

V. A. REID, MD\*,  
H. ADBULHADI, MD, BME\*†,  
K. R. BLACK, MS, BME\*,  
CASEY KERRIGAN †, AND  
D. CROS, MD\*

From \*Massachusetts General Hospital, Boston, Massachusetts, and †Spaulding Rehabilitation Hospital, Boston, Massachusetts.

## Using Posturography to Detect Unsteadiness in 13 Patients With Peripheral Neuropathy: A Pilot Study

■ Our aims were to use posturography to see if sway pattern differed between patients with large-fiber peripheral neuropathy and normal control subjects and, if it did, to compare posturography with conventional electromyography (EMG) as screening tools for large-fiber peripheral neuropathy. Thirteen patients who came to our neurophysiology laboratory with a preliminary diagnosis of peripheral neuropathy (made by their referring physicians) were compared with 7 nonmatched control subjects. All subjects received a neurologic examination and underwent posturography and conventional EMG. Results of posturography and conventional EMG were compared. Posturography showed abnormal sway patterns only in patients who had EMG abnormalities consistent with large-fiber peripheral neuropathy. These sway patterns differed significantly from those of the control subjects. Posturography seems to be a useful and well-tolerated screening test for patients with a history suggestive of peripheral neuropathy, and results of posturography agree with those of conventional EMG. Moreover, posturography directly measures increased sway in these patients and may be used as a more direct screen for risk of falls in this population. ■

**Keywords:** polyneuropathy, posturography, balance, electromyography (EMG)

Address correspondence and reprint requests to Didier Cros, MD, Massachusetts General Hospital, Bigelow 12, 55 Fruit Street, Boston, MA 02114 (e-mail: dcros@partners.org).

© 2002 American Academy of Clinical Neurophysiology

Large-fiber peripheral neuropathy affects many people and is prevalent in the elderly. Risk of falling is higher in patients with large-fiber neuropathy (Richardson et al, 1992; Kimura, 1993), and falling is increasingly recognized as an important cause of morbidity and mortality in the elderly. One third of the elderly who are not in nursing homes fall every year (Purdham and Evans, 1981).

Proprioception is a function of large myelinated fibers (Dyck et al, 1972), and pathology of these fibers results in abnormal postural control (Dalakas, 1986; Jamal and Donaghy, 1989). Electromyography (EMG) and nerve conduction studies are the gold standard for diagnosing peripheral neuropathy. These tests can be grueling for both the patient and the testing physician and so are probably underused in diagnosing peripheral neuropathy, especially in the elderly. Moreover, the tests do not quantify the risk of falls associated with peripheral neuropathy in this population. Posturography is a quantitative, noninvasive test measuring the amount of sway during quiet standing. In posturography, a force platform measures standing balance, with *balance* defined as the time-varying coordinates of the center of pressure (COP) under the feet of the subject. This test can be a less invasive way to help diagnose and assess the incidence, prevalence, and natural history of large-fiber polyneuropathy.

In this pilot study, we compared data from patients with polyneuropathy and from normal control subjects to see if posturography might be a useful screen for polyneuropathy. We also compared posturography results with conventional EMG results to see if posturography is a reliable indicator of large-fiber peripheral neuropathy.

### METHODS

Posturography was used to study 13 patients with a preliminary diagnosis of peripheral neuropathy and 7 control subjects. All subjects were interviewed and received a detailed neurologic evaluation. Patients were randomly selected from those referred to our electrophysiology laboratory for investigation of polyneuropathy. All patients and control subjects received a standard electrophysiologic examination.

### Clinical Neurologic Evaluation

For all subjects, a standardized history was obtained, and a physical examination was performed. The specific symptoms of distal symmetric sensory loss, weakness, and autonomic abnormalities were solicited. Questions were also designed to exclude central nervous system disorders (history of stroke, cerebellar disease, or vestibular disease) that may affect balance. Medical or environmental causes of peripheral neuropathy

thy were screened for. Included in the physical examination were tests of peripheral sensation (position sense, vibration sense, pinprick sensation) and deep-tendon reflexes.

