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Diabetes and Postural Stability: Review and Hypotheses

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ABSTRACT. Among the complications associated with diabetes mellitus is postural control. The authors reviewed 28 studies in the literature that focused on the magnitudes of postural sway that people with and without diabetes exhibit. The general observation is that postural sway is greater for people with diabetes, especially if their condition includes neuropathy. Peripheral sensory neuropathy seems to be the primary factor, but the available evidence does not rule out diabetes per se, other neuropathies (central, autonomic, motor), or an inability to exploit fully optical and inertial information about posture. The authors' review raises the issue of foot disorders and the possibility of increased sway as a useful adaptation; it also calls for better neuropathy assessments, postural tasks, and measures.

Keywords: diabetes complications, information integration, neuropathy, noise, postural strategies

Diabetes mellitus is a disorder characterized by hyperglycemia resulting from issues in insulin production, insulin action, or both (Cimbiz & Cakir, 2005). Diabetes mellitus[1] is characterized by many symptoms including frequent urination, extreme thirst, weight loss, increased hunger, blurred vision, irritability, and extreme unexplained fatigue (e.g., Walker, 2005). Worse than the symptoms of chronic hyperglycemia are the complications. Most notable, neuropathy (literally, disease of the nerves) leads to a global destruction of the body over the years. Long-term diabetes is also associated with macro- and microvascular disease, retinopathy (eye disease), arteriosclerosis (vessel disease), nephropathy (kidney disease), digestive disorders, infections, numbness, abnormal sensations, pain, cramping, hair loss, and muscle weakness (Cavanagh, Simoneau, & Ulbrecht, 1993; Dyck, 1988). Peripheral nerve damage affects up to 25% of people with diabetes after 10 years, up to 50% after 20 years, and up to 70% after 30 years (Simmons, Richardson, & Pozos, 1997).

Diabetes mellitus exacts a heavy cost not only for individuals, but also for the health care system. The total economic burden of diabetes in the United States in 2002 was approximately $132 billion (American Diabetes Association, 2003). Much time and effort is spent to improve the lives of people with diabetes in an effort to help them avoid or overcome the dependency that is often brought about by complications of the disease. One particularly salient difficulty provided our focus. Compared with healthy people, those with diabetic neuropathy have an increased risk of falls by a factor of 15 (Cavanagh, Derr, Ulbrecht, Maser, & Orchard, 1992); in general, older adults also have a high incidence of serious injury as a result of falls (Tinetti, Speechley, & Ginter, 1988). The fact that people in the diabetic population sometimes have a subjective feeling of instability (Greene, Sima, Albers, & Pfeifer, 1990) suggests a consequence of the disease on postural control. Postural instability has been mentioned in the literature for at least 40 years (cf., Greenbaum, Richardson, Salmon, & Urich, 1964).

In this review, we consider research on the relation between diabetes mellitus (with or without neuropathy) and posture (its sway and control). If there is a positive relation between postural sway and falling (e.g., Campbell, Borrie, & Spears, 1989), then research directed at people with diabetes could suggest how to help prevent falls and injuries (Corriveau et al., 2000) and provide methods for monitoring changes in the pattern of sways over time (Ahmmed & Mackenzie, 2003). Given that the number of people with diabetes increases every year (Engelgau et al., 2004), such research is of increasing importance.

Our review covers the postural behavior of people with diabetes standing on two feet in the standard configuration (i.e., feet side by side). The experimental task is frequently that of unperturbed or quiet standing—the behavior of standing upright in one place without any additional behavioral requirements or changes in forces external to the body. In unperturbed or quiet standing, a person is never perfectly still (e.g., Hinsdale, 1887). The body fluctuates continuously, albeit at the scale of millimeters. For simplicity, we introduced the label *static* for the protocol of quiet standing and the label *dynamic* for departures from that protocol (i.e., additional behaviors, changes in external forces).

Our review does not examine falls or gait directly (cf. Van Deursen & Simoneau, 1999). We did not include questionnaire data, case studies, data from artificially induced neuropathy, or data from diseases other than diabetes. We also omitted dissertations, abstracts, and papers not written in English. In all, 28 studies over the past 45 years fell within these limitations (see Table 1). These cover Type I, or insulin-dependent diabetes (developed when the body's immune system destroys pancreatic beta cells and therefore eliminates the production of insulin), and Type II, or noninsulin-dependent diabetes (caused by an insufficient production of insulin or a relative insensitivity of the cells to the action of insulin; Engelgau et al., 2004). Gestational

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TABLE 1. Number of Participants Per Group, Time Per Trial, and Experimental Manipulations Beyond Static Eyes Open–Eyes Closed EO–EC for Each Cited Article

Citation	Number of participants			Time (s)	Manipulations beyond static EO–EC
	DN	D	HC		
A. U. Ahmmed & I. J. Mackenzie (2003)	15	15	15	20	—
P. S. Bergin, A. M. Bronstein, N. M. F. Murray, S. Sancovic, & K. Zeppenfeld (1995)	25	—	32	60	Firm or foam surface
P. Boucher, N. Teasdale, R. Courtemanche, C. Bard, & M. Fleury (1995)	12	—	7	30	EC to EO during a trial
P. R. Cavanagh, G. G. Simoneau, J. S. Ulbrecht (1993)	16	16	16	30	EO
H. Centomo, N. Termoz, S. Savoir, L. Beliveau, & F. Prince (2007)	—	15	15	30	Reaching during standing; EO
H. Corriveau et al. (2000)	15	—	15	120	—
R. Dickstein, R. J. Peterka, & F. B. Horak (2003)	8	—	10	8	Light, heavy, or no touch with platform translation; EC
R. Dickstein, C. L. Shupert, & F. B. Horak (2001)	8	—	8	40	Light, heavy, or no touch; firm or foam surface
W. M. Di Nardo et al. (1999)	14	28	24	20	Fixed or moving ground; sway-referenced optics
P. Giacomini et al. (1996)	10	23	21	90	—
F. B. Horak, R. Dickstein, & R. J. Peterka (2002)	13	—	12	40	Sway-referenced platform motion
F. B. Horak (2001)	8	—	8	6	Firm or foam surface; galvanic stimulation; EO
E. C. Katoulis et al. (1997)	40	20	20	30	—
B. J. Kim & C. J. Robinson (2006)	6	—	7	4	Platform translation; EO
D. Lafond, H. Corriveau, & F. Prince (2004)	11	—	20	120	—
L. A. Lavery et al. (1998)	26	—	—	20	Footwear; EO
S. R. Lord, G. A. Caplan, R. Colagiuri, & J. A. Ward (1993)	—	25	40	30	Firm or foam surface
Y. Mimori et al. (1982)	75	13	44	30	—
A. Nardone & M. Schieppati (2004)	22	—	13	51	Stance width; surface rotation
A. Nardone et al. (2006)	14	—	20[a]	20, 51	Sinusoidal platform motion
U. Oppenheim, R. Kohen-Raz, D. Alex, A. Kohen-Raz, & M. Azarya (1999)	20	8	30[a]	—	Head position; firm/elastic surface
A. A. Priplata et al. (2006)	15	—	12[a]	30	Subsensory mechanical noise; EC
R. W. Simmons, C. Richardson, & R. Pozos (1997)	23	27	50	20	Sway-referenced optics and platform
G. G. Simoneau, J. S. Ulbrecht, J. A. Derr, M. B. Becker, & P. R. Cavanagh (1994)	17	17	17	30	Head position; straight or back
G. G. Simoneau, J. S. Ulbrecht, J. A. Derr, & P. R. Cavanagh (1995)	17	17	17	30	Head position; straight or back
L. Uccioli et al. (1995)	10	23	21	90	—
L. Uccioli et al. (1997)	7	18	31	90	—
R. Yamamoto et al. (2001)	32	123	55	60	—

Note. EO = eyes open only; EC = eyes closed only; HC = healthy controls; D = people with diabetes but without diabetic neuropathy; DN = people with diabetes and diabetic neuropathy.
[a]Included nondiabetic clinical controls.

diabetes, which occurs in 4% of pregnancies (Engelgau et al.), has not been included in investigations of postural stability and, therefore, is not included in the present article.

Although the various authors we reviewed do not necessarily make their hypotheses explicit, we have identified three broad categories of what may be conjectures about the source of instability in diabetic populations; namely, diabetes per se, peripheral sensory neuropathy, and inadequate information processing. We also suggest two new hypotheses that may frame future investigations. One

hypothesis reflects biomechanical concerns; the other reflects the contemporary understanding that noise plays a constructive role, promoting order and enhancing or initiating existing processes. The participant populations under consideration were people with diabetes but without neuropathy (D), people with diabetes who present with neuropathy (DN), healthy controls (HC), and people with clinical disorders other than diabetes (Cl). Two previous reviews of D and DN relative to HC and Cl were restricted to 6 (Van Deursen & Simoneau, 1999), and 4 (Horak, 2001) of the 28 articles reviewed in the present article.

First Observations

Mimori et al. (1982) appear to be the first researchers to have recorded postural sway of people with diabetes. They compared DN, D, and HC (although they did not describe how the presence of neuropathy was diagnosed). Their sway measure was the area covered by motions of the center of pressure (COP) collected by a transducer attached to the floor. Four findings are notable:

1. COP area for DN was significantly larger than for D and HC, who did not differ from one another.
2. The duration of the disease increased COP area significantly in DN (i.e., people having neuropathy for more than 10 years exhibited broader COP motions than people having neuropathy for 5–10 years).
3. People treated with oral hypoglycemic drugs fluctuated significantly more than people not treated with these drugs.
4. People with paresthesia (abnormal sensation with no apparent cause) fluctuated significantly more than people with hypesthesia (diminished sensation).

On the basis of case studies from Greenbaum et al. (1964), Mimori et al. (1982) suggested that the most important contributors to the increased postural sway of DN were age and neuropathy at the level of the CNS. However, the larger span of COP motions for DN compared with HC was limited to participants older than 65 years of age, clouding the interpretation of neuropathy's role. Moreover, the significant roles of abnormal sensation and medication further complicated the interpretation. In summary, it is not possible to discern which of the measured factors was important in explaining the amplified postural sway of DN.

