Adaptation of Postural Control in Normal and Pathologic Aging: Implications for Fall Prevention Programs

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Exercise interventions aimed at reducing falls in the elderly rest on several very important assumptions. One important pair of assumptions is that falling in the elderly is related to poor control of balance and that balance can be improved by practice and exercise. Fortunately, basic studies of postural control provide at least partial support for these assumptions. Extensive studies of postural control in both humans and animals suggest that the postural control system is not a fixed system of stereotyped reflexes, but rather is a highly complex and adaptable system, capable of accommodating changes in the biomechanical characteristics of the environment, changes in task requirements, and even the effects of disease (for reviews, see Horak, 1996; Horak & Macpherson, 1996). These assumptions are further supported by several exercise interventions that have been shown to reduce the incidence of falling (Horak, Jones-Rycewicz, Black, & Shumway-Cook, 1992a; Hu & Woollacott, 1994a, 1994b; Shepard & Telian, 1995; Shumway-Cook, Gruber, Baldwin, & Liao, 1997; Snow, 1999; Wolf & Gregor, 1999; Wolter & Studenski, 1996).

Most interventions for fall prevention, however, consist of a fixed set of exercises performed by all participants, and this points to another important assumption. Interventions relying on exercise or balance training programs assume that the elderly all fall for similar reasons. However, experimental studies have shown that postural control is not a simple function; rather, it consists of many different components (Dietz, 1992; Horak, 1997; Horak, Shupert, & Mirka, 1989; Horak & Macpherson, 1996; Massion, 1992). Every voluntary movement is associated with a specific postural adjustment that anticipates the destabilizing forces generated by the movement. In addition, rapid, automatic postural responses are evoked whenever there is a perturbation to the body that causes dysequilibrium or alters postural orientation. To produce these responses, the central nervous system needs to detect the direction and magnitude of the perturbation and select...
the response needed to restore equilibrium. The response must then be executed in time to prevent a fall. The sensorineural, neuromuscular, and musculoskeletal functions involved in the detection of balance perturbations, the selection of appropriate responses, and the execution of these responses make up the components of postural control.

Because the control of balance is a complex behavior consisting of multiple components, the reasons for falling in elderly individuals may differ depending on which components of the postural control system are not working optimally. For example, some individuals may have losses of sensory function that impair the ability to detect falling and result in delayed postural response times. Other individuals may have central nervous system disorders that limit their ability to adapt their postural responses to different environmental conditions. Still others may have relatively intact neural function but lack the muscular strength to recover from a loss of balance. These results suggest that a complete assessment of a balance disorder requires the comprehensive evaluation of many components of postural control, including biomechanical components, such as strength and range of motion; sensory components, such as vestibular, visual, and somatosensory function; and motor components, such as scaling response amplitudes to perturbation magnitudes. Intervention strategies to prevent falls must take into consideration the specific postural control deficits in each individual. The fact that the most successful interventions to date have included both balance training and strength building exercises provides evidence for this approach (Horak, Jones-Ryceveciz et al., 1992; Hu & Woollacott, 1994a, 1994b; Shepard & Telian, 1995; Shumway-Cook et al., 1997; Snow, 1999; Wolf & Gregor, 1999; Wolter & Studenski, 1996). Combined programs would necessarily address more different components of postural control.

In this paper, the results of basic studies of the effects of two different neural pathologies on automatic postural responses are presented, both to illustrate the different components of postural control, and to illustrate the adaptability of the postural control system. Studies of postural control in patients with loss of somatosensation in the feet due to diabetic peripheral neuropathy are presented to show the role of sensory information in postural control and examine the effects of sensory loss. Studies of postural control in patients with Parkinson's disease are presented to show one of the roles of the central nervous system in postural control and to demonstrate how the normal postural control system adapts to changes. Although the studies presented here were aimed at determining the effects of pathology on postural control, the studies have direct implications for both the assessment of balance disorders in the normal elderly population and for the development of specific fall prevention interventions.

