moment arm (MOMAX), double-limb support time, and stance duration.[14] Whole-body MOMAX is the greatest root mean square distance between the CG and the CP in the horizontal plane (Fig 1), during single-limb stance.[19] Subjects with poorer balance have smaller momaxes, slower forward velocity, and longer stance and double-limb support durations.[11,14]

Clinical measures. Clinical tests were modified from the Fregly Ataxia Battery[20] and Herdman et al[6] and included timed tandem standing eyes open (TSEO), unilateral standing eyes open (USEO), and unilateral standing eyes closed (USEC). Three trials of all standing tests were done with the subjects barefoot with arms folded across the chest. The sum of all 3 trials' times was the score for that test. For TSEO feet were positioned nondominant heel to dominant toe. The maximum duration of each trial of TSEO was 60 seconds. For tandem trials the patients were instructed to place the heel of his or her foot just in front of the toes of the other foot with both feet pointing forward in a straight line. A business card with the word "open" printed in capital letters was placed 5 ft in front of the subjects. Patients were not instructed to look at the card. If a patient looked at the card for the first trial he or she was instructed to look at the card for subsequent trials, if a patient did not look at the card for the first trial he or she was not allowed to look at it in the next 2 trials. For USEO, subjects stood on the dominant limb with eyes open. Limb dominance was determined by having the subject kick a ball (leg used to kick the ball). The patients were instructed to pick their (nondominant) foot up off the floor a few inches and balance on the (dominant) leg." During USEC the subject stood on the dominant limb with eyes closed. The patients were instructed as above and were told to then close their eyes. The maximum duration of each unilateral standing trial was 30 seconds.

Timed clinical locomotor measures were also performed with bare feet, but with the arms relaxed at the side. During tandem gait eyes open (TGEO) subjects walked on a 7.5 cm × 3 m piece of black tape and were told to try to place their entire foot on the line as they walked, with 1 foot just in front of the other. Subjects performed 3 trials and were scored by summing the number of steps taken before stepping off the tape, across the 3 trials. Ten steps maximum for each trial were counted.

Walking with head rotation required subjects to walk along a 6 m walkway demarcated by three 7.5 cm parallel lines, 15 cm apart, while performing head yaw rotations of 45 degrees left and right, respectively, at every other step. A metronome was set at 60 beats/minute to control their walking cadence. Subjects were instructed to walk forward the way they would normally walk down the street but also to turn their heads from side to side in time with the ticking as they walked. Subjects performed 3 trials whose scores were summed: (1) walking with head rotation task completed (WHRTC) was scored yes (1 point) or no (0 points); and (2) walking with head rotation time (WHRTI) was the duration (in seconds) per completed trial. The task is scored incomplete (0 points) when a subject steps outside of either outer line with more than 25% of either foot before completing the 6.2 m walkway.

Data Analysis

Frequencies, means, standard deviations, and Pearson correlation were used to test the hypotheses, with alpha = 0.05.[21]

RESULTS

Descriptive data for all tests are presented in Table 2. Pearson correlation coefficients for BVH and UVH data are summarized in Tables 3 through 7.

BVH Patients

Posturography scores for SOTs 1 through 3, in which the platform remained stable and the visual field was removed (SOT 2) or altered (SOT 3), were relatively high (means of 91.4, 81, and 82.6, respectively; the maximum possible score for each SOT, 1 to 6, is 100). SOT scores for test 4, in which the platform swayed,

Table 4. Pearson correlation between posturography SOTs 4 through 6 and clinical or locomotor laboratory results for BVH and UVH patients

	Clinical scores						Locomotor laboratory scores			
	TSEO	USEO	USEC	TGEO	WHRTC	WHRTI	DST	ST	MOMAX	GAV
BVH subjects										
SOT 4	0.40	0.41	0.26	0.53*	0.42†	–0.15	0.44†	0.41	0.18	0.27
SOT 5	0.14	0.27	0.67*	0.16	0.16	0.06	0.30	0.38	–0.08	0.12
SOT 6	0.51*	0.65*	0.69*	0.52*	0.35	–0.30	0.25	0.07	0.55*	0.40
n	21	21	21	21	21	10	19	19	18	20
UVH subjects										
SOT 4	0.07	0.06	–0.39	–0.55	NV	–0.14	0.02	–0.46	0.29	–0.07
SOT 5	–0.33	0.266	0.21	–0.01	NV	–0.35	–0.11	–0.27	0.05	0.60
SOT 6	–0.58	–0.10	–0.22	–0.17	NV	0.12	0.50	–0.32	0.049	0.06
n	7	7	7	7	7	7	7	7	7	7