Hypothesis 1 (H_1): Diabetes per se amplifies postural sways.

The early finding that D and HC are alike in the magnitude of postural sway is contradicted in only three studies. Lord, Caplan, Colagiuri, and Ward (1993) evaluated the role of age in the comparison of D and HC, particularly, with respect to sensory-motor functions (assessed with tests of touch pressure, vibration sense, proprioception, and quadriceps strength). Degree of postural motion was recorded using a simple sway meter attached at the level of the waist. Participants stood on a rigid surface or on foam, with eyes open or closed. (It should be noted that when a deformable surface has been placed on a force platform—in the present study and in a number of studies we reviewed—the accelerations and, thus, the forces measured by the platform may not have been representative of an individual's COP motion.) D exhibited significantly greater sway (defined as path length) than HC. D also had worse scores than HC for most of the sensory-motor function tests. Pearson correlation analyses revealed that age was associated with a variety of measures for D (e.g., vibration perception threshold [VPT] and quadriceps strength for female individuals; touch threshold and quadriceps strength for male individuals), but almost none for HC (only with quadriceps strength for female individuals). Lord et al. echoed the view of Mimori et al. (1982) that age is an important factor in explaining instability, with additional influences of sensory-motor impairment (peripheral neuropathy) and distal muscle weakness. However, it should be noted that one half of the D sample in Lord et al.'s study was being treated with oral hypoglycemic agents, which are possible contributors to COP motion (Mimori et al.). Moreover, the criteria to diagnose and rule out neuropathy were not rigorous: The D group did not have symptomatic neuropathy. Given the significant reduction of group members' touch threshold, VPT, and proprioception compared with HC, it is possible that neuropathy may have impaired some participants in this group.

Centomo, Termoz, Savoir, Beliveau, and Prince (2007) presented a different case in defense of the D hypothesis. Whether an experiment reveals D = HC (i.e., no significant sway difference exists between D and HC) or D > HC (i.e., D sway is significantly greater than HC sway) depends on the task. Whereas a simple postural requirement may yield D = HC, a more difficult postural requirement may not. In Centomo et al., D and HC participants performed a 30-s far-reaching task in the anterior–posterior (AP) axis. The task comprised three phases. For the first 15 s, participants stood with their right arm at shoulder height (Phase 1). For the next 3 s, they made their maximal far reach (Phase 2). For the final 12 s, they kept the far reach as still as possible (Phase 3). The COP-dependent variables were path length, mean velocity, root mean square (RMS) amplitude, and range of motion in the AP and mediolateral (ML) axes. In Phase 1, D = HC for all measures except COP range; in Phase 2, D = HC in reaching and all COP measures except range; and in Phase 3, D > HC for COP velocity, amplitude, and range. Centomo et al. suggested that weaker ankle torques caused by neuromuscular deficits in the case of D may have been the reason for the latter outcome.

Centomo et al. (2007) questioned whether their diagnosis of neuropathy was sufficiently rigorous. Their D participants may have had mild neuropathy that was not detectable by the discrimination test they used (the Semmes–Weinstein monofilament test [Kamei et al., 2005]

applied to four plantar sites; cf., Smieja et al., 1999) but that was potentially detectable by electrophysiological measures. However, Centomo et al.'s finding of D > HC is consistent with other indications of D's amplified sway when the postural task is more demanding (see Oppenheim, Kohen-Raz, Alex, Kohen-Raz, & Azarya, 1999, the third study suggesting D > HC that is reviewed below). Centomo et al.'s finding is also consistent with a summary observation that in the studies in which postural sway orders as DN > D and D = HC, often differences (COP motion, body motion, electrophysiological, and clinical) between DN and D are numerically smaller than between DN and HC.

H_2: Peripheral sensory neuropathy amplifies postural sway.

The understanding that measures of postural sway among participants order as DN > D and D = HC has directed attention to the postural consequences of diminished peripheral sensory capability. Such attention is well motivated because of basic empirical findings indicating that postural stability depends more on the somatosensory system than on the visual and vestibular systems (Simoneau, Ulbrecht, Derr, Becker, & Cavanagh, 1994; Simoneau, Ulbrecht, Derr, & Cavanagh, 1995[2]; see Figure 1).

Precise determination of the basis for a diminished peripheral sensory capability is not straightforward. The peripheral sensory system is complex, supporting distinct perceptual subsystems of cutaneous, haptic, and dynamic (or effortful) touch involving mechanoreceptors in skin, muscles, tendons, and ligaments (Gibson, 1966). Researchers have sought to identify relations between impairments in the mechanoreceptor basis of one or more of these subsystems and the amplification of postural sway exhibited in the comparison of DN and D. We set the stage for our review of the relevant literature through the experiment of Uccioli et al. (1995). Of potential importance, as Uccioli et al. made evident, are the measures used to assess the state of the peripheral sensory systems. The measures tend to partition into two familiar groups of quantitative sensory testing and nerve conduction studies (Perkins & Bril, 2003): (a) tests or measures of sensory discrimination and (b) tests or measures of neural responsiveness. Measures of sensory discrimination are less standardized and more subjective than measures of neural responsiveness; in particular, they are more involving of the person's awareness of his or her sensory capabilities. Measures of sensory discrimination are in the psychophysical tradition, with a focus on thresholds and the quantification of a person's judgments about differences. In contrast, measures of neural responsiveness are in the electrophysiological tradition, with a focus on the delay with which a nerve reacts to a stimulus and on the strength and speed of the conducted signal. The measures of neural responsiveness are strictly by instruments. A pertinent question is whether the two classes of measures differ in their prediction of the difference in postural sway between DN and D.

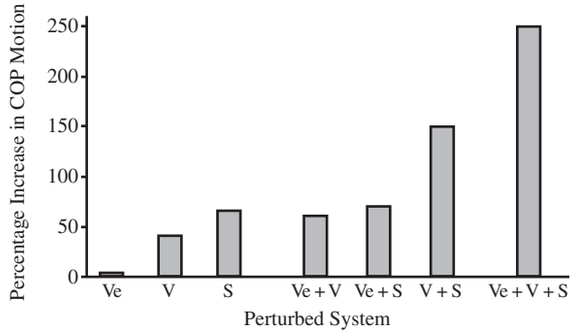

FIGURE 1. Percentage increase in center of pressure (COP) excursion because of impaired somatosensory system (S; diabetic neuropathy); eliminating vision (V; eyes closed); and reducing vestibular efficacy (Ve; head tilted back). Healthy controls are represented by all bars without S. Adapted with permission from G. G. Simoneau, J. S. Ulbrecht, J. A. Derr, & P. R. Cavanagh (1995).

Uccioli et al. (1995) assessed neuropathy with the San Antonio Consensus Conference guidelines (American Diabetes Association, 1988). Postural sway of HC, D, and DN were recorded on a force platform. DN exhibited significantly greater sway compared with D and HC for COP path length, area, and mean velocity. DN also had significantly poorer measures than D and HC (who did not differ) on the VPT test of both halluces (at the malleolus and hallux) and on latency, amplitude, and velocity measures of sural and peroneal nerves. The measures of COP area, path length, and mean velocity correlated significantly with 7 of the 12 sensory discrimination measures and 18 of the 36 neural responsiveness measures.

Impaired Sensory Discrimination as the Basis of Increased Postural Instability of DN

Cavanagh et al. (1993) distinguished D and DN by VPT (Bloom, Till, Sonksen, & Smith 1984). A force platform registered postural sway (indexed by several measures such as COP path length, range in the AP and ML axes, mean power frequency) in eyes-open and -closed conditions for HC, DN, and D. Cavanagh et al. found that DN swayed significantly more than D or HC in five of the seven sway measures. Although Cavanagh et al. examined many measures of somatosensory, vestibular, visual, motor, and functional impairment (see Cavanagh et al.'s Table 1), only the sensory discrimination measure of lower limb neuropathy related significantly to the differences in sway among DN, D, and HC.

In an effort to isolate the best sensory discrimination predictor of instability, Simoneau et al. (1994) focused on COP path length for HC, DN, and D. Neuropathy was assessed with the VPT test (Bloom et al., 1984). In all conditions, DN fluctuated significantly more than HC or D, who did not differ from one another. DN sway in the easiest condition (eyes open, head straight) was of

the same magnitude as HC sway in the most difficult condition (eyes closed, head back). COP path length (during eyes open, head straight condition) was regressed on sensory discrimination measures (e.g., VPT), neural responsiveness measures (e.g., sensory latency response of the sural nerve), and physical characteristics (e.g., age). The only significant models involved age in conjunction with a sensory discrimination measure: Age and VPT, age and Semmes–Weinstein monofilament, and age and joint motion perception threshold accounted for 53.6%, 54.7%, and 51.9% of the variance, respectively.

Both subjective and objective measures of neuropathy were used in Bergin, Bronstein, Murray, Sancovic, and Zeppenfeld's (1995) study. COP path length in the AP axis was recorded with the participants' eyes open or closed while the participants stood on a rigid or foam surface for HC and DN. Neuropathy was characterized objectively by an electrophysiological assessment (see Donofrio & Albers, 1990) and subjectively by VPT assessed with a neurothesiometer, a semiquantitative tuning fork, and the bone vibrator of a conventional audiometer. HC and DN differed in the VPT scores at the ankle and tibia as assessed by all three methods. For DN, the correlations between COP path length in the AP axis and VPT were significant for all four combinations of open or closed eyes and rigid or foam surface; for HC, the only significant correlation occurred with eyes open and participant standing on a rigid surface. Neither group exhibited any significant correlation between neural responsiveness measures and COP indexes of postural sway.

A subsequent division of DN into participants with axonal neuropathy and participants with demyelinating neuropathy revealed that the correlations between VPT and COP path length in the AP axis held only for DN axonal neuropathy. (The two DN subgroups were indistinct with respect to mean VPT and the variable of sway in the four conditions.) Bergin et al. (1995) concluded that tests of sensory discrimination are better than tests of neural responsiveness as indicators of instability. Bergin et al. supposed that impaired sensation of vibration could be related to axonal neuropathy, but no specific analysis was provided to support the supposition.