Findings that were abnormal in 2 of 3 categories—neuropathic symptoms, sensory deficits on examination, and impaired reflexes on examination—and that were attributable to a distal symmetric neuropathy constituted a definite abnormal neurologic examination indicating clinical neuropathy.

### Electrophysiologic Examination

Nerve conduction studies and needle EMG were performed on each patient according to the standard protocol (Oh, 1984). A handheld infrared thermometer was used to monitor temperature closely throughout the test, and temperature was kept above 32°C. We used the normal nerve conduction values for our laboratory (Table 1). Nerve conduction studies included but were not limited to sural amplitudes and latencies; peroneal motor amplitudes, distal latencies, and F responses and tibial motor amplitudes, distal latencies, F responses, and H reflexes. The needle EMG was performed to document the presence or absence of a gradient pattern of denervation–reinnervation in which distal muscles were more affected than proximal muscles. A patient was considered to have electrophysiologic evidence of polyneuropathy if abnormalities (decreased or absent amplitudes, increased late responses, evidence of denervation on needle EMG) were bilateral and involved at least 2 anatomically distinct nerves. Using this definition, we classified patients either as having or not having polyneuropathy. We did not address the quantitative nature of the polyneuropathy.

**TABLE 1.** Normal Nerve Conduction Values for Our Laboratory\*

Nerve	Latency, ms (upper limit of normal)	Amplitude, mV	Velocity, m/s (lower limit of normal)
<i>Motor nerve</i>			
Median	4.5	4.5–17.5	49
Ulnar	4	5.0–17.5	47
Tibial	7.0	2.5–14	37
Peroneal	6.5	2.5–11	39
<i>Sensory nerve (orthodromic)</i>			
Median		6–26	44
Ulnar		4–24	45
Sural		4–26	39

\*All sensory studies are orthodromic. Latencies depend on distances used, and upper limits of normal for motor studies in our laboratory are reported (there is no lower limit of normal for latencies).

### Posturographic Examination

Postural sway was measured using an AMTI Accu-Sway force platform (Advanced Mechanical Technology, Inc, Watertown, Mass) interfaced with a 486/33 computer and BEDAS-2 data acquisition and analysis software. COP data were collected during quiet standing with feet together and feet apart and with eyes open and eyes closed.

In the feet-together position, subjects stood barefoot with their heels together and with their medial sides forming a 20° angle; in the feet-apart position, subjects' heels were 10 cm apart, and their medial sides formed a 20° angle. For these trials, the force platform was marked to ensure that the positions were consistent. In the eyes-open trial, subjects focused on an eye-level mark made on a blank wall approximately 1 meter away; in the eyes-closed trial, subjects closed their eyes.

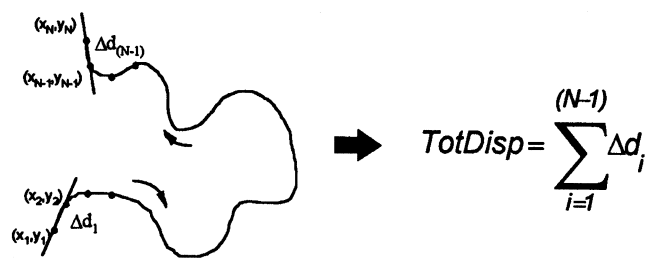
The resulting 4 positions were eyes open and feet apart (OA), eyes closed and feet apart (CA), eyes open and feet together (OT), and eyes closed and feet together (CT). Each trial lasted 30 seconds, and a 30-second rest period was allowed between trials. Longer rest periods were allowed between position changes. Data were sampled at a frequency of 50 Hz. Six to 10 trials were collected at each position. Position order was randomized. Descriptive analysis and diffusion analysis were performed to assess postural sway.

