"Wide-based gait" is considered indicative of imbalance. No quantitative gait analyses, however, have related base of support to steadiness during gait. To determine whether patients with cerebellar or vestibular disorders had a wider base of support than matched healthy individuals, we analyzed 102 balance-impaired patients and healthy subjects during free and paced gait. Kinematic data were collected using a high-precision optoelectronic system. There were no significant differences in the base of support between unsteady and healthy subjects, nor between patients with vestibular and cerebellar diagnoses. The base of support correlated with the body mass index and waist circumference in all subject groups. These data suggest that base of support during gait fails to identify balance-impaired subjects and is related more to biomechanical than to neurological factors. Therefore, "wide-based gait" should no longer be considered the sine qua non of ataxic or unsteady gait. Clinicians should not focus on decreasing base of support as a therapeutic goal for chronic, unsteady patients.

**Key words:** stride width, gait, vestibular, cerebellar.

INTRODUCTION

A broad base of support (BOS), also referred to as "stride width," is believed to be characteristic for people with unsteady gait and balance problems. For example, stride width was one of the main variables shown to be higher in healthy elderly persons than in younger healthy adults (1, 2), especially in people with a history of falls compared with elderly subjects without frequent falls (3). Static standing stability certainly improves with a wider base of support even in patients with cerebellar and vestibular lesions (4–6), but there is a paucity of literature on walking parameters of patients with various disease-related balance disorders. Although posturography appears to be an established method for evaluation of imbalance in patients with vertigo, gait analysis better reflects performance changes and the balance problems during everyday life of these people (7). Thus, we sought to identify an association of gait base of support with balance disorders.

The BOS is not a standardized parameter in gait analysis (8, 9). For example, BOS was defined as the lateral distance between the heels and a slant of the foot placement in relation to the direction of body motion (10). Equally, stride width was reported as the perpendicular distance of consecutive mid-heel or medial malleolar placement locations relative to the line of walking progression (8, 11). We use the term BOS to define distance between the centers of mass of the left and right feet projected onto the frontal plane at heel strike, when both feet are on the ground and the body is in an inherent state of instability (12) (Fig. 1).

The purpose of this study was to determine whether neurologically-induced balance impairment factors were more...
important than chance or biomechanical factors in determining gait base of support. We hypothesized that unsteady subjects would walk with a wider BOS than healthy controls.

**METHODS**

**Subjects**

A total of 102 subjects, comprising 32 subjects with cerebellar disorders (age 44.96 ± 13.20 years); 36 subjects with vestibular disorders (age 44.61 ± 13.80 years), 8 with left side and 10 with right side unilateral vestibular hypofunction; and 34 healthy subjects (age 46.91 ± 14.97 years), were compared (Table I). Groups were matched by age, sex, and, then, body mass index. No subject had any other neuromuscular or skeletal dysfunction and all usually walked without an assistive device.

Bilateral vestibulopathy (VSP) was diagnosed by reduced caloric responses and vestibular ocular reflex (VOR) gains greater than 2.5 SD below normal using sinusoidal vertical axis of rotation tests at a frequency range of 0.01–1.0 Hz. Unilateral VSP patients had reduced caloric response of at least 30% to cool and warm water ear stimulation of 27 and 44°C, respectively (13). Subjects with cerebellopathy all had MRI- or CT-conﬁrmed lesions conﬁned to the cerebellum. Subjects with vestibular or cerebellar disorders had not received any vestibular physical therapy and had at least 6 months of stable symptoms; all were referred from neurologists who agreed with the patient that he/she had an unsteady gait and required balance therapy to address their unsteadiness. Informed consent of all subjects was obtained prior to this data collection as approved by the Massachusetts General Hospital institutional review board for human research.