**Pathology and the Decline of Postural Stability**

Many studies of postural control in the ostensibly normal elderly show declines in stability with age (Bates, Pruess, Souney, & Platt, 1995; Horak et al., 1989; Tinetti, Williams, & Mayewski, 1986; Vellas, Toupet, Rubenstein, Albone, & Christen, 1992; Woollacott, Moore, & Hu, 1993). In fact, this finding is so common that it is often assumed that age alone accounts for the increase in instability (e.g., see Belal & Giorig, 1986). Many studies have documented a wide variety of deficits in many of the critical systems for postural control, including changes in sensory thresholds, slowing in central nervous system functions, and biomechanical deficits, including muscle weakness (see Horak et al., 1989, for a review). Nevertheless, some elderly individuals enjoy excellent sensory and motor function and display, well into advanced age, balance control comparable to young healthy individuals. This suggests that the decline in stability due to
Figure 1 — Schematic representation of a model for the effect of multiple pathologies on postural stability as a function of age. The shaded area shows the threshold for falling. The effect of age alone is small and is represented by the solid line. Dashed lines show the hypothetical effect of pathologies for two individuals. Pathologies add to the aging affect to bring individuals to the threshold at an earlier age. Both are prone to falling, but they have different underlying causes for their falls. Adapted from Horak et al., 1989.

age-related degeneration of musculo-skeletal, neuromuscular, and sensory systems can be relatively small.

However, in addition to a small decrease in stability due to age alone, the elderly also have an increased probability of developing pathologies that could lead to an accelerated degeneration in these systems. It may be possible that general increases in instability in the elderly are not due to age itself but to the effects of pathologies, many of which may be subclinical. These concepts are illustrated schematically in Figure 1. Individuals developing more severe pathologies or greater numbers of pathologies in multiple systems could be expected to develop difficulty with balance at an earlier age than individuals who remain healthy. The idea that the accumulated effects of pathology may account for the increase in falling in the elderly is supported by a study by Gabell and Nayak (1984). In this study, the authors screened 1,187 individuals 65 years of age and older and were able to identify only 342 who had no history of falling and were completely free of any pathology that might affect balance control. The members of this elite group showed no difference from a group of young adults on a variety of gait measures. These results suggest that pathologies affecting balance are relatively common in the elderly and that decrements in performance may be more closely related to pathology than to age alone. Thus, a better understanding of the effect of different pathologies on different components underlying postural control will allow intervention programs for fall prevention to be focused specifically on the disordered components in each individual.

**Somatosensory Loss: Delayed Latencies and Poor Scaling**

Losses of sensitivity in peripheral sensory systems are common among the elderly, and among the most common findings are losses in somatosensation (Brocklehurst, Robertson, & James-Groom, 1982). *Somatosensation* is a general term used to describe a variety of sensory functions, including both proprioception mediated by muscle spindles, Golgi tendon organs, and joint receptors and cutaneous sensation mediated by receptors in the skin. Losses of vibration sensation in the foot and ankle are commonly reported
in the elderly population as well as losses of joint position sensation at the ankle (see Horak et al., 1989, for a review). Sensory information from muscle spindles may be responsible for detecting body sway and triggering medium latency (roughly 100 μsec) muscle responses in the legs that act to restore equilibrium in response to balance perturbations induced by movements of a support surface (Diener, Dichgans, Guschlbauer, & Mau, 1984; Dietz, 1992). Spindle information may also be involved in “scaling” postural responses—that is, in matching the magnitude of the postural response to the magnitude of the perturbation (Horak & Macpherson, 1996). Cutaneous information can provide important information about the characteristics of the surface on which subjects stand (e.g., compliant or firm, slippery or rough), and thereby contribute to the selection of appropriate postural responses.

To clarify the role of somatosensation in balance, we have conducted studies of automatic postural responses in subjects with profound somatosensory loss in the feet due to diabetic neuropathy (Inglis, Horak, Shupert, & Jones-Rycewicz, 1994). In these studies, we compared the postural responses to backward platform translations of varying velocities in a group of nine subjects with absent or abnormal sensory nerve conduction velocities and losses of vibration sense and proprioception in the feet, and a group of age-matched healthy controls (age range = 48–67 years). Although diabetic neuropathy often affects motor, as well as sensory, nerve conduction velocities, these studies were confined to subjects with adequate strength for postural control. (All subjects could stand independently on toes or heels.)

