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REVIEW ARTICLE

Proximal Postural Control Mechanisms May Be Exaggeratedly Adopted by Individuals With Peripheral Deficiencies: A Review

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ABSTRACT. In quiet stance, it is understood that healthy individuals control their posture primarily by a peripheral mechanism for anteroposterior sway and by a proximal mechanism for medialateral sway. The authors proposed the hypothesis that patients suffering from disease-related deficiencies, at their feet and legs, may exaggeratedly adopt proximal control mechanisms at their hip in the anteroposterior and medialateral axes. They critically reviewed the literature to test the proximal control hypothesis against published findings. The selected articles analyzed postural control mechanisms in individuals with diabetic peripheral neuropathy and in healthy controls. The data selected were kinematic and electromyographic. In the anteroposterior axis, 4 authors had previously tested the proposed hypothesis, but the findings are contrasted. In the medialateral axis, one study failed to validate the tested hypothesis. Overall, the published studies did not conform with the proximal control hypothesis. However, these studies did not specifically or deeply test such a hypothesis. The lack of results is critical because individuals suffering from peripheral disease-related deficiencies may be unstable, in part, because of a change in postural control mechanisms. For improvement of their stability and appropriate interventions, scientific explorations of the proximal control hypothesis should be investigated. Two proposals are made to move forward.

Keywords: ankle–hip, diabetic neuropathy, load–unload, peripheral issues, postural strategy

In stance, individuals sway all the time (Figure 1) with no possibility to be immobile (Hinsdale, 1887). Even if individuals try not to sway at all, they are continuously constrained by internal forces (e.g., the heart beating) and external forces (e.g., gravity). These forces prevent individuals from complete immobility (Figure 1, black arrows) because the body is a tall, multilink, living system that stands on a small base of support (the rectangle surrounding the feet on the ground). Consequently, postural control mechanisms need to work continuously to keep postural stability (Figure 1, white arrows). At the theoretical level, the scientific community is interested in discovering the functioning of these postural control mechanisms. In the present study, the focus is on the flexibility of known postural control mechanisms. The question we asked is whether disease-related issues at the peripheral level of the body may lead individuals to stabilize their posture more with proximal than with peripheral postural control mechanisms in the anteroposterior (AP) and medialateral (ML) axes. Throughout the article, *proximal* and *peripheral* refer to the hip and ankle levels, respectively. When proximal and peripheral mechanisms are discussed, hip and ankle dynamics are involved, but either predominantly at the hip or ankle levels, respectively.

In stance, the number of elements that need to be controlled to keep equilibrium is not as high as the number of elements in the system (approximately 10^2 joints, 10^3 muscles; Turvey, 1990). Indeed, movements are coordinated with one another and they can move together as units (Bernstein, 1967). In fact, only movements around the ankle and hip joints may be fundamental (i.e., absolutely necessary) for postural control in stance. Movements around other joints (e.g., flexion of the knees) can additionally stabilize the body, but these movements do not seem to be fundamental to keep postural stability. For example, the role of knee joints in postural control is compensatory or complementary (Gage, Winter, Frank, & Adkin, 2004). On this basis, researchers have searched for the existence of postural control mechanisms around the ankle and hip joints (e.g., Bardy, Marin, Stoffregen, & Bootsma, 1999). In the rest of this article, we only discuss the postural coordination around the ankle and hip joints although we are aware that more complex types of postural coordination have been modeled (e.g., Alexandrov, Frolov, & Massion, 2001).

In quiet stance, Nashner and McCollum (1985) proposed that AP body movements in healthy individuals resemble an inverted pendulum because the body rotates essentially around the ankle (Figure 2A). This was called the *ankle strategy* (Nashner & McCollum). In certain challenging conditions (e.g., on a beam; Horak & Nashner, 1986), movements of the body resemble a double inverted pendulum because the body rotates essentially around the ankles and the hips (Figure 2B). This was called the *hip strategy* (Nashner & McCollum). Although this model is more than 25 years old, it has received a wide acceptance in the postural control literature (Bardy, Oullier, Lagarde, & Stoffregen, 2007). With feet side-by-side and in quiet stance, it was found that individuals control their posture mostly with the ankle strategy. However, they adopted the hip strategy in some challenging conditions (e.g., Horak & Macpherson, 1996; Horak & Nashner, 1986). An updated version of the ankle and hip strategies recently revealed that hip and ankle motions can be found in quiet stance (Creath, Kiemel, Horak, Peterka, & Jeka, 2005). Other authors believed that the inverted pendulum model and, more generally, the ankle and hip strategies model were not appropriate to characterize the AP postural coordination (e.g., Bardy et al., 1999; Bardy, Oullier, Bootsma, & Stoffregen, 2002). In their study, Bardy et al. (2002) proposed that AP postural coordination should be investigated with kinematics data and they recorded angular displacements at the hip and ankle levels. Healthy young adults were instructed to sway back and forth at