### Descriptive Analysis

Total displacement (TotDisp), the total distance traveled by the COP trajectory was measured for each trial (Fig. 1). (For details, see Lehman et al, 1990.) COP was under the feet of the subjects and was measured relative to the surrounding environment.

### Diffusion Analysis

Diffusion plots (plots of mean squared displacement of COP against increasing time intervals) were also measured for each trial (Fig. 2). Mathematical formu-



**FIGURE 1** Method for measuring total planar displacement (TotDisp) of N data points ( $x_1, y_1; x_2, y_2; \dots x_N, y_N$ ).

lae used in analysis were those outlined by Collins and De Luca (1993, 1995; Collins et al, 1995).

Diffusion plots have 2 regions. In the region over short-term time intervals, mean squared displacement COP changes rapidly with increasing time intervals; in the region over long-term time intervals, changes are slower with increasing time intervals (Collins and De Luca 1995; Collins et al, 1995; Mitchell et al, 1995). Straight lines were fitted over each of these regions. The point at which they intersected was defined as the critical point. Trials in which the critical point could not be clearly defined were discarded. We examined Ctime and Cdisp—the coordinates of the critical point—in detail.

Diffusion parameters have been used as indicators of neuromuscular function in posture (Collins and De Luca, 1995; Collins et al, 1995; Mitchell et al, 1995). Diffusion analysis considers the dynamic nature of COP, relating displacement to time. Location of the critical point is used to indicate the region in which postural control switches from a short-term open-loop control scheme to a long-term closed-loop control scheme (Mitchell et al, 1995). The temporal and spatial location of the critical point is used to differentiate patients and control subjects. Data were grouped by subject type and position. Analysis of variance was used to test for statistical significance. In

addition, the eyes-open position was compared with the eyes-closed position, and the feet-together position was compared with the feet-apart position.  $P < .05$  was used to describe significant differences.

## RESULTS (TABLE 2)

### Clinical and Electrophysiologic Results

Of the 13 randomly selected patients referred to our laboratory for investigation of peripheral neuropathy, 4 had both electrophysiologic and clinical evidence of large-fiber polyneuropathy, 2 had electrophysiologic but no clinical evidence of large-fiber polyneuropathy, and 7 had no electrophysiologic or clinical evidence of large-fiber polyneuropathy (see Methods section for definitions used for electrophysiologic and clinical evidence of polyneuropathy). These were the conclusions after examination and neurophysiological testing by us in the laboratory, even though each of the subjects had been referred for work-up of a peripheral neuropathy.

### Posturographic Results

#### Patients With Both Electrophysiologic and Clinical Evidence of Polyneuropathy Versus Control Subjects

Cdisp (amount of displacement before critical point is reached) was significantly greater in these patients than in the control subjects in all positions. Ctime (time before critical point is reached) did not differ statistically in any position. TotDisp (total displacement) was significantly greater in these patients than in the control subjects in all positions. (The control subjects' Cdisp and Ctime means and standard deviations were similar to those reported in other studies; Collins and De Luca, 1993, 1995.)

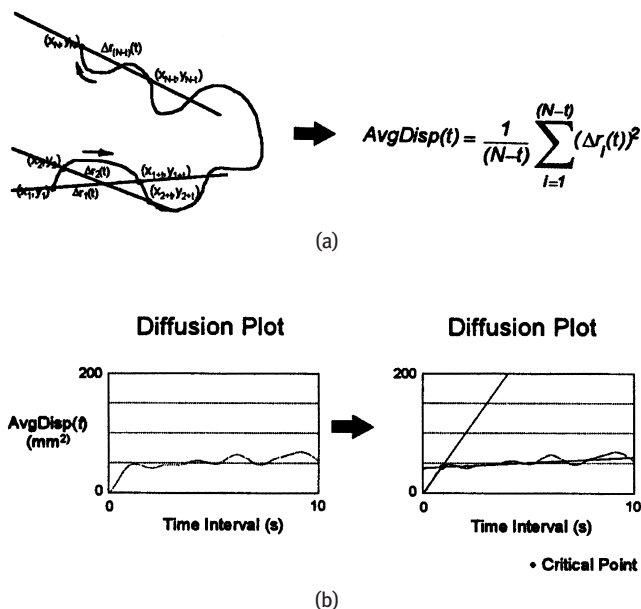
#### Patients With Electrophysiologic but No Clinical Evidence of Polyneuropathy Versus Control Subjects