The results of the study showed that people with diabetic neuropathy have both delayed muscle onset times and difficulty scaling their postural responses to the magnitude of postural perturbations. Figure 2a shows representative muscle activation patterns for a diabetic subject and a control subject. In response to a backward platform translation, normal subjects activate dorsal muscles starting at the ankle (gastrocnemius) at about 100–120 μsec; ankle muscle activation is followed by activation at the thigh

Figure 2 — Delayed onset latencies and impaired scaling of postural muscles in response to backward surface translations in subjects with somatosensory loss due to diabetic peripheral neuropathy. (2a) Representative muscle responses in a normal subject and a subject with somatosensory loss. (2b) Group average postural muscles latencies show similar delays at all segments (M and SD). Asterisks indicate significant differences between control subjects and subjects with somatosensory loss. (2c) Reduced scaling of postural torque responses to increasing platform velocity in patients with somatosensory loss. Adapted from Inglis et al., 1994.
(hamstrings) and trunk (paraspinals). Muscle activations were delayed 25–35 \( \mu \text{sec} \) for subjects with diabetic neuropathy at all body segments (see Figure 2b). Figure 2c shows that normal subjects increase the rate of change of torque that they exert on the platform as the velocity of the platform perturbation increases. Subjects with somatosensory loss also increase their torque production, but their regression has a lower slope—that is, they over-respond to slow perturbations and under-respond to fast perturbations. Pilot studies from our laboratory also show increases in sway velocity and variance during quiet stance in patients with somatosensory loss, especially when subjects stand on a compliant surface.

These results imply that elderly individuals with somatosensory loss may have trouble detecting body sway, and may therefore have trouble initiating balance responses quickly. They may also have trouble detecting when surface characteristics change and may therefore be at greater risk for falling when changing from carpet to linoleum or when moving from a flat to an inclined surface. These problems will be enhanced when other sensory information, such as vision, is not available. Their postural responses may be delayed, and they may not scale postural responses appropriately. A thorough balance assessment should include an assessment of sensory thresholds. For somatosensory function, the tests should include tests of cutaneous sensation in the feet (vibration sensation, pressure sensitivity using Semmes-Weinstein hairs) and proprioception in the feet and ankles. Balance and walking tests should include situations in which vision is not available and on different support surfaces.

What are the most appropriate interventions for individuals with loss of somatosensation in the feet? Generalized strength training alone may not eliminate instability in cases of sensory loss. Figure 2c shows that the amount of force required to correct balance in the study described here is actually quite low. Balance rehabilitation may make subjects more aware of other sources of sensory information and may encourage subjects to try to make use of other sources but is unlikely to improve muscle onset latencies. Nevertheless, studies by Lackner et al. (in press) have shown that very light (<100 gm) fingertip contact with an external support can be effective as vision for decreasing sway in normal control subjects and patients with vestibular loss. At these levels of force, the external support is not providing significant biomechanical support; it appears that subjects substitute somatosensory information from the hand and arm for absent visual or vestibular function. Pilot studies in this laboratory suggest that light touch can also reduce sway in patients with somatosensory losses by providing a stable sensory reference and in particular by stabilizing the trunk. Therefore, if somatosensory function is normal in the hand, canes may be an effective sensory substitution tool. Thus, in the case of somatosensory loss, educating subjects about the role of the different senses in postural control and providing assistive aids like canes or walkers may be as important as strength and balance training.

**Parkinsonian Postural Dyscontrol**

The postural control system must adapt to changes in conditions. Adaptation in postural control is the process of modifying postural responses based on sensory context, biomechanical constraints, goals or intentions, prior experience, or practice (Horak, 1996). In the course of carrying out the activities of daily life, humans walk on firm unmoving support surfaces like sidewalks and unpredictably accelerating surfaces like the floors of busses. People often use handholds or sit in moving vehicles, and depending on which part of the body is supported, different joints must be moved to exert torque against the
Figure 3 — Typical changes in muscle activation patterns in response to backward surface translations while standing and sitting. (3a) A representative control subject shows elimination of ankle and thigh muscles for translations in sitting. (3b) A representative parkinsonian subject does not adapt to translations in sitting; leg muscles continue to be activated. Adapted from Horak et al., 1992.