DST, Paced-gait double-support time; *ST,* paced-gait stance time; *MOMAX,* paced-gait maximum moment arm distance between CG and CP; *GAV,* preferred speed gait average forward velocity of the body's CG; *NV,* no variance in the data because all subjects scored 3 on WHRTC so correlation cannot be computed.
*$P < 0.01$.
†$P < 0.05$.

Table 5. Pearson correlation between clinical (timed) tests and MOMAX for BVH and UVH patients

Parameter	MOMAX
BVH subjects (n = 19)	
TSEO (s)	0.51*
USEO (s)	0.52*
USEC (s)	0.24
TGEO (s)	0.71†
WHRTC	0.27
WHRTI (s)	–0.52
UVH subjects (n = 7)	
TSEO (s)	0.73
USEO (s)	0.49
USEC (s)	0.14
TGEO (s)	0.25
WHRTC	NV
WHRTI (s)	–0.37

Note that negative correlations are insignificant because the hypothesized relationship was positive.
NV, No variance in the data because all subjects scored 3 on WHRTC so correlations cannot be computed.
*$P < 0.05$.
†$P < 0.01$.

Table 6. Pearson correlation between vestibulo-ocular and posturography scores on SOTs 1 through 3 for BVH and UVH patients

	BVH subjects (n = 17)			UVH subjects (n = 4)		
	SOT 1	SOT 2	SOT 3	SOT 1	SOT 2	SOT 3
SVAR gain	–0.23	0.15	–0.15	–0.93	–0.93	–0.94
Calorics	–0.25	0.15	–0.13	NV	NV	NV

Calorics, ENG response to thermal stimulation; *NV,* no variance in calorics data so correlation cannot be computed.

were lower (mean 62) than for the previous 3. Scores were extremely low for SOT 5, in which the platform swayed and vision was absent, and for SOT 6, in which the platform and visual surround swayed, for patients who were able to complete the tests (mean of 7.0 and 5.7, respectively; Table 2).

Among the clinical timed standing trials, TSEO had the greatest score, and USEC was the most difficult (54.6 and 2.6 seconds, respectively). The maximum possible time for the standing tests was 60 seconds. Walking with head yaw rotations for 6.2 m was completed by only 1 subject, although most subjects were able to partially complete the task (Table 2).

The locomotion laboratory measures of double-support time and stance duration were 16% and 67%—substantially higher than normal values of 10% and 60% of cycle time, respectively—as expected from subjects with postural instability. MOMAX was smaller than normal, also as expected in subjects who have difficulty controlling their CGs (Table 2).

Calorics and SVAR were directly correlated with posturography SOTs 5 and 6 (Table 3). At most, however, 52% ($r^2 \leq 0.72^2$) of the vestibulo-ocular tests' variance was shared with posturography.

As indicated in Table 4, SOT scores were only infrequently significantly correlated with other vestibulospinal stability measures. Only SOTs 4 and 6 correlated with any locomotor laboratory measures ($r \leq 0.55$); SOT scores were unrelated to stance duration

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Table 7. Pearson correlation between posturography scores on SOTs 1 through 3 and clinical and locomotor laboratory for BVH and UVH patients

	Clinical scores						Locomotor laboratory scores			
	TSEO	USEO	USEC	TGEO	WHRTC	WHRTI	DST	ST	Momax	GAV
BVH subjects										
SOT 1	0.38	0.36	0.20	0.40	0.22	0.06	0.23	–0.08	0.16	0.20
SOT 2	0.10	0.11	0.10	0.22	0.29	0.08	0.13	–0.09	0.05	–0.10
SOT 3	0.07	0.09	0.09	0.11	0.19	0.51	0.12	–0.09	0.04	0.04
n	22	22	22	22	22	10	20	20	19	21
UVH subjects										
SOT 1	0.24	0.67	0.63	–0.03	NV	–0.13	–0.40	0.40	0.21	0.36
SOT 2	0.10	0.19	0.28	–0.48	NV	0.35	–0.15	0.51	0.01	–0.17
SOT 3	0.29	0.45	0.38	–0.20	NV	0.28	–0.05	0.53	0.27	–0.13
n	7	7	7	7	7	7	7	7	7	7

DST, Paced-gait double-support time; *ST,* paced-gait stance time; *GAV,* preferred speed gait average forward velocity of the body's CG; *NV,* no variance in calorics data (all subjects scored 3 on task completed).