Statistical tests were performed only for the CA position. The critical point could not be clearly defined in enough trials to perform statistical analysis for the other positions.

Cdisp was significantly greater in these patients than in the control subjects, but Ctime was not statistically different. TotDisp was greater in these patients than in the control subjects ( $P = .02$ ). Therefore, in comparisons with control subjects, these patients' results were the same as those of patients with both electrophysiologic and clinical evidence of polyneuropathy.

#### Patients With No Electrophysiologic or Clinical Evidence of Polyneuropathy Versus Control Subjects

Cdisp, Ctime, and TotDisp were not significantly different in these patients compared with control subjects



**FIGURE 2** (a) Method for measuring average squared planar displacement,  $AvgDisp(t)$ , as a function of time interval  $t$  for center of pressure of  $N$  data points  $(x_1, y_1; x_2, y_2; \dots, x_N, y_N)$ . (b) Diffusion plot resulting from plotting  $AvgDisp(t)$  over all intervals (0–10 seconds). The critical point (Ctime, Cdisp) is located at the intersection of the straight lines fitted to the 2 regions of the diffusion plot.

**TABLE 2** Group Mean, Standard Deviation, and Number of Trials for Each Parameter Within Each Subject Group

Parameter	Subject Group											
	E + C Evidence			E Evidence Only			No Evidence			Normal Control Subjects		
	Mean	SD	N	Mean	SD	N	Mean	SD	N	Mean	SD	N
Cdisp, mm <sup>2</sup>												
OA	77.72	63.71	28	18.03	16.52	35	15.90	12.31	44			
OT	105.84	79.62	30	24.28	14.10	19	30.35	26.47	67			
CA	83.73	46.87	19	78.68	40.25	14	19.55	16.73	17	18.69	12.06	36
CT	244.21	165.46	30	43.75	34.95	26	55.26	45.16	47			
Ctime, s												
OA	0.89	0.32	28	1.16	0.63	35	0.88	0.70	44			
OT	1.01	0.48	30	1.24	0.55	19	1.20	0.52	67			
CA	0.66	0.20	19	0.71	0.42	14	0.98	0.74	17	0.84	0.31	36
CT	0.96	0.50	30	0.94	0.34	26	1.07	0.42	47			
TotDisp, mm <sup>2</sup>												
OA	61.12	23.83	28	40.41	8.17	35	44.83	9.26	44			
OT	73.17	29.93	30	40.89	7.07	19	45.07	8.09	67			
CA	80.05	29.74	19	73.00	8.67	14	41.94	7.82	17	46.16	9.54	36
CT	116.33	50.70	30	50.56	7.68	26	53.98	9.55	47			

\*“E + C evidence” indicates both electrophysiologic and clinical evidence for polyneuropathy; “E evidence only,” electrophysiologic but no clinical evidence for polyneuropathy; “no evidence,” no electrophysiologic or clinical evidence for polyneuropathy; Cdisp, mean squared displacement; Ctime, interval; TotDisp, total planar displacement; OA, eyes open and feet apart; OT, eyes open and feet together; CA, eyes closed and feet apart; CT, eyes closed and feet together; dash, no data available.

in all positions. Therefore, the results of these patients—patients referred for polyneuropathy investigation—were the same as those of the control subjects (ie, there were no false-positives regardless of the referring diagnosis; cf results of conventional EMG).

