

Romberg's sign

Development, adoption, and adaptation in the 19th century

Douglas J. Lanska, MD, MS, MSPH; and Christopher G. Goetz, MD

Article abstract—In the first half of the 19th century, European physicians—including Marshall Hall, Moritz Romberg, and Bernardus Brach—described loss of postural control in darkness of patients with severely compromised proprioception. Romberg and Brach emphasized the relationship between this sign and tabes dorsalis. Later, other neurologists evaluated the phenomenon in a broader range of neurologic disorders using a variety of simple but increasingly precise and sensitive clinical tests. Although now known as Romberg's sign, among neurologists in the late 19th century this phenomenon was sometimes credited to Romberg, sometimes to both Brach and Romberg, and sometimes discussed without attribution.

NEUROLOGY 2000;55:1201–1206

The Romberg sign detects proprioceptive loss by demonstrating loss of postural control in darkness. In examining a patient for the presence of the Romberg sign, the examiner typically observes the patient's postural stability with the patient's feet together, initially with eyes open and then with the eyes closed. The Romberg sign is present when a patient is able to stand with feet together and eyes open, but sways or falls with eyes closed.

The Romberg sign developed in the 19th century from a patient-reported symptom into a bedside-elicited sign. Early in its history, the sign was linked to a specific diagnosis, tabes dorsalis or progressive locomotor ataxia, later known to be caused by syphilis. Although neurosyphilis is only rarely seen today, the Romberg sign persists as an important element of the neurologic examination because of its utility in detecting abnormal function of the proprioceptive system. This study examines the origins of the Romberg sign and the way that early neurologic examiners elicited and interpreted it.

Original descriptions. Marshall Hall, Moritz Romberg, and Bernardus Brach first described the sign in the early 19th century, but other 19th-century clinicians were pivotal to its interpretation and positioning among the key signs of the contemporary neurologic examination.

Marshall Hall (circa 1836). In 1836, in his *Lectures on the Nervous System and its Diseases*, the

English physician Marshall Hall (1790–1857), already famous for his formulation of the concept of the spinal reflex arc,¹ described the loss of postural control in darkness of a patient with severely compromised proprioception:

“I have this day seen a patient with a slight degree of paralysis of feeling and of voluntary motion of the lower limbs. He walks safely while his eyes are fixed upon the ground, but stumbles immediately if he attempts to walk in the dark. His own words are ‘my feet are numb; I cannot tell in the dark where they are, and I cannot poise myself.’ The voluntary motions are regulated by the sense of touch, when this is unimpaired; or by that of sight, when the touch is paralyzed.”^{2, p 27}

The identical statement is made in the later edition of his text,³ but he did not develop this idea further in terms of anatomic or physiologic function,⁴ nor did he develop this symptom into a clinical test and sign.

Moritz Romberg (circa 1840). Several years after Hall's initial description, German neurologist Moritz Heinrich Romberg (1795–1873) was apparently inspired in his clinical studies of tabes dorsalis by Hall⁴ and by his mentor Ernst Horn.⁵ In the second edition of Romberg's *Lehrbuch der Nervenkrankheiten des Menschen*, published in German in 1851,^{4,6,7} Romberg described the loss of postural control experienced by patients with tabes dorsalis after closing their eyes or in darkness. Building on Hall's patient-based description, he devised a test to demonstrate the phenomenon as a bedside neurologic sign. Romberg's description is taken from the translation of the second edition into English by Edward Sieveking for the Sydenham Society in 1853:

Additional material related to this article can be found on the *Neurology* Web site. Go to www.neurology.org and scroll down the Table of Contents for the October 24 issue to find the title link for this article.

From the Veterans Affairs Medical Center (Dr. Lanska), Great Lakes VA Health Care System, Tomah, WI; the Department of Neurology (Dr. Lanska), University of Wisconsin, Madison; and the Department of Neurological Sciences (Dr. Goetz), Rush University, Chicago, IL.

Received March 27, 2000. Accepted in final form June 13, 2000.