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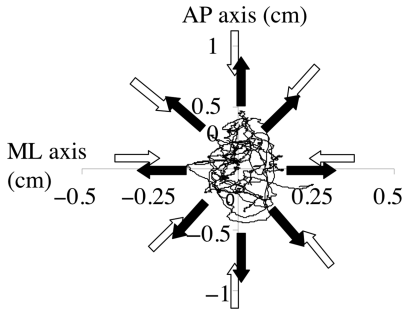

FIGURE 1. Example of a measure of the center of pressure sway (of the main author) recorded for 35 s on a force platform. The data are represented in centimeters in the anteroposterior (AP) and medialateral (ML) axes. The data are centered around the mean. For this figure, the center (0, 0) thus represents a complete stability. The black arrows going outward reveal that the body in posture is continuously pushed away from its stability (by many constraints). The white arrows going inward reveal that postural control mechanisms continuously works to keep postural stability. Control mechanisms necessarily work continuously because individuals rarely fall in stance.

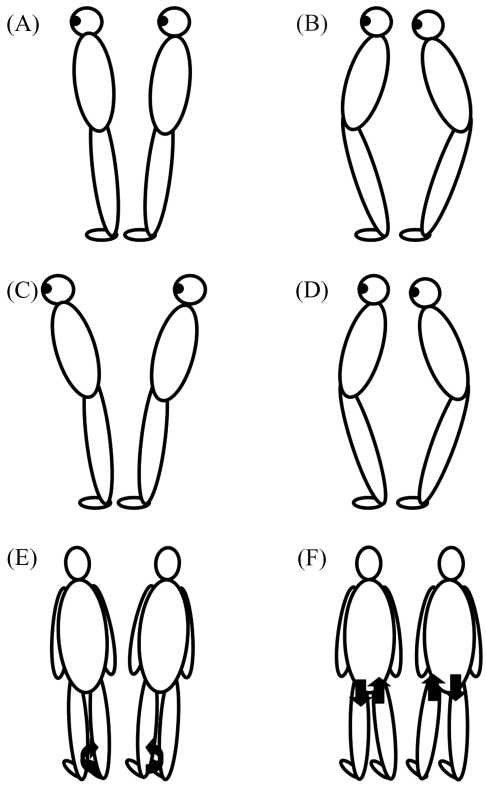

FIGURE 2. Postural control mechanisms adopted to reverse sway toward a point of complete stability. (**A**) The ankle strategy (Nashner & McCollum, 1985), with rotation of the body essentially around the ankle (as an inverted pendulum) in the anteroposterior axis. (**B**) The hip strategy (Horak & Nashner, 1986), with rotation of the body essentially around the hip in the anteroposterior axis (Nashner & McCollum). (**C**) The in-phase mode of coordination (Bardy et al., 2002), with in-phase rotation of the body around both ankle and hip. (**D**) The antiphase mode of coordination (Bardy et al.), with antiphase rotation of the body around both ankle and hip. (**E**) The inversion–eversion mechanism: with rotation of the body around the ankle in the medialateral axis (Winter et al., 1993). (**F**) The load–unload mechanism in the medialateral axis, which consists of loading the left leg and unloading the right leg or vice versa (Winter et al.).

constant peak-to-peak amplitude (10 cm) either at increasing (up-condition) or decreasing (down-condition) frequencies (by increments of 0.05 Hz between 0.05 Hz and 0.8 Hz). Participants had to keep a target square at the same size at their eyes while it was expanding and contracting on the screen. Bardy et al. (2002) only found two modes of coordination, the in-phase (Figure 2C) and antiphase (Figure 2D) modes of coordination with no intermediary—or other—mode of coordination. In the in-phase mode, the ankles and hips were moving in-phase. In the antiphase mode, both joints were moving in antiphase. Each mode of coordination was preserved under a large range of frequencies, thus showing that only two distinct modes of coordination existed. In another experiment, the in-phase and antiphase modes of coordination were found to emerge while participants simply stood in a moving room with a stationary floor (Oullier, Bardy, Stoffregen, & Bootsma, 2002).

Two similarities, between the ankle and hip strategies and the in-phase and antiphase modes of coordination, are relevant to this article (for a review of the differences, see Bardy et al., 1999; Bardy et al., 2002). The muscle activation was found to be peripheral to proximal in the ankle strategy and proximal to peripheral in the hip strategy (Nashner & McCollum, 1985). In parallel, Bardy et al. (2002) found that ankle movements were done before hip movements in the in-phase mode (hip-ankle relative phase = 29°) and hip movements were done slightly before ankle movements in the antiphase mode (hip-ankle relative phase = 171°). In the hip strategy and in the antiphase mode of coordination, body movements are greater at the hip than at the ankle (Bardy et al., 1999; Nashner & McCollum, 1985). In the ankle strategy and in-phase mode of coordination, ankle movements are either greater or equal to hip movements depending on whether individuals stand quietly or sway intentionally back and forth, respectively (Bardy et al., 1999; Nashner & McCollum). In the ankle strategy and in the in-phase mode of coordination, the control of posture was predominant at the peripheral level in terms of amplitude and priority of muscles activation. Inversely, the control of posture was predominant at the proximal level in the hip strategy and antiphase mode of coordination. For these reasons, we refer to the ankle strategy and the in-phase mode of coordination as the *AP peripheral control mechanism* in this article. The hip strategy and the antiphase mode of coordination are referred to as the *AP proximal control mechanism*.