**Instrumentation**

Data collection instrumentation, determination of kinematic data and center of mass (COM) position estimates are described in detail elsewhere (14, 15). Briefly, the instrumentation included 4 Selspot II® optoelectronic cameras, 64 infrared light-emitting diodes and 2 Kistler piezoelectric force plates. The light-emitting diode arrays were firmly attached to 11 body segments, right and left feet, shanks, thighs and arms; as well as the pelvis, trunk and head. Each body segment was modeled as a rigid body with 6 degrees of freedom (3 translations and 3 rotations), TRACK software (Telemedically Rapid Acquisition of Kinematics; Massachusetts Institute of Technology, Cambridge, MA) was used to analyze the data. System precision is <1 mm of linear displacement and orientation <1 degree of angular displacement. Floor reaction forces were collected with 2 Kistler force plates, which have accuracy equal to ±1%. Measuring anthropomorphic data for each subject allows included waist and mid-thigh circumference; these data were included in measurements used to determine the relative COM location of individual segments. Thus, calculation of the whole-body COM can be estimated, knowing the COMs of the individual body segments and their location in space. Both kinematic and kinetic data were sampled at a rate of 150 Hz. The BOS was determined during free walking (self-selected speed) as well as during paced walking (120 beats per minute) by measuring the frontal plane distance between the left and right foot’s center of mass (Fig. 1) at heel strike, as detected by the forceplates.

**Procedure**

All subjects walked barefoot and in shorts and a T-shirt, without an assistive device, on a 10-m walkway. Subjects were first asked to walk at preferred speed (“as if you are taking a brisk walk in the park”) and then paced using a metronome set at 120 beats per minute (“walk the way you normally do but keep up with this beat”). Preferred speed walking was studied because it should reflect the individual’s optimal, self-selected stride and cadence. Paced walking was studied because differences in gait velocity or cadence might independently affect stride and BOS characteristics, but controlling cadence by use of a metronome pace should reduce those velocity-dependent, between subject differences. Two independent trials of preferred and of paced gait were gathered, each of which is included in the analyses.

**Statistics and calculations**

Differences between healthy subject and patient populations were determined by unpaired t-tests of the demographic data and by using one-way ANOVA with Tukey’s multiple comparison test and then ANCOVA to statistically control BMI, for the other outcomes. Unilateral and bilateral VSP groups did not differ in BOS, nor any other variable, and thus are combined into a single VSP group. Pearson correlations between BOS and subjects’ physical parameters between and within groups were calculated.

**RESULTS AND DISCUSSION**

The base of support does not differ between patient groups with vestibular or cerebellar disorders.

Subjects with vestibular diseases did not differ significantly in BOS from patients with cerebellar disorders or healthy subjects (Fig. 2). All patients walked without canes or support; severity of gait unsteadiness varied from nearly unable to walk without support to healthy subjects’ unimpaired gait. The BOS of cerebellar patients was slightly (<25 mm) greater than healthy subjects’ (p = 0.05, Fig. 2), a difference that is clinically insignificant. This small difference cannot be discerned by visual observation and indeed is most likely due to the higher average body mass index (BMI) in the patients with cerebellar disorders and not a direct consequence of cerebellopathy per se, because a positive correlation of BOS with BMI was observed in all 3 groups. We were unable to correct for this slight BMI mismatch between the subject groups, because the majority of

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**Table I. Descriptive data from balance-impaired and healthy subjects. Mean values and standard deviations are given**

<table>
<thead>
<tr>
<th></th>
<th>n</th>
<th>Age, years, mean (SD) min–max</th>
<th>Gender (F/M)</th>
<th>BMI, mean (SD) min–max</th>
<th>Most frequent disorders</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients with vestibulopathy</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bilateral n = 18</td>
<td>36</td>
<td>44.61 (13.80)</td>
<td>22/14</td>
<td>27.76 (6.61)</td>
<td>Idiopathic vestibulopathy</td>
</tr>
<tr>
<td>Unilateral n = 18</td>
<td></td>
<td>20.25–70.41</td>
<td></td>
<td>18.56–49.05</td>
<td>Drug-induced ototoxicity</td>
</tr>
<tr>
<td>Patients with cerebellopathy</td>
<td>32</td>
<td>44.96 (13.20)</td>
<td>13/19</td>
<td>33.90 (6.69)</td>
<td>Vestibular nerve section</td>
</tr>
<tr>
<td>Healthy subjects</td>
<td>34</td>
<td>46.91 (14.97)</td>
<td>18/16</td>
<td>23.03 (3.46)</td>
<td>Vestibular neuronitis</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>102</td>
<td>53/49</td>
<td>28.78 (6.80)</td>
<td>Status post cerebellar stroke</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Pan-cerebellar degeneration</td>
</tr>
</tbody>
</table>
patients with cerebellar diseases had a higher BMI than the vestibulopathic patients (Table I). It is reasonable to suggest that greater BMI may be due to the cerebellar subjects’ inactivity prior to their physical rehabilitation program. When BMI was statistically controlled in the ANCOVA, there were no between group differences.