Address correspondence and reprint requests to Dr. Douglas J. Lanska, Chief of Staff (11), Veterans Affairs Medical Center, Tomah, WI 54660; e-mail: Douglas.Lanska@med.va.gov

"If the patient [with *tabes dorsalis*] is told to shut his eyes while in the erect posture, he immediately begins to move from side to side, and the oscillations soon attain such a pitch that unless supported he falls to the ground . . . The eyes of such patients are their regulators, or feelers; consequently in the dark, and when amaurosis supervenes, as is not unfrequently [sic] the case, their helplessness is extreme . . ." ⁷⁷, pp 226-7

Later in the text, Romberg added:

"The feet feel numbed [sic] in standing, walking, or lying down, and the patient has the sensation as if they were covered with a fur . . . The gait begins to be insecure, and the patient attempts to improve it by making a greater effort of the will; as he does not feel the tread to be firm, he puts down his heels with greater force. From the commencement of the disease the individual keeps his eyes on his feet to prevent his movements from becoming still more unsteady. If he is ordered to close his eyes while in the erect posture, he at once commences to totter and swing from side to side; the insecurity of his gait also exhibits itself more in the dark. It is now ten years since I pointed out this pathognomonic sign . . . [i.e., c 1840]." ⁷⁷, pp 395-6

Bernardus Brach (circa 1840). German physician Bernardus Brach described similar symptoms around the time that Romberg recognized them, or perhaps earlier. His contributions are, however, almost universally overlooked today. In November, 1840, Brach noted that:

"It is known that people with *tabes dorsalis* have an unusual gait . . . While other paralytics drag their legs, a patient with *tabes dorsalis* lifts his leg with a straight knee and with difficulties. When he steps with his feet hard and taps on the floor, his whole body is stiff and strained. With fearful eyes he watches his every step. He is dependent upon his cane for support . . . He doesn't feel the movement he makes with his legs . . . Because of this, he raises his legs slowly to maintain balance . . . He has no sensation [proprioception] in his lower limbs so he relies upon his other senses. When walking he is very fearful of falling and uses his body and arms for counterbalance. He finds it nearly impossible to walk on a level walkway in conversation with others, or in the dark. With two patients I observed these symptoms. With tests such as cold and warm, pressure, pinching, scratching with a needle tip, or a hair, the patients responded just like a healthy person. Thus, one cannot say that they don't have any sensation . . ." ⁷⁸, p 216

Furthermore, despite their inability to stand or walk in the dark, Brach noted that these patients were not weak: indeed, in the fall of 1838 one of the patients made a five-hour journey on foot to visit Brach. This patient, a 36-year-old educated man, had described his symptoms to Brach in a letter:

"I must watch carefully every step and stone. I must use my eyes to guide me. In the dark I have no sense of balance, and even though familiar with the area, I will certainly fall. When walking, I must fully concentrate on the task of walking and not on conversation with people . . . I step so hard on my feet that my soles are sore and inflamed after a short distance. My self-confidence is low when walking up steps, over crooked paths, strange places, or with many people." ⁷⁸, pp 216-7

Adoption and adaptation. Among neurologists in the late 19th century, the loss of postural control in the dark or with the eyes closed was sometimes credited to Romberg, ⁹⁻¹³ sometimes to both Brach and Romberg, ^{11,13,14} and sometimes discussed without attribution. ¹⁵⁻¹⁸ For example, American physician William Osler, ¹⁰ French neurologist Jean-Martin Charcot, ¹² and British neurologist William Gowers ¹⁹ all alluded to the Romberg sign. In contrast, American neurologists Charles Karsner Mills ¹³ and Charles Loomis Dana ¹⁴ referred to the phenomenon as the "Brauch-Romberg sign" [sic] in their neurology texts, and this double designation was retained by some medical dictionaries well into the 20th century ²⁰ (personal communication, Micaela Sullivan-Fowler, MSLS, MS, University of Wisconsin, Madison, 1999). Other 19th-century neurologists, such as Duchenne de Boulogne, ^{15,16} British physician Charles Bland Radcliffe, ¹⁷ and former US Surgeon General William Alexander Hammond, ¹⁸ described the phenomenon as a symptom but did not credit any predecessor with its description. Despite these various descriptions and attributions, it was Romberg who first applied this phenomenon in a clinical test.