Postural control in the ML axis was investigated in 1993 (Winter, Prince, Stergiou, & Powell, 1993). Before 1993, individuals were expected to control their ML sway with an inversion–eversion mechanism at the level of the ankle (Figure 2E). When the feet were side-by-side, invertor and evertor ankle muscles groups were supposed to be activated to keep postural stability. In 1993, Winter et al. (1993) revealed that this inversion–eversion hypothesis was not accurate for ML postural control. Instead, a load–unload postural mechanism was shown to counterbalance ML sway (Winter, Prince, Frank, Powell, & Zabjek, 1996; Winter et al., 1993). ML postural control was done at the level of the hip in loading and unloading the body weight on each foot (Figure 2F). The demonstration was based on kinetic and kinematic measures from two force platforms. In groups of young participants, Winter and colleagues (Winter et al., 1993; Winter et al., 1996) consistently showed that the inversion–eversion mechanism did not play any role in ML postural control (only results in conditions with feet positioned side-by-side are discussed here). Indeed, the inversion–eversion mechanism center of pressure (COPc) was significantly related to neither the load–unload mechanism (COPv) nor the integrated displacement of the COP under both feet (COPnet).[1] Moreover, the crosscorrelation between COPv and COPnet was very high and significant ($r = .99$). Also, the amplitude of COPv (in terms of root mean square) was four times greater than the amplitude of COPc in the ML axis with eyes open (Winter et al., 1993). Two latter results, with healthy old adults (Lafond, Corriveau, & Prince, 2004: Termoz et al., 2008), were slightly different but still consistent with Winter and colleagues (Winter et al., 1993; Winter et al., 1996). In Lafond et al., the relationships between COPc and COPv and between COPc and COPnet were high in old adults ($r =$ 0.64, 0.74, respectively). However COPc and COPv were expressed in percentage of COPnet. Therefore, the results found with healthy old adults (cf. Lafond et al.) may be consistent with the findings obtained in young adults (cf. Winter et al., 1993; Winter et al., 1996). It should be noted that Lafond et al. only searched for differences between healthy old adults and individuals with diabetic neuropathy (see a summary of this comparison latter on). In another study, Termoz et al. studied differences between young adults, healthy old adults, and old adults with Parkinson Disease. In Termoz et al., the three groups of participants showed a high crosscorrelation between COPv and COPnet ($r > .98$) and also a high crosscorrelation between COPc and COPnet ($r > .51$). Termoz et al. concluded that in a condition with feet side-by-side, load–unload and inversion–eversion mechanisms collaborate for ML postural control. Importantly, the load–unload mechanism was still the dominant mechanism. The inversion–eversion mechanism only helped the load–unload mechanism to control ML body sway (Termoz et al.). In the present article, the load–unload mechanism therefore was presented as the *ML proximal control mechanism* and the inversion–eversion mechanism was presented as the *ML peripheral control mechanism*.

To summarize the previous theoretical discussion, in healthy adults, the AP postural control mechanism is more so peripheral than proximal and the ML control mechanism is more so proximal than peripheral in simple quiet stance conditions. In more challenging conditions, the AP postural control mechanism may be predominantly proximal if the peripheral control mechanism is not sufficient anymore to recover postural stability (Horak & Nashner, 1986). In the ML axis, difficult conditions with feet side-by-side supposedly may not alter or change the predominance of the proximal control mechanism. There is no certitude yet because Winter and colleagues (e.g., Winter et al., 1993; Winter et al., 1996) did not vary the difficulty of conditions with the feet only side by side.

AP body sway was found to be more extended than ML body sway (Day, Steiger, Thompson, & Marsden, 1993; Hinsdale, 1887). As a result, the AP peripheral control mechanism may be solicited more often than the ML proximal control mechanism. The somatosensory system at the feet and legs has been shown to contribute significantly to postural stability (e.g., Maurer, Mergner, Bolha, & Hlavacka, 2001). Moreover, Simoneau, Ulbrecht, Derr, and Cavanagh (1995) showed that the increase in COP sway was greater in conditions with reduced somatosensory information (individuals with diabetic neuropathy vs. age-matched healthy controls) than in conditions with reduced visual information (eyes closed vs. open) or with perturbed vestibular information (head back 45° vs. head straight). Consequently, individuals with somatosensory issues at the peripheral level may be expected to be particularly destabilized in stance. In fact, in published articles these individuals with somatosensory issues at the peripheral level were not found to lose their equilibrium, even in conditions with additional visual and vestibular perturbations (e.g., Horak, Dickstein, & Peterka, 2002; Simoneau et al., 1995). An explanatory hypothesis may be that these individuals changed or adjusted their postural control mechanisms in the AP and ML axes. This is the main hypothesis of this manuscript. Horak, Nashner, and Diener (1990) already tested such a hypothesis in the AP axis. In their experiment, six healthy individuals (30–35 years old) were tested before and after receiving hypoxic anesthesia at their feet and ankles; the anesthesia simulated peripheral sensory neuropathy. Participants stood on two adjacent platforms that moved briefly (250 ms) in the AP axis either forward or backward in three conditions: (a) small amplitude: 1.2 cm, 6 cm/s with eyes open; (b) medium amplitude: 6 cm, 15 cm/s with eyes open and eyes closed; and (c) large amplitude: 12 cm, 35cm/s with eyes open (20 trials per condition). For our interest, participants significantly increased the amplitude of the horizontal shear forces at the surface, they significantly increased hip motions and hip muscle activation when they were anesthetized. According to Horak et al. (1990), anesthetized participants adopted the hip strategy in the second and third conditions. When these participants were not anesthetized, the ankle strategy was sufficient in all conditions. During the recovery phase (once participants activated their