A related study (Simmons et al., 1997) distinguished D and DN on the basis of the Semmes–Weinstein monofilament test (see Holewski, Stess, Graf, & Grunfeld, 1988) from their HC counterparts recruited to match in weight, gender, and age. Center-of-gravity displacement, normalized to each participant's height, was estimated with a measure of the COP displacement from dual-force platforms during the six conditions of the Sensory Organization Test (see Figure 2). The difficulty of maintaining balance was manipulated as a function of whether eyes were open or closed, whether the support surface was fixed or yoked to COP motion, and whether a visual surround was fixed or yoked to COP motion.[3] DN swayed more in all conditions according to the nonparametric

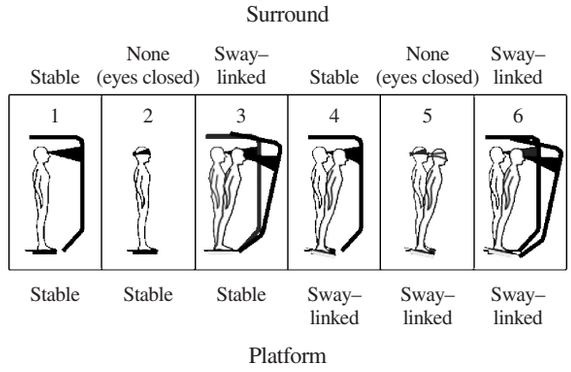

FIGURE 2. The six elements of the Sensory Organization Test. Adapted with permission from W. M. Di Nardo et al. (1999).

tests. Postural control was addressed by means of quantifications of muscle activation associated with *ankle* and *hip strategies* (with a score based on the shear force of 25 lb). DN used the hip strategy significantly more than did their corresponding HC in the two most difficult conditions (i.e., when both somatosensory and visual information were compromised). Simmons et al. concluded that the shift in postural strategy from ankle to hip by DN could be a consequence of diminished sensation at the feet. A change in postural strategy was also found by Giacomini et al. (1996), and we discuss that finding.

Because nonspecific aspects of diabetes (e.g., high blood pressure, loss of hearing) may affect balance, albeit indirectly, one must be cautious in attributing instability to neuropathy. Oppenheim et al. (1999) examined stability in Cl (in four clinical subgroups, each having disorders other than diabetes) in addition to DN, D, and HC. D and DN were distinguished on the basis of the method proposed by Dyck, Melton, O'Brien, and Service (1997). Dyck et al. used a system of four force platforms to record sway, quantified by COP path length and the Fourier power spectrum analysis (in four bands of frequency: 0.01–0.1, 0.1–0.5, 0.5–1, and 1–3 Hz; cf. Cavanagh et al., 1993). Four combinations of eyes open or closed while participant stands on a rigid surface or elastic pad were augmented with four head positions (right, left, up, down), all with the eyes closed while participant stands on a rigid surface. Although DN sway was greater than HC and D sway in some conditions, Cl sway was also greater than HC sway in those same conditions. Postural sway of D + DN (taken together as a group) exceeded Cl sway only with eyes closed and the head turned right or left. For these last two conditions, the power of sway in the 0.5–1-Hz frequency band distinguished D + DN from Cl and HC (and, importantly, also distinguished D from HC; see Figure 3). This circumscribed domain of difference highlights the delicacy of ascribing instability to specific deficits of the peripheral sensory system in the absence of appropriate, perhaps subtle, controls. The

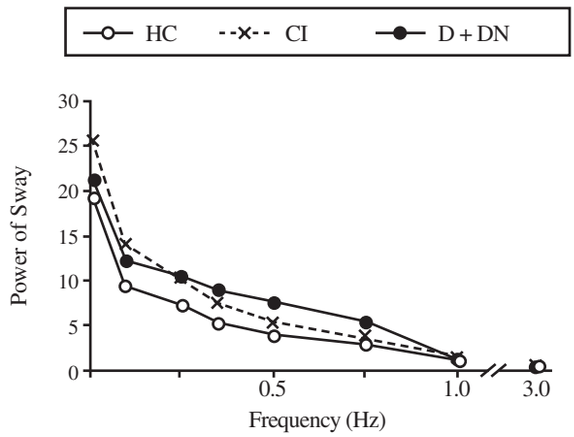

FIGURE 3. Fourier spectral pattern of postural sway in healthy controls (HC), clinical controls (Cl), and people with diabetes (D). Adapted with permission from U. Oppenheim, R. Kohen-Raz, D. Alex, A. Kohen-Raz, & M. Azarya (1999).

difference between D + DN participants and other participants in the 0.5–1-Hz range could be tied to the degree of peripheral neuropathy on the basis of results reported by de Wit (1972), Taguchi (1978), and Kohen-Raz (1991), suggesting that 0.5–1 Hz reflects somatosensory-based postural adjustments mediated by the lower limbs.

Severity of neuropathy was the focus of Corriveau et al.'s (2000) study. HC were compared with DN, whose neuropathy were diagnosed and quantified as mild, moderate, or severe using the method of Valk and colleagues (Valk, Nauto, Striners, & Bertelsman, 1992; Valk et al., 1997). DN scored significantly worse than HC in all sensory tests (VPT at the first toe, Semmes–Weinstein monofilament at the first toe, and Valk's scale). The comparison was in terms of a particularly sensitive postural sway measure; namely, RMS of the arithmetic difference COP minus center of mass (COP–COM). A force platform recorded COP displacement; anthropometric tables served to estimate the corresponding COM displacement. Overall DN sway was greater than HC sway in the AP and ML axes regardless of whether eyes were open or closed. Moreover, the difference persisted in all but the COP–COM in the AP axis in the eyes-open condition when those with severe neuropathy were removed from DN. Last, correlations between the severity of neuropathy and COP–COM amplitude were significant in the AP and ML axes.

Impaired Neural Responsiveness as the Basis of Increased Postural Instability of DN

Di Nardo et al. (1999) addressed whether the deficit in the peripheral sensory system alone explains the instability of DN. Di Nardo et al. compared HC, D, and DN, who were selected on the basis of the San Antonio Consensus Conference guidelines (American Diabetes Association, 1988). The six conditions of the Sensory Organization Test (see Figure 2) were augmented with six dynamic posturography conditions involving brief unexpected movements of the platform with eyes open: Three different amplitudes (small, medium, large) could be in two directions (backward, forward). The different trials were performed on dual-force platforms. For the sensory manipulations, the COP measure was the normalized range of COP motion in the AP axis compared with the maximum possible range (normalized with the maximum theoretical range of COP motion, which is 12.5° of inclination; cf. Diener, Horak, & Nashner, 1988). For the motor manipulations, postural response measures were the latency, amplitude, and symmetry of muscle activations after the perturbation. DN swayed significantly more than HC (for Elements 1, 2, 3, and 6 of Figure 2) and D (for Elements 1 and 2 of Figure 2), but HC and D did not differ. The difference for Sensory Organization Test 6 was unexpected and, although it implies a vestibular problem for DN, Di Nardo et al. were skeptical because their participants had normal electronystagmography scores. The dynamic response conditions revealed only one group difference: Muscle responses latencies for DN were longer than those for HC for small forward perturbations. Although the subjective VPT scores did not differ, the objective sural and peroneal nerve conduction velocities were significantly reduced in DN when compared with HC.

In the large study conducted by Yamamoto et al. (2001), the two diabetic groups were distinguished on the basis of neural responsiveness. Sway data (in the form of area, path length, standard deviation in the AP and ML axes, and Romberg Quotient) were recorded with an Anima gravicorder. DN fluctuated significantly more than both D and HC for sway area and path length. Yamamoto et al. examined 166 correlations between sway measures and various individual characteristics and electrophysiological measures (from both sensory and motor nerves, and power spectrum analysis of heart rate, respectively). It is difficult to discern a pattern because Yamamoto et al. did not provide a rationale for the particular relations that they examined. We can only say that they found significance in 32 of 45 correlations involving motor nerves, 6 of 36 correlations involving sensory nerves, 5 of 12 correlations involving heart rate, and 15 of 66 correlations with individuals' physical characteristics.

Although increased postural instability is usually attributed to dysfunction of large fibers (Nardone & Schieppati, 2004), small fibers may also be important. Nardone and Schieppati pointed out that people affected by Charcot-Marie-Tooth disease type IA have impairment of the large fibers (fibers IA), yet they do not show an increase in postural sway relative to HC. To pursue the contribution of small fibers, Nardone and Schieppati compared the COP areas of DN, for whom both large and small fibers were affected, and HC. Neuropathy was graded by the Neurological Disability Score (Dyck et al., 1980). Participants' feet

could be together or apart with eyes open or closed. They were systematically perturbed by 20–30 toe-up rotations (3° amplitude at 50°/s). The COP area after perturbation was significantly larger for DN than for HC. In addition, the mean position of the COP was significantly farther forward for DN than HC. COP area did not correlate with any clinical variables; that is, the neurological disability score or the composite neuropathy score based on the electrophysiological assessment of fibers IA. However, the correlation became significant when neural responsiveness was based on the assessments of type II fibers both for eyes open and closed. This finding was supported by a significant negative relation between the COP area and the conduction velocity of the Group II afferent fibers.

To buttress the foregoing discovery, Nardone, Grasso, and Schieppati (2006) added a group with Charcot-Marie-Tooth disease type II to the other groups: Charcot-Marie-Tooth disease type IA, DN, and HC. People with Charcot-Marie-Tooth disease type II are known to have neuropathy only of type II fibers. The criterion to diagnose neuropathy was based on England and Asbury's (2004) study. Both the neurology disability (cf. Dyck et al., 1980) and neuropathy scores for the lower limb (cf. Bergin et al., 1995) were evaluated. Participants stood with their feet 10 cm apart, with eyes open or closed in static and dynamic conditions for two trials each. Dynamic trials consisted of sinusoidal movement (0.2 Hz, 60 mm from peak to peak) in the AP axis. COP area was recorded by a force platform; reflective markers at the ankle and head picked up postural response.