As the sign was increasingly used, its significance became better clarified. The anatomic basis of the sign as an indication of proprioceptive sensory deficits solidified, so that patients with cerebellar, vestibular, pyramidal, and muscle diseases were generally excluded by a positive Romberg sign. Simultaneously, the sign that once had been closely allied to the diagnosis of *tabes dorsalis* became linked to all causes of proprioceptive deficits, including myelopathies of many causes and sensory neuropathies. The contributions of several clinical neurologists of the late 19th and early 20th centuries led progressively to the establishment of the Romberg sign as an integral part of the contemporary neurologic examination.

Duchenne de Boulogne (circa 1858-59). In 1858 and 1859, French neurologist Guillaume-Benjamin-Amand Duchenne de Boulogne (1806-1875) published a four-part article "On progressive locomotor ataxia: studies on a disorder specially characterized by generalized problems of coordination." ^{15,16} Duchenne recognized the primary loss of sensation as the origin of ataxia and incoordination in *tabes dorsalis*, as well as the compensatory importance of vision for postural control in such patients. Specifically, Duchenne recognized that vision could be compromised and even lost with progression of *tabes dorsalis*, and that when vision declined, the patient's ataxia worsened. Although Duchenne referenced Romberg's textbook, he did not specifically allude to the Romberg sign in his articles (quotations from Duchenne de Boulogne's work are available on the *Neurology* Web site at www.neurology.org).

William Hammond (circa 1871). New York neurologist William Alexander Hammond (1828-1900) presented his observations on *tabes dorsalis* in 1871 in his *A Treatise on Diseases of the Nervous System*, the first American neurologic text. ¹⁸ Although Ham-

mond acknowledged that both Romberg and Duchenne had provided earlier descriptions of tabes dorsalis, Hammond did not specifically credit Romberg with the observation of increased postural sway in tabetics upon eye closure. Hammond did, however, apply essentially the same “test” as Romberg in clinical practice, and helped to disseminate knowledge of the phenomenon to American colleagues (and also to European colleagues through various translations of his text). Hammond emphasized the sign’s independence from muscle weakness, and claimed as well that it was useful in distinguishing tabes dorsalis from cerebellar disease (for more information, please access www.neurology.org and click on the title link for this article).¹⁸

Francis Dercum (circa 1885–1888). Philadelphia neurologist Francis Dercum (1856–1931) made a number of important observations on the gait of tabetics over the period from 1885 to 1888. Some of his insights came from his clinical work, but much of his detailed observations derived from his collaboration in 1885 with pioneering American motion-picture photographer Eadweard Muybridge.²¹ Muybridge had pioneered efforts at motion picture photography with the collodion and gelatin dry plate technology of the 1870s and 1880s, using sequential images taken with a series of uniformly spaced, mechanically or electronically triggered, single-image cameras, and projection with a rotating disk of transparencies. The Dercum-Muybridge collaboration at the University of Pennsylvania in 1885²¹ produced classic sequential images of abnormal movements in patients with neurologic disease, the earliest examples of motion pictures of medical subjects. Among these were photographic sequences of five patients with tabes dorsalis. One tabetic patient of Dercum’s was photographed walking with eyes open and then with eyes closed²¹ (figure); these sequential images demonstrate a dynamic form of the Romberg phenomenon, with a dramatic increase in ataxia when walking with eyes closed. In addition, using tracings and graphical analyses of these sequential images, Dercum demonstrated that the swing phase of the tabetic gait is characterized by increased lateral sway of the foot, increased height to which the foot is raised, and irregularity of movement.²²

William Osler (circa 1887–1890). Although Romberg’s sign was not included in pioneering clinical studies of postural sway conducted in the 1880s at the Infirmary for Nervous Disease,^{23–25} the phenomenon was certainly known to the clinicians practicing there, and Osler in particular gave an excellent account of it. Osler spent 1884 to 1889 at the University of Philadelphia as Chair of Clinical Medicine, a position that had been vacated by Provost William Pepper. While in Philadelphia, Osler worked with Silas Weir Mitchell (1829–1914) at the Infirmary for Nervous Diseases from 1887 until his departure for Johns Hopkins in 1889. Osler attended afternoon clinics in conjunction with his colleagues Weir Mitchell and Warton Sinkler, and was exposed to Mitchell’s examination techniques and those of his

disciples, including Lewis and Hinsdale. In Osler’s classic and still highly cited textbook, *The Principles and Practice of Medicine*, written from 1890 to 1892 and first published in 1892,¹⁰ he reported seeing 25 tabetics among the 1816 cases in his neurologic dispensary at the Infirmary for Nervous Diseases over the 2-year period from 1887 to 1889. Osler also observed second-hand “a large number of ataxics which frequented the Infirmary” who were under the care of his colleagues. In his text, Osler succinctly described the Romberg “symptom” in tabetics, as well as progression of the tabetic gait, based on his Philadelphia experience:

“The ataxia develops gradually. One of the first indications to the patient is inability to get about readily in the dark or to maintain his equilibrium when washing his face with the eyes shut. When the patient stands with the feet together and the eyes closed, he sways and has difficulty in maintaining his position. This is known as Romberg symptom. On turning quickly he is apt to fall. Gradually the characteristic ataxic gait develops. The patient, as a rule, walks with a stick, the eyes are directed to the ground, the body is forward, and the legs are wide apart. In walking, the leg is thrown out violently, the foot is raised too high and is brought down in a stamping manner with the heel first, or the whole sole comes in contact with the ground. Ultimately the patient may be unable to walk without the assistance of two canes . . . One of the most striking features of the disease is that with marked incoordination there is no loss of muscular power.”^{10, p 843}

Further information and observations from the Infirmary for Nervous Diseases are available at the *Neurology* Web site (www.neurology.org).

Jean-Martin Charcot (circa 1888). Jean-Martin Charcot (1825–1893) was the most celebrated clinical neurologist of the 19th century, and his classroom at the Salpêtrière Hospital in Paris drew students and practicing physicians from around the world. This international audience and his wide reputation made the Charcot classroom a ready vehicle for dissemination of neurologic information and solidification of neurologic terminology. The transcriptions of his informal classroom case presentations (*Leçons du Mardi*) demonstrate the manner in which Charcot used the Romberg sign in differential diagnosis.^{12,26} Charcot considered the Romberg sign as typical of tabes dorsalis, but found it present also in Friedreich’s disease, alcoholic neuropathy, and sometimes hysteria. He recognized the underpinnings of decreased sensory function for Romberg’s sign, and, in a time when the distinctions between spinal cord, nerve, and muscle diseases were still being delineated, he recognized that this sign was useful for excluding conditions without sensory abnormalities (e.g., primary muscle disease). In tabes dorsalis, Charcot linked the Romberg sign directly to the development of ataxia, gait dysfunction, and nighttime falling (quotations and further commentary on Charcot are available on the *Neurology* Web site at www.neurology.org).