### Eyes Open Versus Eyes Closed

In patients with both electrophysiologic and clinical evidence of polyneuropathy and in control subjects, Cdisp was always less with eyes open than with eyes closed—which was significant for these patients with feet together and for control subjects with feet apart. Ctime was always longer with eyes open than with eyes closed—which was significant for patients with both electrophysiologic and clinical evidence of polyneuropathy with feet apart and for control subjects with feet together. TotDisp was always less with eyes open than with eyes closed—which was significant except for patients with both electrophysiologic and clinical evidence of polyneuropathy with feet together. Although the statistical tests were not always significant, patients and control subjects tended to sense smaller displacements and to move overall shorter distances with eyes open.

### Feet Apart Versus Feet Together

In patients with both electrophysiologic and clinical evidence of polyneuropathy and in control subjects,

Cdisp was always less with feet apart than with feet together—which was significant except for patients with both electrophysiologic and clinical evidence of polyneuropathy with eyes open. Ctime was always less with feet apart than with feet together—which was significant except for patients with both electrophysiologic and clinical evidence of polyneuropathy with eyes open. TotDisp was always less with feet apart than with feet together—which was significant except for patients with both electrophysiologic and clinical evidence of polyneuropathy with eyes open. In general, patients and control subjects sensed smaller displacements over shorter intervals and moved overall shorter distances with feet apart.

### DISCUSSION

This pilot study confirms the hypothesis that people with polyneuropathy have a distinct standing sway pattern that distinguishes them from a control population without polyneuropathy.

Critical displacement (Cdisp) was significantly higher in patients with polyneuropathy than in control subjects. Clinically, this meant that, for patients with polyneuropathy, larger COP coordinate translations went undetected before being acted upon by the musculoskeletal system. In other words, the nervous system of patients with polyneuropathy is less

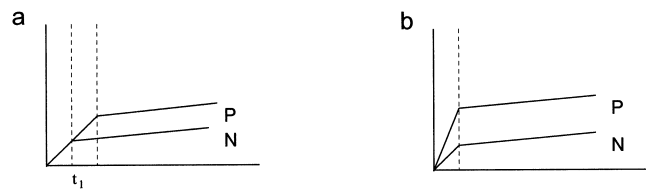
sensitive to changes in position of COP. As a result, COP wanders in a larger area before being detected and before the diseased peripheral nervous system attempts correction. This delay translates to a less tight swaying pattern and, consequently, poorer standing balance.

TotDisp also was significantly higher in patients with polyneuropathy, which means that COP wandered farther per unit of time in these patients than in control subjects. Critical time (Ctime), however, was not significantly different between the two groups. In other words, COP in the patients with polyneuropathy wanders not only farther but faster. Therefore, tighter control provided by healthy peripheral nerves limits total displacement and the speed of COP wandering. This control mechanism is defective in the patients with polyneuropathy. These findings are confirmed clinically when the position sense is examined in patients with polyneuropathy. A patient who has large-fiber polyneuropathy and who cannot detect small or slow changes in big-toe position may be able to detect larger or faster changes. This situation raises the question as to whether larger and faster displacement in COP is a compensatory mechanism that enables someone with polyneuropathy to detect changes in position of COP.

Also pertinent is that the short-term postural control scheme used by patients with polyneuropathy differs from that used by control subjects. We noted this when examining the location of the critical point in the diffusion plots (Fig. 3). Plot 3a represents 2 short-term control schemes that start off similarly but diverge at some time  $t_1$  and arrive at their respective critical displacements at different times, and plot 3b represents 2 short-term control schemes that differ at the start but arrive at their respective critical displacements at the same time. In plot 3a, P may be interpreted as a patient who has lost the ability to sense small displacements and who requires a larger displacement before switching from short- to long-term control schemes (thus, more time is required before switching control schemes); in plot 3b, patient P sways faster and reaches the larger critical displacement in the same amount of time. Plot 3b is consistent with our findings—which suggests that the short-term control scheme used by the patients in this study was different from that used by the control subjects.