In the static conditions, DN and Charcot-Marie-Tooth disease type II exhibited significantly greater COP area than HC and Charcot-Marie-Tooth disease type IA, although there was no significant relation between COP area and the neurology disability score or neuropathy score for the lower limb. However, COP area was negatively related to nerve conduction velocity of type II fibers, albeit only with eyes closed. In the dynamic conditions, the cross-correlation between the movements of the head and ankle was significantly higher with eyes open, but without differences as a function of participant group. However, the cross-correlation of the time lag between the head and ankle did show an interaction with participant group. Only for HC did head displacement precede ankle displacement. In the DN and Cl groups, head movement was delayed with eyes open, but it was not delayed with eyes closed. These findings support impairment of type II fibers as the leading amplifier of postural sway, and suggest that measuring degree of neuropathy should include a quantification of impairment in type II fibers given its potential as a marker of instability. Nardone et al. (2006) and Nardone and Schieppati (2004) indirectly suggested that methodological changes are necessary to quantify neuropathy in the future.

Status of Sensory Discrimination Versus Neural Responsiveness

In 19 of the 28 articles, participants performed quiet standing with no external perturbations (e.g., displacement of the support surface) and with no additional movement requirements (e.g., reaching with an arm). The other nine articles involved perturbations of upright standing or movement requirements supplementary to upright standing. For simplicity, the contrast between the two collections of studies can be expressed as *static* versus *dynamic* (see the introduction).

Whether differences in postural sway among DN, D, and HC are best predicted by participant-dependent sensory discrimination measures or participant-independent neural responsiveness measures seems to devolve on the static versus dynamic contrast. The static studies tend to support sensory discrimination measures, and the dynamic studies tend to support neural responsiveness measures, although it should be noted that neural responsiveness measures were less frequently used in the static studies.

In summary, the results of the reviewed studies thus far tend to be consistent with a peripheral sensory neuropathy hypothesis of greater DN instability at a general level. However, whether sensory discrimination or neural responsiveness is more successful at predicting the amplification of sway in DN seems moot. There is evidence for and against both the subjective and objective measures as primary predictors. A causal distinction along the lines of impaired subjective factors versus impaired objective factors determining the observed differences among DN, D, and HC looks unlikely.

Other Neuropathies

One difficulty for interpretation is that different individuals can be affected by different kinds of neuropathy. Peripheral neuropathy may be the first cause of instability in DN even though other neuropathies overtake it as the main culprit. In what follows, we evaluate evidence relevant to other neuropathies—central (in contrast with peripheral) and motor or autonomic (in contrast with sensory)—as sources of instability.

Central Neuropathy

The neuropathy affecting people with diabetes usually begins at the distal level before engaging the proximal level (for a review, see Cavanagh et al., 1993). However, this association between diabetes and symptoms at the peripheral level is not systematic. Moreover, central neuropathies are difficult to investigate (Uccioli et al., 1997), leading most researchers to exclude people with neurological issues detected by clinical or functional tests (for a review of exclusion criteria, see Simoneau et al., 1994). But does the exclusion of visible central impairment adequately avoid central neuropathy in DN? Evidence can be found in postmortem analyses of six DN: Every person had structural changes or degeneration of neurons or CNS areas (Greenbeaum et al., 1964). More generally, the central motor conduction velocity has been shown to be reduced in people with diabetes independently from the presence of peripheral neuropathy (e.g., Abbruzzese et al., 1993).

The relations among central neuropathy (i.e., in dorsal columns and corticospinal tracts), peripheral neuropathy, and COP motion were studied explicitly by Uccioli et al. (1997). Neuropathy was diagnosed by the San Antonio Consensus Conference guidelines (American Diabetes Association, 1988). The two experimental conditions were (a) participant's standing up on a force platform with eyes open and (b) eyes closed. To evaluate central neuropathy, somatosensory- and motor-evoked potentials were registered with methods from Rossini et al. (1994) and Rossini and Treviso (1983), respectively. The COP-dependent variables were path length, area, mean velocity and its standard deviation, and VFY (parameter derived from the velocity variance and mean position in the AP axis). DN exhibited significantly greater COP measures of sway than did D or HC. At the peripheral level, sural and peroneal nerve conduction velocity and amplitude were significantly reduced for DN. At the central level, somatosensory-evoked potential latencies were always significantly higher for DN than for the two other groups. Regression analyses showed that peripheral electrophysiological variables accounted for 73.6% of the total variance in postural sway; central electrophysiological variables did not play a significant role. Thus, although peripheral and central pathways can be affected in DN, only the peripheral neuropathy seems to affect COP motion. The independence of central and peripheral impairments has been supported elsewhere, albeit without postural sway measures (Abbruzzese et al., 1993).

In summary, although a central neuropathy hypothesis cannot be discounted solely on the basis of the foregoing; it has been rejected at least implicitly by the field, which has pursued other directions.

Autonomic Neuropathy

The American Diabetes Association (1988) recommended that at least one measure of autonomic function be included in the diagnosis of neuropathy. However, it is typical that studies of postural instability exclude people with visible impairment of autonomic function (e.g., symptomatic postural hypotension). Exceptions include studies by Di Nardo et al. (1999) and Yamamoto et al. (2001). The former found no impairment and the latter identified correlations (between heart rate and six COP measures) suggesting that decreased activity in DN parasympathetic and sympathetic systems could impair stability. At a minimum, the autonomic neuropathy hypothesis of instability cannot be ruled out.

Motor Neuropathy

Motor neuropathy has not been the primary focus of any investigation. The standard inclusion criteria are the ability to stand up without the aid of an assistive device and absence of marked symptoms of unstable posture. Because motor neuropathy is not reliably ruled out by these previous precautions, investigators often monitor muscle strength and motor nerve conduction (but not always; see Ahmmed & Mackenzie, 2003; Katoulis et al., 1997; Kim & Robinson, 2006; Lafond, Corriveau, & Prince, 2004; Lavery et al., 1998; Oppenheim et al., 1999; Simmons et al., 1997).

The degree of attention to motor neuropathy varies. Data may be (a) reported in unanalyzed tabular form (Boucher, Teasdale, Courtemanche, Bard, & Fleury, 1995; Cavanagh et al., 1993; Dickstein, Peterka, & Horak, 2003; Dickstein, Shupert, & Horak, 2001; Horak & Hlavacka, 2001; Horak, Dickstein, & Peterka, 2002), precluding conclusions about DN–HC differences, or (b) subjected to statistical analyses without comment. Those analyses revealed apparent differences in DN relative to HC: weaker in at least one of the analyzed muscles, or otherwise equivalent (Lord et al., 1993; Simoneau et al., 1994; Simoneau et al., 1995; Corriveau et al., 2000), and either slower in nerve conduction velocity (Di Nardo et al., 1999; Giacomini et al., 1996; Nardone et al., 2006; Simoneau et al., 1994; Simoneau et al., 1995; Uccioli et al., 1995; Uccioli et al., 1997) or smaller in motor activation (Giacomini et al.; Nardone et al.; Uccioli et al., 1995; Uccioli et al., 1997).

Despite evidence of significant correlations between neural responsiveness measures and measures of postural sway, researchers have not taken that evidence as suggestive of motor impairment as a cause of heightened postural instability (Di Nardo et al., 1999; Giacomini et al., 1996; Mimori et al., 1982; Uccioli et al., 1995; Uccioli et al., 1997; Yamamoto et al., 2001). Multiple regression analyses have revealed no dependence on motor impairment: Motor nerve conduction velocity and muscle strength have failed to predict postural sway (Bergin et al., 1995; Simoneau et al., 1994). DN with decreased lower limb muscle strength can be included among the more stable individuals (e.g., Nardone & Schieppati, 2004). People with Charcot-Marie-Tooth disease type IA have sustained motor fiber damage and lack strength in the tibialis anterior and triceps surea. Despite these impaired motor factors, their postural sway is comparable to that of HC and significantly less than that of DN (Nardone et al., 2000; Nardone & Schieppati).

Information Processing

The information-processing hypothesis of instability is not based on clinical or electrophysiological tests, but instead on specific conditions and analyses of postural sway. As noted in the previous section, neuropathy at the CNS level is not strongly related to amplified postural sway in DN. However, we have not yet addressed the functioning of the CNS in the control of stance, to which we now turn. Two types of information-processing hypotheses can be identified whereby amplified sway in DN arise from (a) an integration problem involving reweighting the importance of different kinds of information about posture or (b) underinformed selection of postural strategies and mechanisms.

Reweighting Hypothesis

It has been argued that deficiency in the peripheral sensory system should affect the integration of the information useful

for postural control; that is, visual, vestibular, and somatosensory information (e.g., Boucher et al., 1995; Simoneau et al., 1995). The suggestion is that the cause of DN instability is linked to the inability of the CNS to appropriately integrate available information for postural control.

One test compared three conditions—eyes open, eyes closed, and changing from eyes closed to eyes open in the same trial (Boucher et al., 1995)—for two groups of participants: DN and HC. Postural sway was recorded by a force platform, and the neuropathy was quantified using the scoring method developed by Valk et al. (1992). With other variables, the analyses were done on COP range in the AP and ML axes and on the mean COP velocity. On these measures, DN swayed significantly more than HC. Moreover, DN themselves swayed significantly more when their eyes had been opened halfway through the trial than when their eyes had been from the start. HC were not similarly affected. The transition cost of opening the eyes was ascribed to the necessary reweighting of sensory information that is beyond the human integrative capacity of DN; however, this reweighting does not imply a cognitive deficit. It is not clear why this should be more costly for DN because they have less information to integrate. The simple fact of not having enough peripheral sensory information seems a sufficient explanation.