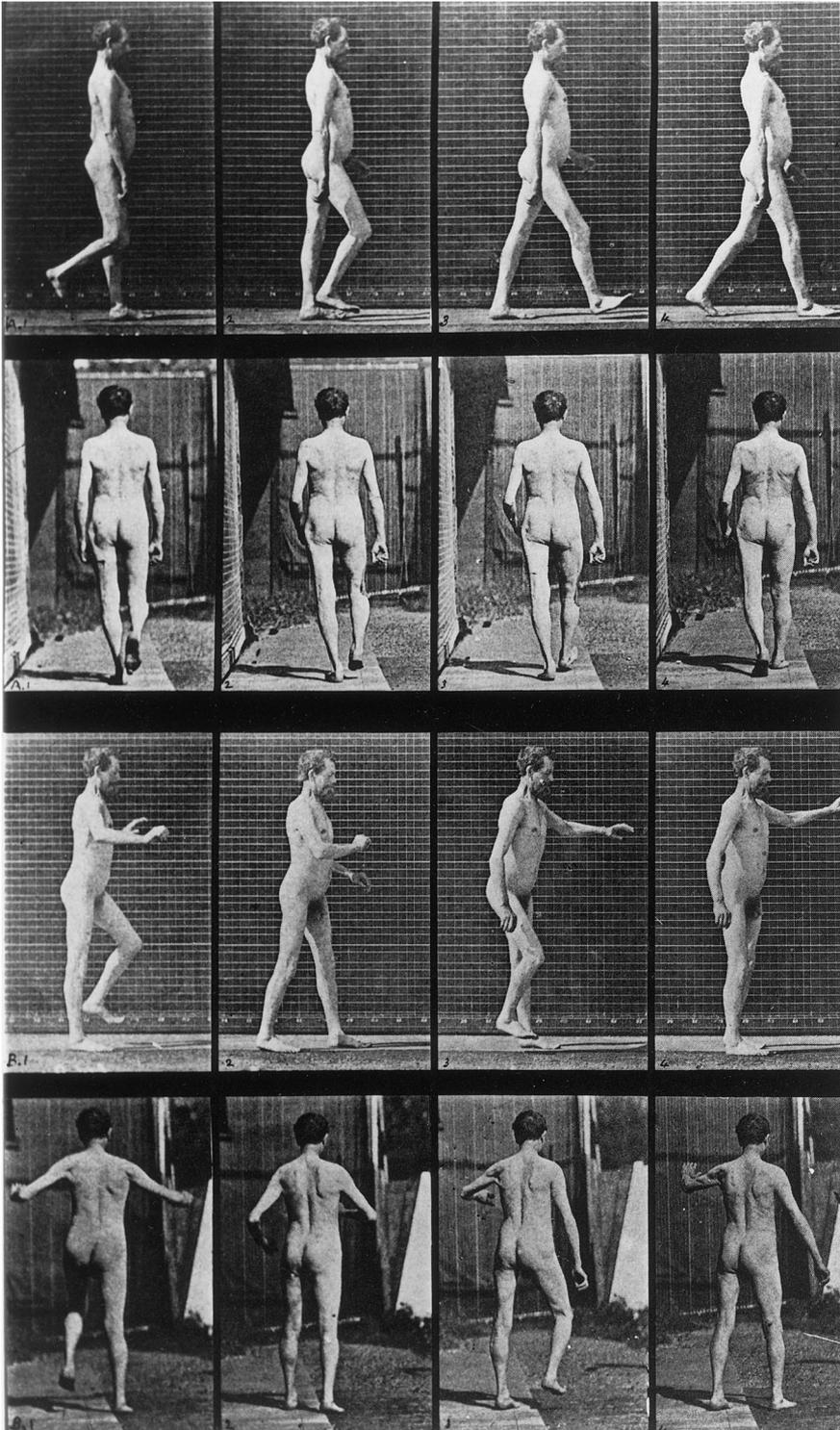


Figure. Sequential images of a patient with *tabes dorsalis* (locomotor ataxia) photographed by Muybridge with the collaboration of neurologist Francis Dercum,²¹ showing a dynamic form of the Romberg phenomenon during ambulation. In the upper two rows, the patient is shown walking fairly normally with his eyes open. In the bottom two rows, the patient demonstrates a wide-based ataxic gait while walking with his eyes closed²² due to loss of visual correction for the severe proprioceptive deficit. The captions in the version published by photographer Muybridge do not indicate the clinical circumstances correctly, but instead indicate “arms down” and “arms up” for the upper and lower rows, respectively.²¹ The correct clinical information was provided by Dercum in a separate publication.²² The “arms up” in the lower rows are a corrective response by the patient to facilitate the maintenance of balance. The images are a portion of Plate 550 from Muybridge’s *Complete Human and Animal Locomotion* (1887).²¹

William Gowers (circa 1888). In his classic text, published in 1888 and entitled *A Manual of Diseases of the Nervous System*, British neurologist William Gowers (1845–1915) gave a clear account of the specific details of the modern Romberg test, adding the instructions that the patient should assume a narrow base as part of the test:

“The characteristic incoordination of movement [in *tabes dorsalis*] develops gradually. It is always increased (as Romberg first pointed out) by closure of the eyes, and at first may only exist when the guiding

influence of vision is thus withdrawn. Before it causes ataxy of movement, it may render difficult the maintenance of equilibrium when the base of support is narrowed by the feet being placed close together, toes and heels. If then the eyes are closed, the patient sways, and may even tend to fall. In health slight unsteadiness is thus produced, varying in degree in different persons, but never amounting to even a suggestion of a fall. The effect of closure of the eyes is greatest when sensation in the soles of the feet is defective, but does not depend on this loss; it may be marked when sensation on the soles of the feet is

perfect. The early defect in coordination may be discovered by the patient when he walks in the dark, or, not uncommonly, when he shuts his eyes in the process of washing the face. In a further degree of incoordination there is inability to stand with the feet together even when the eyes are open, and the patient is only steady when the feet are wide apart. If the feet are bare, the difficulty is greater, because muscular action has to replace the rigid base of the boot . . .^{219, pp 289-90}