Findings from this pilot study agree with findings from other investigations (Lehman et al, 1990; Collins and De Luca, 1995). Standing balance is worse with eyes closed and/or feet together than with eyes open and/or feet apart.

In this study, posturography was used to detect balance abnormalities in patients with electrophysiologic and clinical evidence of polyneuropathy. Postu-



**FIGURE 3** Diffusion plots representing possible differences between control schemes used by patients P and control subjects N. (a) Short-term open-loop control schemes start off similarly but diverge at some time  $t_1$  and arrive at their respective critical displacements at different times. (b) Short-term open-loop control schemes differ at the start but arrive at their respective critical displacements at the same time.

rography was also used to detect balance or sway-pattern abnormalities in patients who had polyneuropathy detected by conventional EMG but who had no symptoms or signs of neuropathy. The posturography results of patients with normal EMG results were the same as those of control subjects, which implies that posturography has the potential to become a sensitive screening test for large-fiber polyneuropathy—comparable to the gold standard of conventional EMG. In addition, for patients with polyneuropathy, posturography may be useful in conducting serial noninvasive evaluations and thus in assessing the results of therapeutic interventions. This benefit may be of special importance to elderly patients with polyneuropathy, a disease that increases the risk of falling. Posturography may also be used to assess the effects of walking aids such as canes and walkers (Tideiksaar, 1989; Ledin, 1990; Waespe et al, 1993; Simoneau et al, 1994; Richardson and Hurvitz, 1995).

This study is a pilot study with a few shortcomings. First, our patients had no symptoms or signs of abnormal visual or vestibular function, and we did not undertake any specialized testing of these systems. Visual function and vestibular function, however, should be tested in patients undergoing posturography, as abnormal function affects postural sway. Indeed, our data confirmed that vision is important in maintaining a tight sway pattern. Taking a full history and conducting a thorough examination should reveal any abnormalities in these systems. Suspected central abnormalities of the sensorimotor system can be confirmed by neuroimaging.

Second, we did not take into account age-related effects, and we did not age-match our patients and control subjects.

Third and last, statistical significance was reached in many parameters, but the power of this study would have been greatly enhanced if more study pa-

tients had been involved. Our patients constituted a random sample of patients referred to our laboratory, so, essentially, referred patients with no electrophysiologic or clinical evidence of neuropathy formed another control group. Results of these patients—referred for polyneuropathy investigation but showing no evidence of polyneuropathy on electrophysiologic and clinical testing—differed significantly from results of patients with electrophysiologic evidence of polyneuropathy.

Conventional EMG results and physical findings do not correlate well (Dyck, 1993; Bergin et al, 1995). Physicians sometimes consider low nerve conduction as evidence of severe neuropathy, even when the patient is without symptoms or signs. For instance, in nerve conduction studies of patients with Charcot-Marie-Tooth disease, slowing does not correlate with degree of weakness or functional performance. As opposed to conventional EMG, posturography is a functional test with results that reflect performance. Posturography may therefore be the test of choice for assessing functional deficits in patients with peripheral neuropathy—specifically, the deficits that increase the risk of falling. Posturography, however, cannot and should not replace conventional EMG, as posturography cannot differentiate types of peripheral neuropathy. On detection of polyneuropathy, conventional EMG should be performed to determine its nature.