muscles to recover stability after platform motion), participants exhibited four times greater changes in hip joint angle than in ankle angle when they were anesthetized. All these results were consistent with the present hypothesis that individuals can change their AP postural coordination in response to the reduction of available peripheral sensory information. In the ML axis, no authors have tested the hypothesis that individuals with somatosensory issues at the peripheral level may exaggeratedly adopt a proximal control mechanism. We think that it should be the case. Indeed, individuals with somatosensory issues—especially diabetic neuropathy—have been shown to sway significantly more than control individuals (C) in the ML axis (even more so than in the AP axis; Bonnet, Carello, & Turvey, 2009). Therefore, these individuals may exaggerate adopting the—still functioning—loading–unloading mechanism to save their ML equilibrium.

Overall, in the present study we proposed the proximal control hypothesis: that individuals with somatosensory issues at the peripheral level would exaggeratedly adopt proximal mechanisms in AP and ML axes to adapt their postural control to their disease-related deficiencies (Figures 3A, 3B, and 3C). The hypothesis was not related to the quantity of body sway (more or less sway did not matter) but rather to the quality of the postural control mechanism (the kind of mechanism preferentially adopted). The goal of the article was to critically review the literature to (a) reveal consistent and inconsistent published findings with the proximal control hypothesis, and (b) propose protocols to test such a hypothesis in the future.

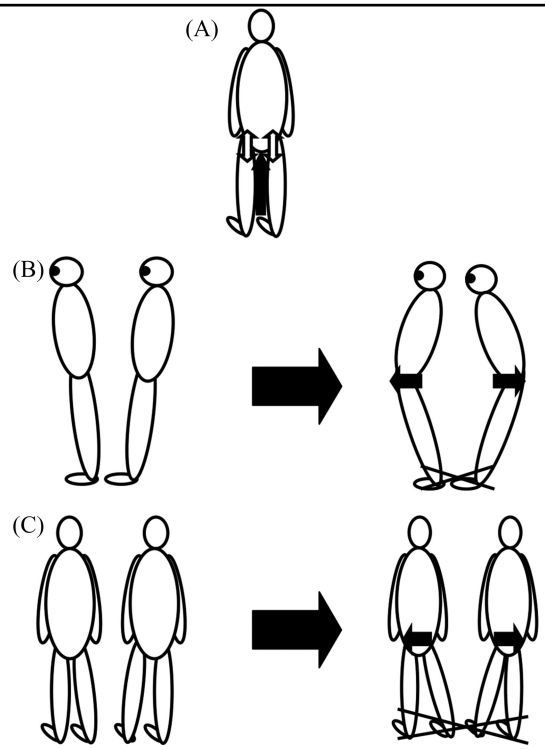

FIGURE 3. (**A**) Schematic representation of the proximal control hypothesis: switch from a peripheral to a proximal postural control mechanism in the anteroposterior axis (vertical black arrow) and exaggerated use of the load–unload mechanism in the medialateral axis (vertical white arrows). (**B**) In quiet stance situations, healthy individuals (on the left) adopt the peripheral control mechanism but individuals with disabilities at the peripheral level (on the right) may exaggeratedly adopt the proximal mechanism. (**C**) Healthy individuals (on the left) adopt the medialateral proximal control mechanism to control their medialateral sway (Winter et al., 1993), but individuals with disabilities at the peripheral level (on the right) may exaggeratedly adopt such a mechanism to adapt their postural control to their disease-related deficiencies.