Contemporary use and interpretation of the Romberg sign. Optimal balance requires continuous monitoring of body sway and other orientation information provided by the somatosensory, vestibular, and visual systems. The functional ranges of these systems partially overlap, allowing partial compensation for deficits or distortions.^{27,28} For example, a normal subject can maintain upright stance either with vision eliminated (e.g., with eye closure), with proprioception disrupted (e.g., standing on a moving or tilting surface), or with vestibular function distorted (e.g., as a result of rotationally induced vertigo). Loss or distortion of inputs from two or more systems is often associated with disequilibrium and falls; thus, a patient with profound loss of proprioception, or with uncompensated unilateral or bilateral vestibular dysfunction, may fall if vision is eliminated (e.g., with eyes closed). This is the basis of the Romberg sign.

Following the initial descriptions in the 19th century, Romberg's sign was variously applied and its clinical utility was debated. Although Romberg's approach was simply to observe a change in postural control when patients were asked to close their eyes (without any stated preferred position of the feet), various "Romberg tests" and "sharpened Romberg tests" were subsequently described and often incorrectly attributed to Romberg. Some felt or implied that the phenomenon was a specific sign of tabes dorsalis, whereas others suggested it was a more general sign of deafferentation, and later authors suggested that it may be present in some patients with vestibular or even cerebellar lesions²⁹⁻³² (although not without disagreement³³⁻³⁵). There is still no standard approach to applying the "Romberg test" in clinical neurology³⁶ and the criteria for and interpretation of an abnormal result continue to be debated.^{34,36}

In 1910, Bárány emphasized the importance of vestibular inputs to postural control, and noted that patients with acute unilateral labyrinthine disease swayed and fell toward the impaired side (i.e., in the direction of the slow phase of the accompanying nystagmus).²⁹ The Romberg phenomenon is, in fact, easily demonstrated with rotationally induced physiologic vertigo, but nevertheless the existence of this phenomenon in patients with acute vestibulopathy has been questioned by some contemporary authors.^{34,35} Some of the confusion over this results from differences in examination technique and selection of compensated (i.e., less acute) unilateral vestibular disease patients for study. The Romberg test is insensitive for detecting chronic unilateral vestibular impairment. Chronically,

compensation occurs so that the patient may either not sway, or instead may sway and fall to the intact side.

Romberg's sign has been considered useful in distinguishing between sensory and cerebellar ataxia, but postural sway with eyes closed is increased both clinically³⁷ and with more sophisticated measurement and recording techniques³⁰⁻³² in patients with cerebellar disease, particularly in patients with disease of the vestibulo-cerebellum or spino-cerebellum.^{30,32} Moreover, Romberg's sign is not invariably absent in patients with cerebellar disease,³⁰ as had been claimed in the past.³³ Despite unsteadiness due to muscle incoordination and disordered regulation of vestibulo-proprioceptive reflexes in patients with cerebellar disease,³⁸ the vestibulopostural loop remains largely unaffected, and patients generally retain the ability to use vision to control much of their unsteadiness.³¹

Romberg's sign is not specific for any particular cause of ataxia, but it is most closely associated with proprioceptive sensory loss, as for example in tabes dorsalis as described by Romberg.^{6,7} Although Romberg's test is relatively insensitive to compensated unilateral vestibular or cerebellar dysfunction, it may be present in bilateral vestibular loss,³⁹ acute unilateral vestibular loss,²⁹ sometimes with pathology of the spino-cerebellum (i.e., the anterior vermis and paravermis of the anterior lobe), and less often with other cerebellar dysfunction.^{30,32} Sensitivity in routine clinical settings can be increased by narrowing the patient's base of support (e.g., with a "sharpened Romberg test" with feet in a heel-to-toe position) or by standing on foam rubber to distort proprioceptive input from the feet.^{35,40}