## REFERENCES

- Bergin PS, Bronstein AM, Murray NMF, et al. Body sway and vibration perception thresholds in normal aging and in patients with polyneuropathy. *J Neurol Neurosurg Psychiatry*. 1995;58:335–340.
- Collins JJ, De Luca CJ. Open-loop and closed-loop control of posture: a random-walk analysis of center-of-pressure trajectories. *Exp Brain Res*. 1993;95:308–318.
- Collins JJ, De Luca CJ. The effects of visual input on open-loop and closed-loop postural control mechanisms. *Exp Brain Res*. 1995;103:151–163.
- Collins JJ, De Luca CJ, Burrows A, Lipsitz LA. Age-related changes in open-loop and closed-loop postural control mechanisms. *Exp Brain Res*. 1995;104:480–492.
- Dalakas MC. Chronic idiopathic ataxic neuropathy. *Ann Neurol*. 1986;19:545.
- Dyck PJ. Quantitative severity of neuropathy. In: Dyck PJ, et al. *Peripheral Neuropathy*. Vol 1, 3rd ed. Philadelphia, PA: Saunders; 1993:686.
- Dyck PJ, Lambert EH, Nichols PC. Quantitative measurement of sensation related to compound action potential and number and sizes of myelinated and unmyelinated fibers of sural nerve in health, Friedreich's ataxia, hereditary sensory neuropathy, and tabes dorsalis. In Cobb WA, ed. *Handbook of Electroencephalography and Clinical Neurophysiology*. Vol 9. Amsterdam, Netherlands: Elsevier; 1972:83.
- Jamal GA, Donaghy M. A peripheral mechanism for the ataxia associated with the Miller Fisher syndrome of acute ophthalmoplegia. *J Neurol Neurosurg Psychiatry*. 1989;52:1210.
- Kimura J. Nerve conduction studies and electromyography. In: Dyck PJ, et al. *Peripheral Neuropathy*. Vol 1, 3rd ed. Philadelphia, PA: Saunders; 1993:598.
- Ledin T. Effects of balance training in elderly evaluated by clinical tests and dynamic posturography. *J Vestib Res*. 1990;1:129–138.
- Lehman JF, Boswell S, Price R, et al. Quantitative evaluation of sway as an indicator of functional balance in post-traumatic brain injury. *Arch Phys Med Rehabil*. 1990;71:955–962.
- Mitchell SL, Collins JJ, De Luca CJ, et al. Open-loop and closed-loop postural control mechanisms in Parkinson's disease: increased mediolateral activity during quiet standing. *Neurosci Lett*. 1995;197:133–136.
- Oh SJ. *Clinical Electromyography: Nerve Conduction Studies*. Baltimore, MD: University Park Press; 1984.
- Purdham D, Evans JG. Factors associated with falls in the elderly: a community study. *Age Ageing*. 1981;10:141–146.
- Richardson JK, Ching C, Hurvitz EA. The relationship between electromyographically documented peripheral neuropathy and falls. *J Am Geriatr Soc*. 1992;40:1008–1012.
- Richardson JK, Hurvitz EA. Peripheral neuropathy: a true risk factor for falls. *J Gerontol A Biol Sci Med Sci*. 1995;50:M211–M215.
- Simoneau GG, Ulbrecht JS, Derr JA, et al. Postural instability in patients with diabetic sensory neuropathy. *Diabetes Care*. 1994;17:1411–1421.
- Tideiksaar R. Geriatric falls: assessing the cause, preventing recurrence. *Geriatrics*. 1989;44:57–61, 64.
- Waespe W, Hafner M, Diener R, Bachli E. Differential diagnosis of chronic gait disorders of neurologic origin in old age [in German]. *Schweiz Med Wochenschr*. 1993;123:317–327.

**EDITOR**

Keith H. Chiappa, MD

**ASSOCIATE EDITOR**

Didier Cros, MD

**ELECTRONIC MAIL**

chiappa@helix.mgh.harvard.edu

*Neurology and Clinical Neurophysiology* is a peer-reviewed and electronically published scholarly journal that covers a broad scope of topics encompassing clinical and basic topics of human neurology, neurosciences and related fields.