(Table 4). There were more correlations between SOT scores and clinical measures ($r \leq 0.69$) (Table 4). Table 5 shows that tandem standing and other clinical tests were well correlated with the locomotor laboratory measure, MOMAX ($r \leq 0.71$).

Tables 6 and 7 list the correlations between SOTs 1 and 3 and all of the vestibulo-ocular and vestibulospinal measures. There were no significant correlations.

UVH Patients

No significant correlations were found among any variables (Tables 4 through 7).

DISCUSSION

Few published reports have examined whether posturography and other measures of vestibular function are related. In this study, among the BVH patients posturography SOTs 4, 5, and 6 significantly correlated with most peripheral vestibulo-ocular tests (ie, caloric stimulation and SVAR).[7] SOT 4 correlated significantly with calorics, but only 16% ($r^2 = 0.42^2$) of the variance was explained (Table 4). SOTs 5 and 6 correlated with calorics (49% variance explained) and SVAR (36% and 49% variance explained, respectively). This correlation is especially impressive given that calorics and SVAR primarily stimulate the horizontal canal, whereas posturography primarily should stimulate the anterior canals and otoliths. Posturography is, however, intended to assess in vivo human vestibulospinal system function.[2,5,7]

Determining the function of both the vestibulo-ocular and vestibulospinal systems is important to ensure correct identification of all patients with peripheral vestibular disorders.[5,6] Black and Wall[5] reported static platform posturography to be more sensitive and specific than calorics and rotation tests in diagnosing Meniere's disease and benign paroxysmal positional vertigo. Black and Wall then paired the vestibulospinal test (static platform posturography) with each of the vestibulo-ocular tests to determine which combination was more sensitive. The combination of posturography and rotational testing was the most sensitive for the Meniere's group, with 83% of the patients correctly classified. Caloric testing in the standing position engenders VSRs including body sway, as well as nausea and vertigo, in normal persons and subjects with vestibular hypofunction.[22] Hadj-Djilani[22] found that 25% of patients with normal VORs to caloric stimulation in sitting had abnormal VSRs to caloric stimulation in standing, concluding that VSR was more sensitive than VOR to caloric testing. Our data suggest posturography at best modestly enhances the diagnosis of peripheral vestibular hypofunction.

Among BVH patients, 7 of 18 posturography-to-clinical test correlations statistically exceeded chance expectations (Table 4). Statistically significant relationships included TSEO, USEO, USEC, and TGEO, which vary proprioceptive input from, and subject patients to, different biomechanical constraints than normal standing. Decreased base of support during these clinical tests decreases sway stability in healthy and vestibulopathic subjects.

Shupert et al[23] also noted that BVH subjects standing tandem or with a reduced base of support increase body sway and head movement when compared with normal subjects. Patients with BVH lack head stability and show greater angular head acceleration and angular displacement than normal individuals during gait.[24] Patients with VOR impairment have difficulty stabilizing their gaze during such body and head movements,

obviating a world-stable visual reference frame for spatial-orientation estimation. This visual instability also occurs when the visual surround is moving on SOTs 3 and 6.

Allum and Pfaltz[25] hypothesized that vestibular information controls 65% of body stability, with visual and proprioceptive systems contributing 35%. During SOT 6 the vestibular system is presumably the main sensory control because SOT 6 seeks to corrupt visual and somatosensory input.[13] SOT 6 and USEC may have correlated strongly because both had a high number of 0 (fall) scores, implying a strong visual dependency among the vestibulopathic patients. SOT 6 also correlated significantly with whole-body CG/CP MOMAX (Fig 1) during single-limb stance of gait. In SOT 6, when the somatosensory and visual inputs are confounded, postural control degenerates. In functional ambulation BVH patients keep their CGs close to their CPs to maintain postural stability.[26] After vestibular rehabilitation, this CP-CG MOMAX improves substantially.[14]