Method

The population investigated, to test the proposed hypothesis, was the diabetic neuropathic population. This specific population was chosen because many diabetic individuals suffer from peripheral neuropathy (England & Asbury, 2004). Neuropathy was a relevant disease because it damages all kinds of nerves (i.e., sensory, motor, and autonomic at the peripheral level; Cavanagh, Simoneau, & Ulbrecht, 1993). Moreover, diabetes is the disease that leads to a greater proportion of peripheral neuropathy than any other disease in developed countries (England & Asbury). Individuals with diabetic neuropathy (DN) have many foot problems, such as muscle weaknesses, bone deformations (e.g., Delbridge, Ellis, & Robertson, 1985), and foot deformities (e.g., clawed or hammer toes). The stiffened tendons, ligaments, and plantar soft tissue (e.g., Brownlee, Cerami, & Vlassara, 1988) may additionally result in reduced flexibility of joints in the feet (Simmons, Richardson, & Pozos, 1997; Simoneau, Ulbrecht, Derr, Becker, & Cavanagh, 1994). Moreover, peripheral sensory and motor nerve activities are either absent or significantly reduced in individuals with DN (e.g., Cavanagh et al.). Peripheral nerve conduction velocity is usually reduced (e.g., Di Nardo et al., 1999; Simoneau et al.) and individuals with DN may have delayed muscle activation in front of perturbations (e.g., Inglis, Horak, Shupert, & Jones-Rycewicz, 1994). All these weaknesses and complications can lead to functional changes in postural control mechanisms. In other words, all these weaknesses and complications can lead proximal postural control mechanisms to be exaggeratedly adopted by individuals with DN.

To test the proximal control hypothesis the prerequisite criterion, in the article selection process, we included individuals with DN and C. In the first step, relevant articles with terms relating to postural control (postural control, postural sway, postural stability, stance, balance) and disease (diabetes, DN) were found using MEDLINE. Articles that did not include age-matched groups were not included in the selection. Additionally, individuals had to stand on two feet, side-by-side. Only original articles written in English and with quantitative data were reviewed. In other words, no review articles, no PhD dissertations, no abstracts, and no

articles analyzing qualitative measures were investigated. In the second step, the reference lists of these articles were investigated. In the third step, only articles and data revealing in which manner participants behaved were considered. In the AP axis, the articles had to reveal which strategy (hip or ankle) or mode of coordination (in-phase or antiphase) individuals adopted. In the ML axis, the articles had to analyze the load–unload and inversion–eversion mechanisms. Therefore, data and analyses about whether individuals exhibited more or less COP or postural sway were not discussed. The fact of swaying more or less does not describe the postural coordination. Moreover, electromyographic data were not discussed if they were not investigated at the ankle and hip levels. Indeed, having less muscle activation, exclusively at the ankle or hip, does not detail the postural coordination.

Results

After the first two steps of selection in MEDLINE, 28 articles were found: Ahmmed and Mackenzie (2003); Boucher, Teasdale, Courtemanche, Bard, and Fleury (1995); Cavanagh et al. (1993); Corriveau et al. (2000); Di Nardo et al. (1999); Dickstein, Peterka, and Horak (2003); Dickstein, Shupert, and Horak (2001); Giacomini et al. (1996); Hijmans, Geertzen, Zijlstra, Hof, and Postema (2008); Horak and Hlavacka (2001); Horak et al. (2002); Inglis et al. (1994), Katoulis et al. (1997); Lafond et al. (2004); Mimori et al. (1982); Nardone, Galante, Pareyson, and Schieppati (2007); Nardone, Grasso, and Schieppati (2006); Nardone and Schieppati (2004); Oppenheim, Kohen-Raz, Alex, Kohen-Raz, and Azarya (1999); Priplata et al. (2006); Simmons and Richardson (2001); Simmons et al. (1997); Simoneau et al. (1994); Simoneau et al. (1995); Turcot, Allet, Golay, Hoffmeyer, and Armand (2009); Uccioli et al. (1995); Uccioli et al. (1997); and Yamamoto et al. (2001). In the 28 articles, most of the analyses quantified COP or body sway (more or less sway), or quantified electromyographic data exclusively at the lower level of the body. Only five studies recorded dependent variables revealing the postural coordination of participants and discussed their findings in terms of changes in AP or ML postural mechanism (Di Nardo et al.; Giacomini et al.; Horak & Hlavacka; Inglis et al.; Lafond et al.).

In the AP axis, Giacomini et al. (1996) specifically investigated differences in postural control strategy between DN and C. Participants were young (DN: *M* age = 35 ± 1.9 years old; C: *M* age = 31 ± 0.9 years old) and seemed to be healthy (no visible instability, no neurological disorders, correct visual acuity).[2] The presence of the neuropathy was tested according to the clinical San Antonio Consensus Conference guidelines (see American Diabetes Association, 1988). The severity of the neuropathy (not reported) seemed to be high because electrophysiological assessments showed reduced or eliminated nerve conduction velocity, latency, and amplitude of activation in the sural and peroneal nerves. The vibration perception threshold at the malleolus and hallux were also significantly reduced in DN participants compared to C. Participants in each group (10 DN and 21 C) stood quietly in two conditions: with eyes open and with eyes closed. The dependent variable revealing the postural coordination of participants was the VFY, measured with a force platform (Giacomini et al.). The VFY is the standard deviation of the velocity of COP sway as a function of the AP COP position. It is known that the higher the VFY, the higher the stiffness around the ankles and consequently the more individuals adopt a hip strategy (Giacomini et al.). As expected, the VFY was greater in individuals with DN (0.91 ± 0.39) than in C (–0.44 ± 0.29) in the eyes open–eyes closed conditions (there was no indication whether the computation was done with eyes open or eyes closed). In conclusion, DN were said to adopt the hip strategy more than C in stance.