The kind of information (e.g., COP, COM, ankle angle) that is lost or degraded by peripheral neuropathy is not well established (Horak et al., 2002). This issue provided the focus for a comparison of HC and DN, with neuropathy assessed by the San Antonio Consensus Conference guidelines (American Diabetes Association, 1988) and postural sway measured as the range of COP and COM motion in the AP axis (and their velocity variances). Eyes open and eyes closed provided static baseline conditions for three sway-referencing conditions. In the COP sway-referencing condition, the platform moved so as to stabilize the displacement of the COP estimated on a 0.5-Hz COP filtering basis. In the ankle angle sway-referencing condition, Horak et al. used the linear displacement of the hip to guide movement of the platform so as to stabilize the ankle angles. In the COM sway-referencing condition, the displacement of the COM was estimated by a double model with kinematic data at the shoulder and hip. Although DN swayed significantly more than HC in the static conditions, they did not differ in the sway-referencing conditions. DN may have little peripheral sensory information available because they swayed significantly less in the static condition than did HC in the sway-referencing condition (the difference was less important with the COP sway-referencing condition than with the two other sway-referencing conditions). These comparisons suggest that DN have less access to information about COP motions (understood as the controlling responses; Winter, Prince, Frank, Powell, & Zabjek, 1996) than about COM (understood as the controlled variable; Winter et al.) or ankle angle. A still unanswered question is whether their instability arises because they use deficient COP information or other less relevant but available information.

One possibility is that peripheral neuropathy encourages increased vestibulospinal sensitivity as a substitute (Horak & Hlavacka, 2001). Displacement of COP, COM, and trunk were recorded while the participant's head was turned toward the right shoulder. HC and DN, with neuropathy assessed by the San Antonio Consensus Conference Guidelines (American Diabetes Association, 1988), were recruited in different conditions. Galvanic vestibular stimulation, a small current passed between the mastoid processes, caused significantly larger sway for DN than HC, especially for the highest levels of stimulation. Both groups fluctuated more on 5 cm of foam than on a rigid platform, and DN could not even stand on the 10-cm foam. The galvanic stimulations had the same consequence in DN and HC in the rigid surface and the 5-cm foam: The angular displacement of the trunk was twice as large as the angular displacement of the COM. If both peripheral sensory loss and increased gain of vestibulospinal pathways are considered, then the data fit the model of Hlavacka, Mergner, and Krizkova (1996). It seems that the increased gain of vestibulospinal pathways can be directly available: HC participants with disruption of peripheral sensory information when standing on the 5-cm foam behaved the same way as did DN participants. It was suggested that the hip strategy was preferred by all participants under galvanic stimulation, thus confirming the crucial role of the vestibular system in the control of such a strategy. The implication is that DN participants are unstable because the importance accredited to the vestibular information does not increase enough to balance the lack of peripheral sensory information.

It has been shown that postural sway with eyes closed can be reduced substantially by light touch; that is, touching a surface in a way that does not provide support (e.g., Jeka & Lackner, 1994; Riley, Wong, Mitra, & Turvey, 1997). Whether the use of light touch differs for HC and DN is of relevance to the reweighting hypothesis. Dickstein et al. (2001) compared HC and DN participants in 12 conditions; their neuropathy was assessed by measures of neural responsiveness. The touch surface was slightly forward and to the side of participants. During no-touch trials, participants held their hand 10 cm above the surface; during light-touch trials, participants were limited to no more than 1 N of pressure with their right index finger; and during heavy-touch trials, participants could use the plate as a mechanical support using the index finger. DN applied significantly more force than HC on the mechanical support in the heavy-touch condition and in the lateral axis. The correlation between fingertip force and postural sway was significantly higher in DN than in HC in the ML axis. DN swayed significantly more than HC in the no-touch condition on foam, but the two groups did not differ in the light- or heavy-touch conditions. In the touching conditions, the RMS of COP in the AP and ML axes was reduced

significantly relative to RMS in the no-touch condition for all participants, but more so for DN. Not surprisingly, heavy touch reduced sway more than light touch. Touch reduced sway significantly more with eyes closed than with eyes open and when participant stood on foam versus the rigid surface. The pattern of RMS of trunk velocity in both AP and ML axes was similar for both light and heavy touch and was reduced with respect to the no-touch condition. DN and HC differed during the no-touch condition on foam: DN had a higher trunk velocity than HC (p = .06, .04 in the AP and ML axes, respectively), implicating more hip strategy than ankle strategy for DN (Dickstein et al.). The results implicate the potential usefulness of a handheld probe (e.g., long cane) as a means of augmenting DN's postural control through extended haptic perception (Burton, 1993; Carello, Fitzpatrick, & Turvey, 1992; Fitzpatrick, Carello, Schmidt, & Corey, 1994).

The contribution of surface touch in dynamic posturography conditions has also been examined to assess whether DN shorten their latencies or increase the scaling of their postural response to some surface translations (Dickstein et al., 2003). HC and DN, with neuropathy mostly diagnosed electrophysiologically, were compared in different conditions. Several results characterized both groups: (a) Gastrocnemius latency responses were unaffected by the velocity of the perturbation; (b) AP COP response latency was significantly shorter in the highest platform velocities; (c) the slope of the scaling of the initial AP COP velocity (because of a plantar flexion response) to platform velocity was highest during both touch conditions; and (d) the direction of the COP vector was toward the touch platform (toward the right side of participants) in the heavy-touch condition compared with the light-touch condition and, similarly, in the light-touch condition compared with the no-touch condition. However, HC had a significantly quicker postural reaction time than DN for both gastrocnemius (electromyogram-triggering signal) and AP COP latency (COP from the dual force platforms) in the different conditions. Moreover, the scaling of the initial AP COP velocity to platform velocity was significantly lower in DN than in HC. Further, this scaling was significantly improved (i.e., increased) with both levels of touch for HC, but only for heavy touch in DN.

Postural Strategies Hypothesis

Posture is controlled through a variety of strategies (Horak & Nashner, 1986; Nashner & McCollum, 1985). Although rarely evaluated directly in studies of diabetes, researchers have conjectured in different instances that DN switch from an ankle- to a hip-based strategy (Ahmmed & Mackenzie, 2003; Giacomini et al., 1996; Uccioli et al., 1995; Uccioli et al., 1997). Such a switch would be rationalized by the loss or reduction of peripheral somatosensory information at the feet (see Horak & Nashner; Horak, Nashner, Diener, 1990). The possibility of alteration in style of postural control is supported by an increase in the use of vestibular information for DN (Horak & Hlavacka, 2001).

A direct assessment of postural strategies (Giacomini et al., 1996) involved a comparison of DN, D, and HC, with the neuropathy selection criterion based on the clinical San Antonio Consensus Conference guidelines (American Diabetes Association, 1988; see Uccioli et al., 1995[4]). Focusing on COP in the AP axis, its mean velocity of sway, standard deviation, and VFY were significantly higher in DN than in D and HC. The VFY parameter is used to distinguish ankle and hip strategies (e.g., Gagey, Toupet, & Heuschen, 1992). Although Gagey et al. interpreted increase in VFY as use by DN of the hip strategy, the criterion for the interpretation was not made explicit, leaving some questions about its validity. (In addition, 22 of 42 correlations between measures of neural responsiveness—sural and peroneal latency, amplitude, and velocity—were significant but minimally interpreted by Giacomini et al.)

Of previously considered results, the larger measures of COP (RMS in AP and ML axes) and trunk velocity in DN than in HC, especially on a foam surface (Dickstein et al., 2001), may reflect an increased reliance on a hip strategy. And although head movement of DN and HC did not differ, the coordination between head and ankle (indexed by a cross-correlation) revealed that the time lag between the head and feet was significantly different between the two groups (Nardone et al., 2006). Others have found no significant difference in postural strategy between HC and DN probably because all the participants may have used a hip strategy (e.g., Horak & Hlavacka, 2001). In general, the literature provides insufficient evidence to conclude that DN differences from D and HC arise from differences in postural strategies.

Viability of the Information-Processing Hypothesis

From the information-processing view, DN could be unstable for two reasons: (a) a problem of reweighting available information and (b) the need to use a less stable postural strategy. These two possibilities are distinct from the peripheral sensory neuropathy hypothesis. For both possibilities, there is the persistent idea that the vestibular system gains importance for postural control in DN. However, the summarized research indicates that an intact vestibular system compensates incompletely for impairments of perceptual subsystems grounded in the mechanoreceptors of muscle, tendons, and skin. The efficacy of information about head acceleration for controlling posture stability does not match the information about the relations of the limbs to the body, to each other, and to the ground.

Biomechanical Consequences of Changes in Foot Morphology

Muscle weakness can lead to bone deformation, directly or indirectly resulting in callosities (e.g., Delbridge, Ellis, & Robertson, 1985) or ulcers (Cavanagh et al., 1993). A change in the morphology of the feet could be partly responsible for the postural sway differences between DN

and HC. Destabilization in DN could arise from a reduction in the geometrical base of support (e.g., Koozekanani, Stockwell, McGhee, & Firoozmand, 1980; van Wegen, van Emmerik, Wagenaar, & Ellis, 2001), a reduction in the functional base of support (King, Judge, & Wolfson, 1994), or both. The aforementioned possibilities may be considered as variants of a biomechanical hypothesis. Two studies found that DN had reduced mobility at the ankle relative to HC (Simmons et al., 1997; Simoneau et al., 1994). In both studies, the reduced mobility was recognized as an additional effect with peripheral sensory neuropathy as the main cause of instability.

Reduced Geometrical Base of Support

The geometrical base of support is roughly defined as the rectangular area with AP and ML boundaries derived from the dimensions of the feet in full contact with the floor (van Wegen et al., 2001; see Figure 4A). If one's intent is to maintain a strictly upright posture, then the vertical projection of the body's center of gravity (see Rougier, Burdet, Farenc, & Berger, 2001) should stay within the limits of the geometrical base of support. If it does not, then the individual would have to take a step or grab something to avoid loss of upright posture. For a multijointed body—with its many sources of variability—atop a characteristically small base of support, continuous sway is inevitable even for HC. Given that clawed or hammer toes reduce the contact area of foot and floor for DN (Walker, 2005), as depicted in Figure 4A, exaggerated sway in circumstances of intended upright posture may be expected. This possibility follows from experiments with HC participants showing that reducing the width of the support surface relative to the dimensions of the feet magnifies postural sway (e.g., Streepey, Kenyon, & Keshner, 2007; Wang & Lin, 2008).