Gait variables including double-support time and stance duration for paced-gait and free-gait velocity did not correlate highly with posturography SOTs 4 through 6 among BVH subjects. Paced-gait double-support, cycle time, stance duration, vertical CG excursion, and paced- and free-gait velocity are impaired in BVH.[11] The patients included in this study exhibited significant gait deviations; their average gait velocity was markedly reduced, and their double-support time was 70% longer than normal subjects'. We therefore expected free- and paced-gait stability variables to correlate well with SOT scores. Nashner[13] described similar postural adjustments during perturbed walking and perturbed standing on a moveable platform. He also noted that body motions relative to the support surface during perturbed gait in the single-support phase resemble the anteroposterior sway motions (balance adjustments) during standing on the moveable platform. During late single-support phase in gait, the CG is ahead of the CP; forward displacement is arrested by placement of the other leg forward. Nashner did not sway reference the platform, but rather used platform perturbation stimuli similar to the motor subtest of posturography. Nonetheless, this hypothesized relationship between dynamic posturography and gait function is belied by the clear lack of relationship between scores on EquiTest SOTs 4 through 6 and traditional measures of gait stability in our vestibulopathic subjects. This poor correlation may be because during gait, subjects were likely to use a variety of compensatory mechanisms to improve their balance that were not available during the more restricted SOTs. During ambulation the subjects had all senses available, and the visual field was orientationally correct, which is analogous to the conditions for SOT 1. Whereas SOT 5 did not correlate with gait, SOTs 4 and 6 showed very modest correlations with 2 gait parameters (Table 4). The difference may be that vision is absent for SOT 5. Indeed, BVH subjects report difficulty in walking on uneven terrain, in poorly lighted environments, and in complex visual environments, where self- versus-external object movements must be distinguished (eg, crossing busy streets, walking in busy grocery store aisles).[15,17] None of these sensory conditions is present during typical laboratory gait assessments.

As expected, SOTs 5 and 6 were very difficult for patients with vestibular hypofunction: 16 of the 29 patients scored 0, having maximum sway or taking a step, at platform rotation gains of 1.0. When the platform is sway referenced, the inputs from the ankle are supposed to be corrupted.[8] Vestibulopathy combined with corrupted proprioception may add a delay in responding to platform stimuli, causing an underdamped, increased body sway after perturbation. Shupert et al[23] proposed that vestibulopathic patients use greater hip movements because they lack trunk stability, causing greater CG excursions. Our data clearly indicate peripheral vestibular hypofunction patients exhibit greater sway on a moving than on a stable platform (Table 2). Sway magnitude on a stable platform and vestibular impairment are uncorrelated (Table 6). Patients exhibited marked sway for SOT 5 (absent vision), and for SOT 6 (moving visual field), it would seem that vestibular ocular and vestibulospinal abnormalities coexist in these patients. The moving visual surround may introduce conflict into both the optokinetic–vestibulo-ocular and visual vestibulospinal systems.

The mechanics of the EquiTest device's stimulus delivery may account for the SOT scores' modest relationship to vestibulo-ocular function. Barin[3] suggested body sway may be modeled in an oversimplified manner by treating the body as a single inverted pendulum. Inverted pendulum sway is assumed by the "ankle strategy" model. If the inverted pendulum model were valid, CG position could be derived from the smoothed upward projection of the CP position.[3] EquiTest platform sway is governed by this smoothed CP position.[27] The CG estimation from CP data is reasonable for near-static standing, but the CG/CP relationship is not maintained for more dynamic movements used by vestibulopathic subjects.[2,27] Because the CG-CP mechanical couple increases as movement dynamics increase, the sway-referenced platform may behave as a sway stimulus because the platform reacts to the greater CP dynamics whereas the patient's CG has less excursion

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than can be estimated from CP kinematics. Our group recently reported that the CG/CP displacements used in posturography significantly misrepresent posture dynamics in vestibulopathic subjects: CG/CP velocity and perhaps higher time derivatives are needed to capture dynamic postural behavior.[27,28] Both Gu et al[29] and Barin[3] suggest that measuring the excursion of the CP and estimating how far the CP moves within the base of support may be more informative about postural stability than estimating CG displacement from CP kinematics. Parker[8] evaluated vestibulopathic subjects during posturography by reducing the sway-referenced gain of CP travel to platform sway from the factory preset 1.0 to 0.75 or 0.5. Reduced platform rotation gain decreases platform sway and enables quantitative scoring of BVH: the patients can then generate nonzero scores. The platform is no longer as highly reactive to CP dynamics, and the patient is no longer as deprived of support-surface input. Thus decreasing the gain changes the nature of the test, but it gives quantitative information about the patients' abilities to stand.[8]