**EDITORIAL BOARD**

Robert Ackerman  
*Massachusetts General Hospital, Boston*

Barry Arnason  
*University of Chicago*

Flint Beal  
*Massachusetts General Hospital, Boston*

James Bernat  
*Dartmouth-Hitchcock Medical Center,  
New Hampshire*

Julien Bogousslavsky  
*CHU Vaudois, Lausanne*

Robert Brown  
*Massachusetts General Hospital, Boston*

David Burke  
*Prince of Wales Medical Research Institute,  
Sydney*

David Caplan  
*Massachusetts General Hospital, Boston*

Gegory Cascino  
*Mayo Clinic, Rochester*

Phillip Chance  
*The Children's Hospital of Philadelphia,  
Philadelphia*

Thomas Chase  
*NINDS, National Institutes of Health, Bethesda*

David Cornblath  
*Johns Hopkins Hospital, Baltimore*

F. Michael Cutrer  
*Massachusetts General Hospital, Boston*

David Dawson  
*Brockton VA Medical Center, Massachusetts*

Paul Delwaide  
*Hôpital de la Citadelle, Liege*

John Donoghue  
*Brown University, Providence*

Richard Frith  
*Auckland Hospital, New Zealand*

Myron Ginsberg  
*University of Miami School of Medicine*

Douglas Goodin  
*University of California, San Francisco*

James Grotta  
*University of Texas Medical School, Houston*

James Gusella  
*Massachusetts General Hospital, Boston*

John Halperin  
*North Shore University Hospital / Cornell  
University Medical College*

Stephen Hauser  
*University of California, San Francisco*

E. Tessa Hedley-White  
*Massachusetts General Hospital, Boston*

Kenneth Heilman  
*University of Florida, Gainesville*

Daniel Hoch  
*Massachusetts General Hospital, Boston*

Fred Hochberg  
*Massachusetts General Hospital, Boston*

John Hoffman  
*Emory University, Atlanta*

Gregory Holmes  
*Children's Hospital, Boston*

Bruce Jenkins  
*Massachusetts General Hospital, Boston*

Ryuji Kaji  
*Kyoto University Hospital*

Carlos Kase  
*Boston University School of Medicine, Boston*

J. Philip Kistler  
*Massachusetts General Hospital, Boston*

Jean-Marc Léger  
*La Salpêtrière, Paris*

Simmons Lessell  
*Massachusetts Eye and Ear Infirmary, Boston*

Ronald Lesser  
*Johns Hopkins Hospital, Baltimore*

David Levine  
*New York University Medical Center*

Ira Lott  
*University of California, Irvine*

Phillip Low  
*May Clinic, Rochester*

Richard Macdonell  
*Austin Hospital, Victoria, Australia*

Joseph Masdeu  
*St. Vincent's Hospital, New York*

Kerry R. Mills  
*Radcliffe Infirmary, Oxford*

José Ochoa  
*Good Samaritan Hospital, Portland*

Barry Oken  
*Oregon Health Sciences University, Portland*

John Penney  
*Massachusetts General Hospital, Boston*

Karlheinz Reiners  
*Bayerische Julius-Maximilians-Universität,  
Würzburg*

Allen Roses  
*Duke University Medical Center, Durham*

Thomas Sabin  
*Boston City Hospital, Boston*

Raman Sankar  
*University of California at Los Angeles*

Joan Santamaria  
*Hospital Clinic Provincial de Barcelona*

Kenneth Tyler  
*University of Colorado Health Science Center,  
Denver*

Francois Viallet  
*CH Aix-en-Provence*

Joseph Volpe  
*Children's Hospital, Boston*

Michael Wall  
*University of Iowa, Iowa City*

Stephen Waxman  
*Yale University, New Haven*

Wigbert Wiederholt  
*University of California, San Diego*

Eelco Wijdicks  
*Mayo Clinic, Rochester*

Clayton Wiley  
*University of California, San Diego*

Anthony Windebank  
*Mayo Clinic, Rochester*

Shirley Wray  
*Massachusetts General Hospital, Boston*

Anne Young  
*Massachusetts General Hospital, Boston*

Robert Young  
*University of California, Irvine*