Reduced Functional Base of Support

King et al. (1994) defined the functional base of support as the difference between mean COP locations during sustained forward and backward leaning divided by foot length. They found that the latter measure declined with age; when participants were aged 60 years or older, it declined at a rate of 16% per decade. The size of the range of COM motion controllable through COP depends on the sway velocity at any moment, the strength and functionality of postural muscles, ankle mobility, and base of support. Therefore, a reasonable surmise is that DN participants may have a contracted functional base of support compared with HC (see Figure 4B). Foot deformities (e.g., clawed or hammer toes), and reduced flexibility because of stiffened collagen (e.g., Brownlee, Cerami, & Vlassara, 1988) lessen the contribution of the toes to controlling the horizontal projection of the body's COM. Tendons, ligaments, and plantar soft tissue are all stiffer, limiting the mobility of the ankle joint and the joints in the feet (Cavanagh et al., 1993; Simmons et al., 1997; Simoneau et al., 1994). The consequences for stability would be comparable to those for stability of HC standing on a beam (cf. Horak & Nashner, 1986).

Location of COP in the Base of Support

In postural control, the average position of the COP (its AP and ML coordinates) in the base of support depicted in Figure 4C changes with body inclination to the vertical and influences the characteristics of COP motion (van Wegen, van Emmerik, & Riccio, 2002). Several studies have indicated that HC exhibit more COP motion when leaning forward than when standing in the neutral position (e.g., Duarte & Zatsiorsky, 2002; Riley, Mitra, Stoffregen, & Turvey, 1997; Rougier et al., 2001). A neutral position of the COP is when it is positioned at approximately 40–50% of the foot length (Okada & Fijiwara, 1983, 1984). It is telling that an additional potential consequence of the kind of foot deformation common to diabetic neuropathy is the displacement of the position of the COP away from its normal neutral (see Figure 4C). For example, anterior leg muscles (relevant to foot dorsiflexion and toe extension) are weaker than posterior muscles (England & Asbury, 2004). Plantar ulceration may also tip the COP closer to stability limits. (For an illustration of the mechanical etiology of plantar ulceration, see Stokes, Faris, & Hutton, 1975.)

Results are mixed as to whether these deformations are associated with increased postural sway characteristic of DN. A comparison of D, DN, and HC revealed that all groups swayed more with eyes closed than with eyes open, but the difference in sway was greater for DN, especially in the ML axis (Ahmmed & Mackenzie, 2003). No difference was found, however, among the groups in the location of the COP in either the AP or ML axes. Correlations between the location of the COP and other measures (sway in the two axes, age, sex, height, and weight) were not significant. Ahmmed and Mackenzie argued that the extent to which DN partici-

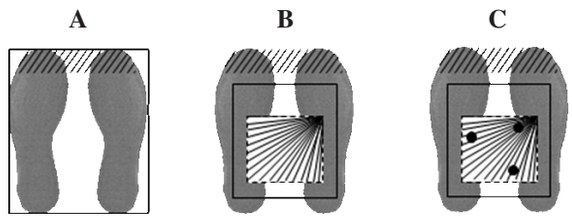

FIGURE 4. (**A**) Geometrical base of support defined by rectangle enclosing feet (E. E. H. van Wegen, R. E. A. van Emmerik, R. C. Wagenaar, & T. Ellis, 2001). The hatched segment represents the part of the geometrical base of support not available to people with clawed toes. (**B**) The exterior rectangle represents the geometrical base of support of healthy controls; the empty inside rectangle represents the functional base of support of healthy controls; the filled interior rectangle represents the hypothesized smaller functional base of support of people with diabetic neuropathy. (**C**) Black dots indicate hypothesized center of pressure positions in the anteroposterior–mediolateral plane displaced from 0, 0 because of deformations or weaknesses of the feet.

pants change their postural strategy (to hip instead of ankle) is not because of a more forward neutral position. In contrast, when participants' COP position was normalized (as a percentage of total foot length in the AP axis and as a percentage of body height in the ML axis), DN participants were found to lean forward significantly more than HC participants (Nardone & Schieppati, 2004). The weaker anterior muscles of DN participants could explain why they sway more than HC participants and, perhaps, why they favor the hip strategy. It is certain that the further the forward lean is, the greater the change in trunk angle from initial inclination relative to the change in COM angle from the ankle axis (see Figure 6 of Horak & Hlavacka, 2001). The trunk can continue to tilt by rotation around the hip when COM tilt has reached its limit (Horak & Hlavacka).

A direct evaluation of a role for foot ulceration in postural sway (Katoulis et al., 1997) used two tests to assess the presence of neuropathy: the clinical neuropathy disability score and VPT (see Dyck, 1988; Young, Boulton, MacLeod, Williams, & Sonksen, 1993). DN participants were divided into those with and without a history of foot ulcer, and they were compared with two control groups. The DN groups did not differ with respect to neurological disability score or VPT. DN participants were compared with HC and D participants in trials with eyes open and eyes closed, using a force platform to record postural fluctuation. The two control groups did not differ from one another. DN with a history of ulcers swayed significantly more than any other group in both visual conditions and in both axes. DN without a history of ulcers swayed significantly more than the two control groups in only one analysis. It is important to note that instability was not related to the severity of neuropathy. Although Katoulis et al. did not elaborate on the relevance of ulcer history, it seems likely that it is related to deformation of the feet with a consequent location of the COP closer to the limits of stability.

Is There a Positive Aspect to the Increased Sway of DN?

Our review to this point has taken the customary stance of interpreting the increased postural sway of DN as abnormal; that is, as indicating degraded posture or inappropriate postural strategies. From this customary stance, all sway is considered to be noise, and an increase in sway is considered as merely more noise. But noise in biological systems occurs in multiple forms and has varied constructive consequences (for reviews, see Riley & Turvey, 2002; Shinbrot & Muzzio, 2001). Could the amplified postural sway of DN be adaptive?

Preventing postural sway can have a deleterious effect on perceiving a surrounding layout of objects and surfaces as they relate to an individual's action capabilities (Mark, Balliet, Craver, Douglas, & Fox, 1990). For an otherwise stationary perceiver, postural sway is a means of detecting information relevant to the possibility of a particular action (in Mark et al., regardless of whether a horizontal surface was sitable). To the extent that a minimum of sway is required to facilitate perception, one may speculate that the minimum is higher for DN than for HC. What might be termed an *exploratory sway* or *useful noise hypothesis* is that DN participants sway more than HC participants—or change their strategy in an adaptive way—so as to pick up useful information for postural control. The hypothesis follows primarily from Riccio's (1993) conjecture that small postural sway generates information about the stability of the system's current state and about the stability or instability of other (macro) states (see also Riley, Mitra, et al., 1997). The consequence of the increase in sway by DN could be negative because of the possibility of falling, but that does not mitigate the need to sway so as to control posture.

This perspective may provide a context for understanding side-to-side or ML sway. Although postural sway is multidirectional, the ankle and hip strategies model postural sway only in the AP axis (cf. Horak & Nashner, 1986). Only a handful of researchers analyze data in the ML axis (Ahmmed & Mackenzie, 2003; Boucher et al., 1995; Cavanagh et al., 1993; Corriveau et al., 2000; Dickstein et al., 2001; Katoulis et al., 1997; Kim & Robinson, 2006; Lafond et al., 2004; Simoneau et al., 1995; Yamamoto et al., 2001). With one exception (Kim & Robinson), a significant DN–HC difference in some postural sway variable in the AP axis is paralleled by a significant difference in the ML axis. Moreover, some DN–HC differences in the ML axis are not apparent in the AP axis (Kim & Robinson; Lafond et al.).

It has been suggested that AP sway is controlled by an ankle mechanism, whereas ML sway is controlled by a hip mechanism (of loading and unloading the weight on one foot; Winter, Prince, Stergiou, & Powell, 1993). If two independent mechanisms are responsible for the control of postural sway in the AP and ML axes (cf. Balasubramaniam, Riley, & Turvey, 2000), then DN's ML instability is somewhat puzzling. To the extent that DN disabilities are at the peripheral level (i.e., lack of sensation, biomechanical problems), then postural control at the (proximal) hip level should be more available than postural control at the (peripheral) ankle level. Why, then, should DN and HC motion in the ML axis differ? Perhaps DN increase their reliance on a postural control mechanism at the hip level to pick up necessary information for their postural control in this ML axis. In this view, it is not so much instability as exploration in the ML axis. Two articles give relevant results supporting this view (Kim & Robinson, 2006; Lafond et al., 2004).

Kim and Robinson (2006) compared HC and DN (with type II diabetes but diagnostics unspecified). They had to detect a perturbation (movement of the platform by the SLIP–FALLS system), and they measured EMG (from the tibial anterior and gastrocnemius soleus muscle groups on both legs) and postural sway variables (COP and head displacements) to index an individual's postural state at the time of the perturbation. Not surprisingly, DN participants were less able to detect the perturbation than HC participants. However, using a neural network-based fuzzy logic inference model, Kim and Robinson determined that HC's detection

was better for AP sway, whereas DN's detection was better for ML sway. Unfortunately, these differences were not evaluated statistically. Nonetheless, they highlight the role of ML sway in facilitating perception by DN.

HC and DN (evaluated with Valk's scale; Valk et al., 1992; Valk et al., 1997) were compared to assess postural control mechanisms (Lafond et al., 2004). Postural sway was indexed by the RMS values of COPc and COPv (postural control at the ankle and hip, respectively; Winter et al., 1993), as well as the RMS values of COP_{left} and COP_{right} (COP coordinates under the left and right feet, respectively). Overall COP path length for DN was significantly higher than for HC in both eyes-open and eyes-closed conditions in the AP and ML axes. However, the relative contributions of COPc and COPv did not differ between the groups.