Among subjects with BVH, SOT 4 correlated significantly with TGEO, WHRTC, and double-support time—tests that measure control of the CG when it is outside the base of support. Patients with vestibulopathy exhibit ataxia during functional ambulation and report oscillopsia.[29] It is important to note that the clinical tests with which SOT 4 data correlate, specifically TGEO and WHRTC, modify the proprioceptive input and increase the level of mechanical difficulty, while allowing intact vision. TGEO and WHRTC also accentuate oscillopsia because of increased head movement and decreased VOR.

The CP-CG MOMAX[19] during paced gait correlated with 3 of 6 clinical standing-balance measures (TSEO, USEO, and TGEO) (Table 5). Small CG-CP moment arm couples are used if normal, destabilizing threats to gait stability, such as ground reaction forces and large joint torques, cannot be controlled.[14] If subjects can maintain a posture while the distance between their CG and their CP is increasing, then they are exhibiting a larger area of stability and a longer moment arm.[19] The clinical measures reduced the patient's base of support during quiet standing, which may increase postural sway. Therefore it is reasonable that measures of gait stability and reduced base of support standing scores would covary.

Essentially no significant correlations were found among the UVH patients' posturography, SVAR, calorics, and locomotor laboratory scores, reflective of our low statistical power. The UVH patients were younger than the BVH patients (mean age 54 and 63 years, respectively). With advanced age, postural sway may increase,[30] and timed balance scores may decrease.[31] The UVH patients walked faster in free gait than the BVH patients (mean velocity 100 and 95 cm/second, respectively). There has been some suggestion in the literature that for UVH patients the correlation of SOT with ENG may be poor because the latter measures low-frequency stimuli of the lateral semicircular canals (during caloric stimulation) whereas the SOTs may measure vestibular function at higher frequencies and measure the vertical semicircular canal stimuli.[32,33] This would not, however, explain why gait and SOT do not correlate, because patients with vestibulopathy exhibit greater angular head acceleration during gait than normal subjects.[24] The literature also suggests that VOR and VSR compensation may occur independently of each other.[34] UVH patients may have a variety of normal and abnormal clinical and posturography findings at presentation.[34] This may be true, but it would not explain the lack of correlation between the 2 VSR measures, posturography and functional gait.

A clear need exists to determine what contribution, if any, posturography can offer to medical decision making.[35,36] Among both our BVH and UVH subjects, SOT 1 through 3 subtests did not correlate with ENG or clinical or gait laboratory measures. Herdman et al[6] reported a similar lack of correlation between posturography SOTs 1 through 3 and vestibulo-ocular tests, although they did not examine other vestibulospinal function markers. Moreover, Herdman et al also found no significant difference in motor latency tests between BVH and healthy controls. Posturography assumes a quasistatic relationship between the whole-body CG and CP,[2,27] which is demonstrably incorrect.[27,28] To our knowledge, the present data are the first prospective, blinded comparison of posturography and other vestibulo-ocular and vestibulospinal markers.

This investigation had several limitations. The small number of UVH patients makes it difficult to draw conclusions about this population. The motor control subtest of posturography was not analyzed, in part because the reliability of those data is suspect. Future studies may examine the correlation of motor control subtests with gait and clinical measures. The 2 populations of vestibular hypofunction patients were not compared, in part because of sample size restrictions, but future studies should consider making such a comparison. Moreover, we cannot know how representative these patients are, but it is true that we included all consenting subjects in an "intention to treat" model and thus believe the results are generalizable.

In summary, our data show that the SOT subtests of posturography, especially SOT 6, correlate at best modestly ($r < 0.72$) with traditional vestibulo-ocular test

results, less well with clinical timed tests of static standing balance, and quite poorly with clinical and laboratory measures of dynamic stability during locomotion. Therefore SOT results cannot be used in isolation to assess functional vestibulospinal control, as measured by actual locomotor performance assessment.

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