To confirm that the minimal increase of the BOS in patients with cerebellar disease was due to higher BMI, we re-analyzed the data after excluding the 12 most obese subjects (~35%) of these patients and the 12 healthy subjects with lowest BMI (~35%) from the analysis of the BOS. This equalization of the BMI of patients with cerebellar disorders to that of healthy subjects resulted in a statistically equal BOS. Thus, we conclude that the BOS is normal in patients with imbalance due to cerebellar or vestibular disorders, suggesting that the BOS is a non-specific gait parameter and does not represent a variable that can be used to distinguish between healthy people or unsteady patients with different diseases.

We have previously shown that frontal plane postural stability of subjects with cerebellar degeneration can be differentiated effectively from non-disabled volunteers by a dynamic, constrained stepping task (6), suggesting that other variables are more specific discriminators of cerebellar ataxia than BOS. These findings extend the results of a posturographic study, which showed that none of the posturographic parameters could distinguish between 2 otherwise similar groups of patients with vestibular or cerebellar lesions (4).

It is well known that a wider BOS increases “standing still” postural steadiness (5): indeed, larger buildings require greater BOS than smaller buildings. However, during dynamic activities such as locomotion, a wider based gait would require greater mediolateral center of mass sway and thus consume more energy, or decrease gait velocity, compared with a narrower base. Bicycles are inherently unstable in “standing still” but during dynamic “locomotion” are quite stable. Hirayama et al. (17) reported 8 patients with peripheral neuropathy who could walk quite adequately, but were unable to stand still. We and other authors have shown that standing still balance does not predict gait (7) or falls in unsteady patients; the present data show that the mechanical demands of standing still, which are improved by increased BOS, are quite different than those of gait, which may not be made more stable with increased BOS.

**Base of support is equal during free and paced walking speed**

The BOS during free walking was essentially equal to that observed during paced gait (120 beats per minute) in both patient groups and in the healthy subjects (Fig. 2). This is most likely because most individuals had nearly identical walking speeds between the 2 trials (Table II). Changes in BOS may be affected by walking speed in some healthy subjects (18), but neither our patients nor healthy controls differed between paced and free gait. However, our study aimed to identify differences between disease-linked imbalance conditions during normal walking, which is what clinicians most often assess, so we did not investigate a wide range of gait speeds that might alter the BOS. We sought the more clinically relevant information, BOS during “normal gait” that might be observed during a neurological examination. Although paced gait was insignificantly faster in all 3 groups of subjects, than was free gait, both are well within previously reported ranges for their respective groups.

**Correlations of the base of support with body parameters**

BOS correlated with the BMI and waist circumference in all subject groups (Fig. 3, BOS: 8.27–36.13 cm; BMI: 18.56–49.05; waist: 61.6–123.8 cm; $r = 0.38$ and 0.37, respectively; $p < 0.001$). Neither age nor thigh circumference, however, correlated with the BOS (age: 20.16–71.08 years; $r = 0.38$ and 0.37, respectively; $p < 0.001$). Neither age nor thigh circumference, however, correlated with the BOS (age: 20.16–71.08 years; $r = 0.38$ and 0.37, respectively; $p < 0.001$). Neither age nor thigh circumference, however, correlated with the BOS (age: 20.16–71.08 years; $r = 0.38$ and 0.37, respectively; $p < 0.001$). Neither age nor thigh circumference, however, correlated with the BOS (age: 20.16–71.08 years; $r = 0.38$ and 0.37, respectively; $p < 0.001$).

**Table II. Walking speed for free and paced gait, by group. Mean values and standard deviations are given**

<table>
<thead>
<tr>
<th></th>
<th>Healthy controls</th>
<th>Patients with vestibulopathy</th>
<th>Patients with cerebellopathy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Paced cadence</td>
<td>130.1 (16.9)</td>
<td>116.6 (17.6)</td>
<td>114.7 (15.1)</td>
</tr>
<tr>
<td>Preferred speed</td>
<td>125.2 (22.5)</td>
<td>103.4 (18.7)</td>
<td>100.3 (18.5)</td>
</tr>
</tbody>
</table>

**Fig. 2.** The base of support is not significantly different between the 3 groups of subjects: 32 patients with gait unsteadiness from cerebellar etiology (ce), 36 patients with vestibular hypofunction (vh) and 34 healthy subjects (he) were analyzed; shown is base of support during free and paced (120 beats/min) gait, as indicated with means ± SD.
allow a clear distinction between elders and younger adults (Table I) and cannot yield an unequivocal result about age-related changes in gait parameters.