Although the Romberg sign is now fairly standardized, there is still some variability in examination technique and interpretation across expert neurologic examiners.³⁶ In particular, there is variability in how much postural instability is required for a positive test (e.g., increased sway only, a step to the side, or a fall); whether sway at the ankles is critical or whether sway from the hips can be accepted; whether the feet should be positioned together, as close together as possible to maintain stance with eyes open, or in tandem position; whether footwear should be worn or removed; whether hands should be held at the side or extended forward or laterally; whether the examiner should gently pull or push the patient to one side; et cetera.³⁶ Such variability may affect both the sensitivity and specificity of the test for dysfunction of proprioceptive, cerebellar, and vestibular pathways.

Acknowledgment

The authors gratefully acknowledge assistance in locating reference materials and information provided by the following individuals: Stephanie Allen, MSLS, of the University of Kentucky Medical Library; Glen Salter, MS, of the Tomah VA Medical Center Medical Library; Micaela Sullivan-Fowler, MSLS, MS, of the William S. Middleton Health Sciences Library, University of Wisconsin-Madison; and Charles B. Greifenstein, Curator of Archives and Manuscripts, College of Physicians of Philadelphia. The authors also thank Ulla Mion for help in translating a portion of Brach's article; John Henry Brook, MD, Bernd Remler, MD, Glen Salter, MS, and Henry S. Schutta, MD, for further help in Ger-

man translation; and Mary Jo Lanska, MD, MS, for critically reviewing the manuscript.