In Horak and Hlavacka (2001), there were 8 DN (*M* age = 57.9 years, *SD* = 11 years) and 8 C participants (age-matched; *M* age = 58.6 years; *SD* = 12 years). The neuropathy was assessed by the San Antonio Consensus Conference Guidelines (American Diabetes Association, 1988). The Semmes Weinstein monofilaments tested the severity of peripheral neuropathy and showed that 3 and 5 individuals with DN had mild and severe somatosensory impairments, respectively. The measures of sensory and motor nerve conduction velocity were almost normal in DN participants. Moreover, clinical analyses showed that participants all had good to excellent muscle strengths (ankles, knees, hips) and no vestibular impairment. All participants were investigated in conditions combining stance on three different surfaces (rigid vs. 5 cm and 10 cm of compliant foam) and galvanic stimulation (no stimulation, stimulation at 0.25, 0.5, 0.75, and 1 mA). Participants turned their head right or left and kept their eyes closed in these conditions. Horak and Hlavacka (2001) expected to reveal a tilt of the upper-body segment relative to the lower-body segment when participants received galvanic vestibular stimulation. In their four-way analysis of variance (ANOVA; Group × Trunk Angle × Center of Mass Angle × Surface), they did not reveal any significant effect including the factor group. Horak and Hlavacka explained that C and DN participants used the same hip strategy in their experiment.

Inglis et al. (1994) verified the hypothesis that peripheral sensory neuropathy resulted in delayed electromyographic muscle activation in response to backward platform displacements (four velocities: 10, 15, 25, 35 cm/s, amplitude of 6 cm; five amplitudes: 1.2, 3.6, 6, 9, 12 cm, velocity of 15 cm/s). To do so, Inglis et al. tested 9 DN (48–67 years old) and 8 C (age-matched). Peripheral sensory neuropathy was diagnosed based on sensory (sural) and on motor (peroneal) nerve conduction velocities. In their usual life, individuals with DN could move without any assistive device. Moreover, they were able to stand on toes and heels. No evidence of visual or vestibular issues or any significant orthopedic, psychological, or neurological complications were found. Only one of Inglis et al.'s results was relevant to test the proximal control hypothesis; this was the coordinated electromyographic

onset latencies of all recorded muscles (medial gastrocnemius, biceps femoris, and paraspinalis of the left leg). Whatever the experimental condition, the results showed that dorsal muscles were activated in a distal-to-proximal manner in individuals with DN and C. Both groups of participants thus adopted the same kind of AP peripheral control in these experimental conditions.

Simmons et al. (1997) tested 23 DN (M age = 61.4 ± 9.3 years) and 50 C (M age = 61.7 ± 9.3 years).[2] Participants did not have any visible injury and they were not diagnosed as being unstable. Participants had good vision—corrected if necessary—and none of them used any medication that could affect their postural stability. Some DN participants reported a chronic complication such as kidney or gastrointestinal diseases, but these complications are not known to increase postural sway. The neuropathy was diagnosed on the basis of the Semmes-Weinstein monofilament test (see Holewski, Stess, Graf, & Grunfeld, 1988). Individuals with DN exhibited significantly reduced sensation on at least 15 of the 20 tests (nine plantar and one dorsal sites on each foot). To specifically analyze the AP postural strategy, the authors calculated the hip-ankle strategy score derived from a dual force platform. This score was based on the shear force used by participants in relation to a theoretical maximum shear force of 25 pounds (Simmons et al.). A score near 100 indicated a predominant use of the ankle strategy whereas a score close to 0 indicated a predominant use of the hip strategy (Simmons et al.). Participants were tested in six conditions (cf. Figure 4). In Conditions 1 and 2, participants stood quietly with eyes open and eyes closed, respectively. In Condition 3, the visual surround was yoked to participants' sway but the platform was immobile. Conditions 4 and 5 were done with eyes open and with eyes closed and with the platform moving in sway-referenced manners. In Condition 6, the visual surroundings and the platform moved and participants kept their eyes open. The results showed that individuals with DN exhibited a lower strategy score than C in Conditions 5 and 6. Accordingly, individuals with DN were assumed to adopt a hip strategy more often than C. Indeed, the reduction of strategy scores between DN and C indicated a greater reliance on hip strategy (Simmons et al.).

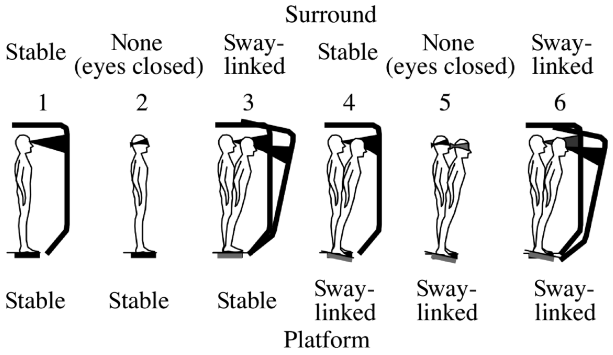

FIGURE 4. The six elements of the Sensory Organization Test. Adapted with permission from Figure 1 of Di Nardo et al. (1999).