It is known that COPc and COPv are strongly and significantly correlated with COP path length in the AP and ML axes, respectively, but Lafond et al. (2004) did not find any significant difference between groups for these comparisons. In the AP axis, a single sway index (normalized cross-correlation between COPc and COP path length with eyes closed) was significantly reduced for DN relative to HC. In contrast, in the ML axis two sway indexes (normalized cross-correlation between COPc and COP path length and between the COPc and COPv, both with eyes open) were significantly reduced for DN relative to HC. This contrast was attributed to impairment at the ankle joint such that motor activities around the left and right evertor or invertor are not as good for DN as for HC (Lafond et al.). This is inconsistent, however, with arguments that the control of stance in the ML axis is based on a load or unload mechanism involving the hip adductors or abductors. Moreover, as previously noted, motor impairment need not increase instability. As an alternative, one may speculate that DN fluctuate more in the ML axis because they can control their sway in this axis and do so to pick up relevant information for postural control. Differentiating sway in the AP and ML axes warrants further investigation (Lafond et al.).

Investigations of the potential improvement in stance by provision of subsensory mechanical noise (e.g., Collins, Imhoff, & Grigg, 1997) lend support, albeit indirect, to the exploratory sway or useful noise hypothesis. The subsensory noise delivered through insoles results in larger sway reduction (by way of a process akin to stochastic resonance) for DN than for HC (Priplata et al., 2006). Numerically, in five of the eight measures, noise reduced DN postural sway (measured with a stabilogram) to the level of HC without noise. A possible interpretation is that DN participants typically sway more than HC because of a greater need to enhance information pickup for postural control. When that need is absent (when the neuropathy is compensated by subsensory noise), the sway is decreased.

Involvement of Effortful (Dynamic) Touch

As previously mentioned in the introduction, the perceptual subsystem tied to circumstances that affect predominantly the receptor states of a muscle, and its attachments (tendons and ligaments) to the skeleton is referred to variously as *effortful touch*, *dynamic touch*, *kinesthetic touch*, and, most classically, *muscle sense*.

Effortful touch is perhaps the most common form of touch, albeit the least apparent. It is functioning whenever one takes hold of (or grasps) something and moves it in some fashion; for example, when one lifts a cup, turns a door handle, carries a book, stacks a plate, hefts a ball, or shakes a stick. It is also functioning whenever one uses a tool or implement to act on or explore the environment. There are strong indications that the functional capability of effortful touch persists in the face of severely impaired discriminative touch.

One well-studied function of effortful touch is the nonvisual perception of the spatial extents of handheld objects by wielding. This ability of effortful or dynamic touch to exploit the mass moments of an object to perceive its length has been evaluated for a participant with surgically treated Arnold-Chiari Type 1 Malformation and cervical syrinx (Carello, Kinsella-Shaw, Amazeen, & Turvey, 2006). The participant lacked discriminative touch in the left arm (but no comparable sensory deficits in the right arm or the lower extremities). In the experiment, she wielded handheld rods of lengths 45, 60, and 80 cm with attached masses (to manipulate moment of inertia). Her nonvisual perception of the lengths by the insensate left arm ordered as the actual lengths (35, 48, and 60 cm) and compared favorably with her nonvisual perception by the unaffected right limb (48, 56, and 79 cm). In a related experiment with a DN participant, rods were held and wielded by the sensate right hand or attached to and wielded by the insensate right foot. The experiment was modeled after that of Hajnal et al. (2007), who demonstrated in healthy participants nonvisual length perceptions of rods wielded by foot that were equal to nonvisual length perceptions of the same rods wielded by hand. With the DN individual, rods of lengths 60, 80, and 100 cm were reported as lengths of 52, 62, and 78 cm, respectively, for wielding by foot and as 66, 76, and 87 cm, respectively, for wielding by hand (Carello, Silva, & Turvey, 2008).

It is clear that people lacking discriminative touch can nonetheless exhibit a very functional haptic perception of (a) attachments to the body by moving them and therefore (b) segments of the body by moving them. Research on effortful (dynamic) touch reveals that the inertial variables that are relevant to perceiving directional properties of attachments to the limbs are also relevant to perceiving directions of the limbs (e.g., Pagano & Turvey, 1995). It is important for the exploratory sway or useful noise hypothesis that successful effortful touch manifests under conditions of fairly minimal movement (Turvey & Carello, 1995), suggesting that, in magnifying postural sway, DN are facilitating perception of the body's orientation by effortful touch. Roughly, the hypothesis may be that the DN individual uses the nonneuropathic body segments to wield and perceive the neuropathic segments. The interpretation of the

case studied by Carello et al. (2006) was that the acceptable perceptual performance with the numb limb could have been because of the effects that the forces in wielding a rod had on more proximate and less sense-impaired tissues such as the muscles of the participant's upper arm and shoulder.

Recommendations for Future Studies

Controlling for Possible Confounds

Group differences in age, gender, weight, height, disease, sight, medication, and footwear can all affect group differences in postural sway and can magnify, reduce, or nullify the reported contrasts among DN, D, and HC. Control against possible confounds in full is challenged by their large number (e.g., see Simoneau et al., 1994). We elaborate on the possible confounds and consistency with which they are recognized as such in the reviewed literature.

Older adults (aged more than 65 years) sway more than younger adults (aged less than 25 years; Kinsella-Shaw, Harrison, Colon-Semenza, & Turvey, 2006). Bergin et al. (1995) did not control age. Recent evidence indicates that women sway less than men, corpulent people sway less than thin people, and tall people sway less than short people (Farenc, Rougier, & Berger, 2003). Of the 28 articles, only 6 matched DN, D, and HC on gender, height, and weight or BMI (Ahmmed & Mackenzie, 2003; Cavanagh et al., 1993; Corriveau et al., 2000; Lord et al., 1993; Simmons et al., 1997; Simoneau et al., 1994; Simoneau et al., 1995).

Postural sway is magnified by disease without peripheral sensory neuropathy. Oppenheim et al. (1999) showed that in the eyes-closed condition, separate groups of participants with Stage II Parkinson's, CNS damage, whiplash, or peripheral vestibular pathology exhibited COP motion equal to DN and in excess of D and HC. The implication is that there is a need to guard against the disease factor in pursuing an understanding of the relation of diabetes to postural control.

Postural sway is often (but not always) magnified when eyes are closed and when conditions of visual support (degree of optical structure, amount of illumination) are less than optimal (Edwards, 1946; Kinsella-Shaw et al., 2006). The relevance of the latter to understanding DN versus D is that neuropathy affects the nerves of the eyes, resulting in problems of visual acuity, loss of binocular vision, retinopathy, and presence of double vision (Simoneau et al., 1994). Of the 28 studies reviewed, 10 measured or controlled some or all of the problems related to sight (Cavanagh et al., 1993; Corriveau et al., 2000; Di Nardo et al., 1999; Giacomini et al., 1996; Katoulis et al., 1997; Simoneau et al., 1994; Simoneau et al., 1995; Uccioli et al., 1995; Uccioli et al., 1997; Yamamoto et al., 2001).

As noted, concerns have been raised about the possibility that magnification of postural sway could be consequent to certain medications. Only 12 of the reviewed papers explicitly excluded from the DN and D groups those individuals taking possible posture-destabilizing medication (Ahmmed & Mackenzie, 2003; Cavanagh et al., 1993; Di Nardo et al., 1999; Horak et al., 2002; Katoulis et al., 1997; Lord et al., 1993; Simmons et al., 1997; Simoneau et al., 1994; Simoneau et al., 1995; Uccioli et al., 1995).

In the reviewed studies, DN may have exhibited amplification of postural sway in part because of special footwear worn for comfort and protection (see Van Deursen & Simoneau, 1999, for a detailed description). In a study addressing a possible relation between footwear and sway, Lavery et al. (1998) examined DN with diabetic neuropathy determined by VPT. Participants performed quiet standing with each of five types of footwear: classical canvas shoes, removable cast walkers, half shoes, and total contact casts with a waking cast boot or cast heel (see Figure 5). Sway was greater (COP path length was the dependent variable) for shoes with total contact cast with a heel compared with canvas shoes and removable cast walkers (see Figure 5). In the reviewed studies, the variable of footwear is typically unstated. Did DN perform the experiment in their usual footwear? In some cases, researchers have reported that the participants were barefoot (Boucher et al., 1995; Dickstein et al., 2003; Dickstein et al., 2001; Simmons et al., 1997; Simoneau et al., 1994; Simoneau et al., 1995). In other cases, the reader is directed to methods as described elsewhere (Cavanagh et al., 1993; Uccioli et al., 1995). In the study of Centomo et al. (2007), participants stood on insoles; in Corriveau et al.'s (2000) study, participants were told expressly to wear flat-soled shoes. A reasonable surmise is that in the majority of the reviewed studies, DN, D, and HC completed the experiment in their daily footwear.

Broader Quantification of Neuropathy

Across the reviewed studies, the methods used to diagnose and quantify neuropathy have been diverse in kind and number, complicating comparisons of findings. Reliable diagnosis and quantification of neuropathy rests on multiple

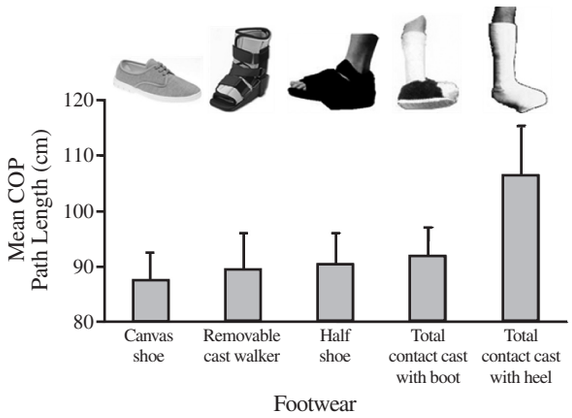

FIGURE 5. Center of pressure (COP) path length for people with diabetic neuropathy with different footwear. Adapted with permission from L. A. Lavery et al. (1998).

tests as underscored by the American Diabetes Association (1988). However, there are practical difficulties with implementing multiple tests and issues about how to achieve consistent interpretations among them (Perkins & Bril, 2003). Two minimal recommendations for future researchers are to use more than a single test (one sensory discrimination or neural responsiveness test marked the research of Bergin et al., 1995; Cavanagh et al., 1993; Centomo et al., 2007; Lavery et al., 1998; Priplata et al., 2006; Simmons et al., 1997; Simoneau et al., 1994; Simoneau et al., 1995; Yamamoto et al., 2001) and to screen HC participants with the same tests used for distinguishing DN and D, because elderly HC adults could have some kinds of neuropathy.