Limitations

Although these subjects were not randomly selected and may not be representative of all ataxic or unsteady gait, this is the largest sample reported to date in which “broad-based gait” was investigated. We included only those subjects able to walk without an assistive device, but we included patients with a range of gait impairments, from unaffected healthy controls to severely unsteady subjects with vestibulopathy or cerebellopathies, who requested balance rehabilitation. Therefore, these data probably reflect the gait of the bulk of chronic, unsteady patients who present for balance rehabilitation. These data may not, however, represent subjects with uncompensated gait disorders such as acute traumatic brain injury or vestibulopathy.

It may well be that people with acute balance disorders compensate with a wider BOS; our patients included only those with chronic, “stable” gait instability and ataxia.

CONCLUSION

A Medline search for wide-based gait or base of support returns over 200 articles, most involving patients with cerebellopathy or vestibulopathy. Wide-based gait is anecdotally described in many neurology texts as being the sine qua non of gait ataxia and unsteadiness. We found only 1 report (21) that objectively compared ataxic patients’ foot kinematics to healthy, matched controls. Pulliyath et al. (21) studied only 10 patients, but they also found no difference in BOS between cerebellar patients and controls. Apparently this teaching, that patients with unsteady gait have greater BOS than normal, is based on tradition and perhaps biased visual observation, rather than rigorous scientific analysis. Our data suggest that BOS does not distinguish healthy individuals from unsteady patients with cerebellar- or vestibular-induced gait ataxia. These patients had clear balance deficits and requested rehabilitation to remedy their unsteady gait and apparently differed from the matched healthy subjects only by the presence of balance pathology. Because BOS did not discriminate healthy from balance-impaired gait, we suggest that “broad-based gait,” no longer be referred to as a condition sine qua non of unsteady gait.

ACKNOWLEDGEMENTS

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REFERENCES

European Board of Rehabilitation Medicine

The next meeting of the UEMS section and Board of Physical and Rehabilitation Medicine will take place in Vienna, Austria between 17–19 October 2002

This meeting is being organised by Dr Haymo Bruhne in association with Professor Veronika Fialka-Moser.

The meeting will commence on Thursday afternoon (17th) with an address by the President of the Section, Professor Fialka-Moser (Austria), followed by the President of the Board, Dr. Angela McNamara (Ireland). A synopsis of these presentations will be provided in advance to the Secretary-General, Professor Alain Delarque, for inclusion in the Annual Report. Reports will also be prepared and presented by the Chairmen of the Clinical Affairs and the Professional Practice Committees respectively.

The new rules of the PRM Section have been prepared over the past year and were finalised in Brighton in May 2002 at the Spring meeting. These reports will now be presented to the General Assembly for adoption.

The terms of office of the President of the UEMS section and the Deputy Treasurer have now expired, nominations are now sought for an election to fill both positions. These nominations will be accepted and an election will take place at the forthcoming meeting.

Professor Guy Vanderstraeten (Belgium) is the Director-General of the PRM Board Examination Committee. Professor Xanthi Michail (Greece) was appointed as Examinations Secretary at Brighton. These officers will report on the new rules for the examination to the General Assembly and will discuss the role of the National Managers on the operation of the examination in each country.

Professor Chantraine (Switzerland) has responsibility for the assessment of training centres and of trainers and he will report on the “site visits” programme.

On Friday (18th) in the morning session, the group will divide into two workshops namely Clinical Affairs and Professional Affairs. The Chairman of the Clinical Affairs group is Professor Bengt Sjolund (Sweden) and he brings significant experience to this group on the processes of accreditation including CARF. The Chairman of the Professional Affairs group is Dr Guy Wanet (Belgium).

On Friday afternoon the general assembly will reconvene and receive reports on the matters discussed itemising the various matters requiring further discussion. This Meeting will continue on Saturday morning (19th) and will conclude at lunchtime.

On Saturday afternoon, a meeting of the European Federation of Physical and Rehabilitation Medicine will take place at the same location.