Only Lafond et al. (2004) tested the ML postural control mechanism in 11 individuals with DN (M age = 69.1 ± 5.1 years) and in 20 C (M age = 72.3 ± 5.8 years). Valk, Nauto, Striners, and Bertelsman's (1992) procedure was used to quantify the severity of the neuropathy. Five individuals with DN were mildly affected by the neuropathy (score of 1–9), four were moderately affected (score of 10–18), and two were severely affected (score of 18–33). No participant had neurological, musculoskeletal, or visual impairments (at least 20/16 on the Snellen card). They all lived independently in their respective communities. Individuals with DN showed a significant reduction in mobility compared to C (the Tinetti score was significantly reduced). Lafond et al. calculated the root mean square values of COP_{left} and COP_{right} (COP coordinates under the left and right feet, respectively) and the root mean square values of COPc and COPv. In two conditions, eyes open and eyes closed, the relative contributions of COPc and COPv did not differ between C and DN participants. The results also did not show any significant difference between groups for the normalized crosscorrelation between COPv and COPnet. However, the normalized crosscorrelation between COPc and COPnet and between the COPc and COPv, both with eyes open, were significantly reduced in individuals with DN relative to C. Lafond et al. concluded that motor activities around the left and right evertor–invertor were not as effective in individuals with DN as in C. Therefore, Lafond et al.'s findings did not support the hypothesis that individuals with DN may exaggeratedly adopt a proximal control mechanism to save their ML equilibrium.

Discussion

In the present study, we postulated that individuals with issues at the peripheral level should exaggeratedly adopt proximal postural control mechanisms in the AP and ML axes. To confirm the validity of this hypothesis, we investigated the literature on the diabetic population. This systematic review revealed that the literature was ambiguous because results were contrasted in the five target studies. Results are still preliminary to conclude whether proximal postural control mechanisms are exaggeratedly adopted by individuals with DN and—in extension—by individuals with peripheral deficiencies. For this reason, we propose two methodologies to test the hypothesis in the future.

Review of the Literature

The literature clearly revealed that individuals with DN are unstable in stance (e.g., Corriveau et al., 2000). Today, there is no certitude about why this is so because many hypotheses—all insufficient—have been proposed to explain increased body sway in individuals with DN (Bonnet et al., 2009). A possibility could be that the lack of peripheral somatosensory information as well as other physiological

issues (foot deformities, muscle weaknesses, reduced flexibility of joints; see *Method* section) can lead individuals with DN to change their postural coordination. Only five articles partially tested such a hypothesis (Giacomini et al., 1996; Horak & Hlavacka, 2001; Inglis et al., 1994; Lafond et al., 2004; Simmons et al., 1997). Contrary to our prediction, Lafond et al. did not reveal any significant change in the ML load–unload mechanism in individuals with DN. In simple quiet stance conditions, individuals with DN did not exaggeratedly adopt the load–unload mechanism to reduce their ML body sway. In other words, the efficient load–unload mechanism of individuals with DN could not compensate for their disease-related deficiencies in the evertor–invertor mechanism. One element lacking in Lafond et al. was that DN participants may have showed reduced ankle inversion–eversion because of their reduced mobility score (Tinetti score). Additionally, their finding could not be generalized to any life conditions. In more difficult conditions, we may still expect that individuals with DN need to adopt the loading–unloading mechanism more than C. Indeed, individuals with DN sway significantly more than C in the ML axis and thus need to save their equilibrium more than C (Bonnet et al., 2009). Such a hypothesis needs to be tested in the future.

In the AP axis, two articles showed that individuals with DN preferred adopting the hip strategy over the ankle strategy in certain challenging conditions (see Giacomini et al., 1996; Simmons et al., 1997). These findings thus support the proximal control hypothesis, although their interpretation can still be questioned. Indeed, with a simple variable (VFY or ankle-hip strategy score), there is an uncertainty as to whether the two groups exhibited different postural mechanisms or exhibited the same ankle mechanism with different kinds of variability in this mechanism. Two other studies did not show any difference in postural strategy between C and DN participants (Horak & Hlavacka, 2001; Inglis et al., 1994). However, the conditions may have been too easy in Inglis et al.—both groups adopted the ankle strategy—or too hard in Horak and Hlavacka (2001)—both groups adopted the hip strategy. Future researchers need to definitively state whether individuals with DN exaggeratedly adopt an AP proximal control mechanism.