Use of Nonlinear Measures of Postural Sway

The dependent measures used in 27 of the reviewed articles were exclusively linear; for example, path length of COP, ellipse area of COP motion, RMS, and standard deviation of COP. (The one exception was Priplata et al., 2006.) Fast Fourier Transform figured in three studies (Cavanagh et al., 1993; Giacomini et al., 1996; Oppenheim et al., 1999). New insights into postural sway and its modes of control are emerging from studies that respect its nonlinear and nonstationary nature (see review in Riley & Turvey, 2002). Recurrence Quantification Analyses is a nonlinear tool that has provided helpful complements to conventional linear measures in the determination of group differences; for example, younger adults versus older adults (Kinsella-Shaw et al., 2006) and Parkinson's disease versus HC (Schmit et al., 2006). Recurrence Quantification Analyses may likewise prove helpful in sharpening the distinctions among DN, D, and HC in postural sway dynamics. A notable disadvantage of standard deviation and path length measures is their insensitivity to the temporal evolution of postural sway. In accordance, as the lone dependent measures of the contrast of DN with D and HC, the conventional linear measures may yield, at best, an incomplete picture and, at worst, an inaccurate picture of postural control differences (see Schmit et al.).

Manipulations Beyond Unperturbed and Perturbed Upright Stance

More demanding postural requirements beyond quiet standing may be needed to reveal the dynamic nature of postural control (Maki & McIllroy, 1996). Quiet standing and similar postures imposing the minimal demands may be insufficient for this purpose (Prioli, Cardozo, de Freitas Junior, & Barela, 2006). As previously observed, D postural sway exceeds HC postural sway when the task of standing upright includes a specified arm posture, one of far reaching (cf., Centomo et al., 2007), and DN postural sway reliably exceeds D, HC, and Cl only under conditions of eyes closed and head turned to the right or left (Oppenheim et al., 1999). In the 28 studies, participants rarely had to execute a specific task on which performance could be measured; that is, a suprapostural task (Riccio & Stoffregen, 1988). We recommend that such conditions be investigated because standing up is almost never a goal in itself, but it is normally a means to an end.

Improved Statistical Analysis

In the 28 articles, alpha adjustments in response to the problem of type I error rate were absent, as were reports of effect size. These omissions hinder evaluation of the practical significance of the results. Although many researchers have reported multiple correlations among measures of postural sway, sensory discrimination, neural responsiveness, and physical characteristic, the researchers' purposes in calculating the correlations have often been weak, and their outcomes have only rarely been interpreted. The single conclusion drawn from the correlations was that peripheral somatosensory impairment was related to instability. Future researchers should focus on specific predictions and avoid the aforementioned correlation analyses.

Last, many researchers have noticed that the pattern of sway for DN was not normally distributed. Therefore, it seems important to verify that the data do not violate the normality assumption for the use of parametric analyses. Researchers have either refrained from commenting on this assumption or proceeded to apply nonparametric analyses to some but not all of their data (Ahmmed & Mackenzie, 2003; Boucher et al., 1995; Di Nardo et al., 1999; Giacomini et al., 1996; Horak & Hlavacka, 2001; Horak et al., 2002; Lafond et al., 2004; Lavery et al., 1998; Mimori et al., 1982; Oppenheim et al., 1999; Uccioli et al., 1995; Uccioli et al., 1997; Yamamoto et al., 2001). It is possible that more significant differences among groups could have been found with nonparametric analyses.

Conclusion

In the present article, we focused on the postural behavior of people with diabetes mellitus. Our motivation for the review was the evidence that the disease may incur heightened postural sway.

Evidence that the magnification of postural sway (relative to HC and Cl) is associated with either diabetes per se, motor neuropathy, central neuropathy, or autonomic neuropathy is not strong. At the same time, however, the available evidence is not sufficient to rule these possibilities out of contention. More substantial is the evidence that amplification of the postural sway of people with diabetes is associated with peripheral sensory neuropathy (as shown by all 28 articles, directly or indirectly). Though preliminary, a hypothesis of peripheral sensory neuropathy seems to comprise three major assertions:

1. An impaired somatosensory system can be expected to magnify the postural sway of upright standing more than an impaired visual or vestibular system (Di Nardo et al., 1999; Simoneau et al., 1995). (Although the availability of intact visual and vestibular systems does not offset the neuropathy, it is offset by the availability of an additional

source of somatosensory stimulation that is provided haptically by nonforceful hand contact with a nearby surface [Dickstein et al., 2001; Dickstein et al., 2003]).
2. Magnification of postural sway by peripheral sensory impairment may be detected in the 0.5–1-Hz frequency band of COP motion (Oppenheim et al., 1999).
3. The neuropathy is more likely to be of the axonal kind than the demyelinating kind (Bergin et al., 1995) and involve type II fibers (Nardone et al., 2006; Nardone & Schieppati, 2004).

Further clarification of the relation between peripheral sensory neuropathy and postural sway of people with diabetes is obviously needed. There is also a need to better understand how sensory neuropathy in the lower limbs is realized as greater sway of the body. This understanding may be advanced through further developments of the central information-processing hypothesis in its two forms and by assessments of the biomechanical and exploratory sway or useful noise hypotheses that we introduced in this review. In our view, the most likely source of insights into the relation between lower limb neuropathy and postural sway is data collection on DN, D, HC, and Cl that incorporate explicit recognition of upright standing as (a) a means to an end (Riccio & Stoffregen, 1988), (b) emergent or self-organizing (Bardy, 2004), and (c) dependent on the capabilities of muscle-based perception (Carello & Turvey, 2004). To date, the preceding recognitions have played a limited role in developing researchers' understanding of diabetes and its complications for posture and movement. A broader role should pay both theoretical and practical dividends.

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NOTES

1. The modifier *mellitus*, Latin for *honey*, highlights the sweetness of urine and blood (Ahmed, 2002).

2. We did not review Simoneau et al.'s (1995) study because its data were included in Simoneau et al.'s (1994) article and in Simoneau's (1992) dissertation.

3. Yoking the visual surround to sway when the eyes are closed is not a meaningful condition of the Sensory Organization Test. Conditions involving those two combinations were omitted, resulting in six conditions.

4. It seems that Giacomini et al. (1996) used Uccioli et al.'s (1995) data but with different sway-dependent variables. Thus, for the results of the electrophysiological assessment, see Uccioli et al.

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Biographies of the Editors of the *Journal of Motor Behavior*

RICHARD G. CARSON spent his youth in Northern Ireland. He received his undergraduate degree in psychology at the University of Bristol. Thereafter he did graduate research at Simon Fraser University in Vancouver, Canada. He held a number of research fellowships at the University of Queensland in Brisbane, Australia, where he was appointed to a research professorship in 2002. At the beginning of 2006, he returned to Ireland to serve as professor at Queen's University Belfast. His research focuses on neural plasticity—that is, the capacity of the central nervous system to adapt in a manner that generates new functional capabilities or restores functional capabilities that have been lost, for example, because of brain injury or aging. He has served as an Executive Editor of the *Journal of Motor Behavior* since 2005.

ROBERT L. SAINBURG did his undergraduate work at New York University, where he received a baccalaureate in occupational therapy and subsequently practiced in clinical neurorehabilitation. He received his master's degree in physiology and neurobiology and his doctorate in neuroscience from Rutgers University. He then did postdoctoral research under the mentorship of Claude Ghez in the Department of Neurobiology at Columbia University in New York. He is now an associate professor of kinesiology and neurology at The Pennsylvania State University. In his research, Dr. Sainburg integrates biomechanical with neurobiological principles to elucidate the neural processes underlying the planning and execution of multijoint movements and bilateral coordination and to describe the mechanisms underlying coordination deficits. His research program is ultimately directed toward effecting improvements in clinical rehabilitation. He has served as Executive Editor of the *Journal of Motor Behavior* since 2007.

DAGMAR STERNAD is a full professor at the Department of Biology and Electrical and Computer Engineering at Northeastern University. Following her undergraduate work at the Technical University of Munich, Germany, she received her master's and doctoral degrees in experimental psychology working with Michael Turvey at the University of Connecticut. After graduating in 1995, she began working as assistant professor at The Pennsylvania State University where she established a productive research group. She has been Executive Editor of the *Journal of Motor Behavior* since 2005. Her interdisciplinary research program has several prongs: (a) the development of a unified framework for discrete and rhythmic movements with nonlinear dynamics as the theoretical framework, (b) a task-dynamic approach to a rhythmic movements with a special emphasis of the role of dynamical stability, (c) variability decomposition to understand learning and development, and (d) the detection of gait. The methods comprise an empirical component with behavioral experiments on human subjects, theoretical work that develops mathematical models for movement generation on the basis of coupled dynamical systems, and brain imaging studies (fMRI) to examine the cerebral activity. More recently, she has applied her experimental paradigms to neurological disorders such as Parkinson's disease and split-brain patients.

DANIEL M. CORCOS, Rapid Communications Editor of the *Journal of Motor Behavior*, obtained his doctorate in motor control from the University of Oregon in 1982 after obtaining his master's degree in psychology in 1980. Dr. Corcos was an assistant professor at the University of Illinois at Chicago from 1987–1993 and was promoted to the rank of associate professor with tenure in 1993 and to full professor in 1997. He served as Executive Editor of the *Journal of Motor Behavior* from 1997 to 2004. In addition to his work as the journal's Rapid Communications Editor, he is on the editorial board for the *Journal of Neuroengineering and Rehabilitation*. Dr. Corcos currently is involved in four lines of research. The first is related to the motor deficits associated with Parkinson's disease and how different neurosurgical interventions can facilitate the control of movement in Parkinson's disease. The second relates to the role of the basal ganglia in the control of a wide variety of movement tasks. The third relates to the central control of reflexes during voluntary movement. Last, he is interested in the role of exercise in reducing the negative impact of progressive neurological disorders.