References

1. Lanska DJ. The history of reflex hammers. *Neurology* 1989; 39:1542–1549.
2. Hall M. Lectures on the nervous system and its diseases. London: Sherwood, Gilbert, and Pyer, 1836:27.
3. Hall M. On diseases and derangements of the nervous system. London: Bailliere, 1841:27.
4. McHenry LC. The emerging clinical neurologist and his manual of the nervous diseases of man. Birmingham: Gryphon Editions, 1983.
5. Schiller F. Venery, the spinal cord, and tabes dorsalis before Romberg: the contribution of Ernst Horn. *J Nerv Ment Dis* 1976;163:1–9.
6. Romberg M. *Lehrbuch der Nervenkrankheiten des Menschen*. Berlin: Duncker, 1851.
7. Romberg MH. A manual of the nervous diseases of man. Translated and edited by E.H. Sieveking. London: Sydenham Society, 1853:226–227,395–401.
8. Brach [B]. Einige Worte über einen nicht hinlänglich beachteten Punkt aus der Physiologie der Nerven und eine eigenthümliche Art von Lähmung. *Medicinische Zeitung* 1840;9: 215–217.
9. Peterson F. Locomotor ataxia. In: Dercum FX, ed. A text-book on nervous diseases by American authors. Philadelphia: Lea Brothers & Co., 1895:633–645.
10. Osler W. Locomotor ataxia (tabes dorsalis; posterior spinal sclerosis). In: The principles and practice of medicine: designed for the use of practitioners and students of medicine. New York: D. Appleton and Company, 1892:840–848.
11. Mitchell SW, Dercum FX. Nervous diseases and their treatment: general considerations. In: Dercum FX, ed. A text-book on nervous diseases by American authors. Philadelphia: Lea Brothers & Co., 1895:2–50.
12. Charcot J-M. *Leçons du Mardi: polyclinique 1887–1888*. Paris: Bureaux du Progrès Médical, 1888.
13. Mills CK. Station and sway. In: The nervous system and its diseases. A practical treatise on neurology for the use of physicians and students. Philadelphia: J.B. Lippincott Co., 1898: 170–171.
14. Dana CL. Diagnosis and methods of examination. In: Text-book of nervous diseases: being a compendium for the use of students and practitioners of medicine. 5th ed. New York: William Wood and Co., 1901:39–59.
15. Duchenne de Boulogne [GBA]. De l'ataxie locomotrice progressive: recherches sur une maladie caractérisée spécialement par des troubles généraux de la coordination des mouvements. *Arch Gén Méd* 1858;12:641–652.
16. Duchenne de Boulogne [GBA]. De l'ataxie locomotrice progressive: recherches sur une maladie caractérisée spécialement par des troubles généraux de la coordination des mouvements. *Arch Gén Méd* 1859;13:36–62,158–181,417–451.
17. Radcliffe CB. Locomotor ataxy. In: Reynolds JR, ed. A system of medicine. Vol. 2. London: MacMillan and Co., 1868:336–353.
18. Hammond WA. Sclerosis of the posterior columns of the spinal cord (locomotor ataxia). In: A treatise on diseases of the nervous system. New York: D. Appleton & Co., 1871:484–516.
19. Gowers WR. Locomotor ataxy (tabes dorsalis: posterior sclerosis). In: A manual of diseases of the nervous system. American edition. Philadelphia: P. Blakiston, Son & Co., 1888:285–323.
20. Brauch-Romberg symptom. In: Taylor NB, Taylor AE, eds. *Stedman's practical medical dictionary*. 16th ed. Baltimore: Williams & Wilkins Co., 1946:160.
21. Muybridge E. Abnormal movements, males & females (nude & semi-nude). In: Muybridge E, ed. *Muybridge's complete human and animal locomotion: all 781 plates from the 1887 Animal Locomotion*. Vol. II. New York: Dover Publications Inc., 1979:1081–1139.
22. Dercum FX. The walk and some of its phases in disease, together with other studies based on the Muybridge investigation. *Trans Coll Phys Phil* 1888;10:308–338.
23. Mitchell SW, Lewis MJ. The tendon-jerk and muscle-jerk in disease, and especially in posterior sclerosis. *Am J Med Sci* 1886;92:363–372.
24. Hinsdale G. The station of man, considered physiologically and clinically. *Am J Med Sci* 1887;93:478–485.
25. Hinsdale G. Observations on station with reference to respiration. *NY Med J* 1890;51:292–294.
26. Goetz CG. *Charcot the clinician: the Tuesday lessons*. New York: Raven Press, 1987.
27. Nashner L, Berthoz A. Visual contribution to rapid motor responses during postural control. *Brain Res* 1978;150:403–407.
28. Nashner LM, Black FO, Wall C. Adaptation to altered support and visual conditions during stance: patients with vestibular deficits. *J Neurosci* 1982;2:536–544.
29. Bárány R. Neue Untersuchungsmethoden, die Beziehungen zwischen Vestibularapparat, Kleinhirn, Grosshirn und Rückenmark betreffend. *Wien Med Wochenschr* 1910;60:2034–2038.
30. Mauritz KH, Dichgans J, Hufschmidt A. Quantitative analysis of stance in late cortical cerebellar atrophy of the anterior lobe and other forms of cerebellar ataxia. *Brain* 1979;102:461–482.
31. Bronstein AM, Hood JD, Gresty MA, Panagi C. Visual control of balance in cerebellar and Parkinsonian syndromes. *Brain* 1990;113:767–779.
32. Deiner H-C, Dichgans J. Pathophysiology of cerebellar ataxia. *Mov Dis* 1992;7:95–109.
33. Dow RS, Moruzzi G. Symptomatology of cerebellar disorders. In: The physiology and pathology of the cerebellum. Minneapolis: University of Minnesota Press, 1958:377–398.
34. Rogers JH. Romberg and his test. *J Laryngol Otol* 1980;94: 1401–1404.
35. Weber PC, Cass SP. Clinical assessment of postural stability. *Am J Otolaryngol* 1993;14:566–569.
36. Anthony TR. Neuroanatomy and the neurologic exam: a thesaurus of synonyms, similar-sounding non-synonyms, and terms of variable meaning. Boca Raton: CRC Press, 1994.
37. Haerer AF. Coordination. DeJong's the neurologic examination. 5th ed. Philadelphia: J.B. Lippincott, 1992:393–401.
38. Nashner LM, Grimm RJ. Analysis of multiloop dyscontrols in standing cerebellar patients. In: Desmedt JE, ed. *Cerebral motor control in man: long loop mechanisms*. Basel: Karger, 1978:300–319.
39. J.C. Living without a balancing mechanism. *N Engl J Med* 1952;246:458–460.
40. Shumway-Cook A, Horak FB. Assessing the influence of sensory interaction on balance: suggestion from the field. *Phys Ther* 1986;66:1548–1550.