Necessity to Test the Proximal Control Hypothesis

More than our initial theoretically and practically grounded proximal control hypothesis (see introduction and *Method* section), other arguments are consistent with its claim. First, the importance of vestibular information for postural control has been shown to be increased in individuals with DN compared with C (Horak et al., 2002). Because the vestibular system controls for trunk orientation (Horak et al., 1990), the adoption of the AP proximal control mechanism may be facilitated in individuals with DN to counterbalance any disequilibrium in the AP axis (Horak et al., 1990). Second, in stance, a normal person cannot exceed 8° of forward sway and 4° of backward sway (Horak et al., 1990). In each article in the literature, individuals with DN were shown to sway significantly more, in a faster manner, or differently than C (Bonnet et al., 2009). Therefore, individuals with DN should critically approach the 8° and 4° limits more often than C, constraining them to change their AP control mechanism more often than C. Moreover, these limits may be shorter than 8–4° in individuals with DN because of the problems at their feet, as discussed in the *Method* section. These limits at least depend on biomechanical and physiological functionalities or constraints at the feet and legs. Third, individuals with DN may lean forward more than C when they stand, thus approaching their limit of forward sway more than C. Individuals with DN may do so to reduce the chance of falling backward in case of a perturbation. Falling forward is less problematic than falling backward in term of related injuries (Cummings & Nevitt, 1989). Individuals with peripheral neuropathy may also lean forward as a consequence of their need to look at the floor. Surfaces are indeed stable and reliable for the perception-action interactions with the environment (Gibson, 1950). Looking at the ground conveys information regarding its properties (rigidity, levelness, flatness, slipperiness, extendedness) and the presence of obstacles (potential causes for falls). Looking at the ground informs one about what the environment affords in terms of stability (Gibson & Mower, 1938).

For theoretical and practical reasons, the lack of relevant investigations of postural coordination in individuals with DN is critical. Indeed, postural coordination in individuals has to be known to give a sense of the effectiveness of postural control (van Emmerik & van Wegen, 2002). In order to better understand why individuals with DN are unstable (necessity for protection, prevention, adaptation of physical care programs), researchers not only need to know if these individuals are unstable but also in which way they are unstable. Future researchers should systematically investigate the analyses of differences in postural control mechanisms.

For practical reasons, if the proximal postural control hypothesis were accurate, individuals with DN may benefit from improving functionality of the hip control mechanism (e.g., muscle strength, sensitivity). In any way, such training should have positive effects for their everyday life. Indeed, exercise and sport activities are beneficial in healthy and diabetic individuals (Carnethon & Craft, 2008). Physical therapeutic programs (see, e.g., Horak, Henry, & Shumway-Cook, 1997; Rogers & Mille, 2003) could facilitate individuals with DN to be confronted to many kinds of perturbations and feedback situations. The training may conjointly increase force production, extension of joint mobility (e.g., axial-trunk rotational mobility; Rogers & Mille), and sensibility for postural control. Training may reduce the level of physical inactivity, which is known to be one factor causing hip fracture in individuals with DN (Janghorbani, Van Dam, Willett, & Hu, 2007). Also, practicing control of posture, specifically at the level of the hip, could lead to a reduction of forefoot peak plantar pressures, which is known to cause neuropathic foot ulcers (Mueller, Minor, Sahrmann, Schaaf, & Strube, 1994).

Potential Investigations to Test the Proximal Control Hypothesis

We recommend that future researchers use the dynamic approach of Bardy and colleagues (Bardy et al., 1999; Bardy et al., 2002) to test the proximal control hypothesis in the AP axis. The best experiment seems to be a replication of Bardy et al. (2002) as presented in the introduction. Indeed, in Bardy et al.'s (2002) methodology and approach, the concept of coordination makes sense as the number of coordinative patterns is reduced to two and only two. In reproducing Bardy et al. (2002) with DN individuals, we would expect that DN individuals would switch from the in-phase mode to the antiphase mode at lower numerical frequencies than C whatever the conditions (up- or down-condition). In the ML axis, the methodology used by Lafond et al. (2004) could be repeated with experimental conditions of contrasting difficulties. In this future experiment, stance width (distance between the feet) adopted by participants could be modulated as well as the difficulty of the task (e.g., easy or hard perceptual or cognitive tasks). To confirm the validity of the proximal control hypothesis, future studies need to find that individuals with DN adopt the load–unload mechanism significantly more than C in these challenging ML conditions.

Summary and Conclusion

The goal of this article was to suggest that individuals with DN should exaggeratedly adopt proximal control mechanisms to manage any kind of disequilibrium (in the AP and ML axes) in comparison to C. The published studies only partially tested such a hypothesis. If the hypothesis were valid, it may—at least in part—explain why individuals with DN sway more than C. For this reason, future researchers need to test the proximal control hypothesis with methodologies such as that of Bardy et al. (2002) and Lafond et al. (2004). These analyses would serve at least for control purposes and at best to understand differences in postural control between individuals with DN and C. Then the question could be asked: whether the proximal control hypothesis can be generalized to all patients with peripheral deficiencies.

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NOTES

1. The letter *c* refers to changes because the inversion–eversion mechanism is responsible for changes in the COP position. The letter *v* refers to vertical because the mechanism is vertically oriented with loading–unloading each foot.

2. The results of DN individuals are not discussed here, or in any other article, as explained in the *Method* section.

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