available stable surfaces and thus, a different set of muscles must be activated. Studies of automatic postural responses in the laboratory have shown that for subjects holding onto a handrail, the earliest postural response to a sudden balance perturbation will occur in the arm rather than the leg (Cordo & Nashner, 1982). For subjects perturbed in sitting, trunk muscles are activated first (Hirschfeld & Forssberg, 1994; Horak, Nutt, & Nashner, 1992). Postural responses can even be suppressed when necessary; subjects instructed to take a step forward when they feel the platform on which they are standing
Figure 4 — Reductions in muscle activation amplitude when translated repeatedly in sitting for a group of patients with Parkinson’s disease and a group of age-matched controls. When translations in sitting are repeated, parkinsonian subjects eventually suppress leg muscle responses while sitting. Adapted from Chong and Horak, in press.

Figure 5 — Parkinsonian subjects fail to suppress leg muscle activation while holding onto a support during a postural perturbation. (5a) Representative muscle responses in a normal and parkinsonian subject. Odd-numbered trials are tibialis muscle responses to a toes-up rotation in free stance; even-numbered trials are responses while holding onto a support. (5b) Mean and standard error of the percent decrease in tibialis muscle activity for holding compared to free stance for age-matched controls and subjects with Parkinson’s disease. Adapted from Chong and Horak, in press.
start to move can completely suppress the normal automatic postural response to the surface movement (Burleigh, Horak, & Malouin, 1995).

Studies of automatic postural responses in people with Parkinson's disease have shown that damage to the basal ganglia can result in a loss of the postural control system's ability to adapt. Figure 3 shows leg muscle activations in response to backward translations in standing and sitting in normal control subjects and in patients with Parkinson's disease. When translated in standing, normal subjects activate ankle muscles first, followed by thigh and trunk muscles (Figure 3a). When translated in sitting, control subjects no longer activate muscles at the ankle and thigh; instead, the trunk is righted by early activation of paraspinal muscles. Subjects with Parkinson's disease show two types of abnormalities (Figure 3b). First, they tend to activate ventral as well as dorsal muscles when translated in standing, and second, they fail to suppress leg and thigh muscle responses when translated in sitting (Horak, Nutt, & Nashner, 1992). However, Figure 4 shows that if subjects with Parkinson's disease are exposed to repeated translations in sitting, the responses in leg muscles (soleus in this case) are eventually suppressed (Chong & Horak, in press). This implies that some learning can occur and may indicate a potential for rehabilitation.

Another example of the difficulty people with Parkinson's disease have in adapting postural responses to conditions of support is shown in Figure 5. These studies show that healthy subjects, but not subjects with Parkinson's disease, suppress postural responses in leg muscles to support surface rotations when holding onto a support (Chong & Horak, in press; Schieppati & Nardone, 1991). Figure 5a displays the activity of the tibialis anterior in response to a toes-up rotation of the feet, which displaces the body backward, for a representative subject with Parkinson's disease and an elderly control subject. On odd-numbered trials, subjects underwent a toes-up rotation in free stance; on even-numbered trials, the subjects underwent the same toes-up rotation while holding onto a support. The control subject suppresses the leg muscle response when holding on, but the suppression is much less complete for the subject with Parkinson's disease. Figure 5b shows a histogram giving the activation level of tibialis in the holding condition as a percentage of the activation level in the free stance condition for a group of subjects with Parkinson's disease and a group of elderly control subjects. The subjects with Parkinson's disease show significantly less suppression; their activation of tibialis when holding a support is on the average 60% of the activation level in free stance, whereas the activation level for elderly controls averages 25%.

The healthy elderly show declines in basal ganglia function even without the diagnosis of Parkinson's disease and have often been reported to show signs of extrapyramidal dysfunction, such as flexed postures, slowed movements, and tremors (Martin, 1967, 1977). The level of dopamine in the substantia nigra decreases with age; the diagnosis of frank Parkinson's disease is not usually possible until at least 20% of substantia nigra cells are still intact (see Nutt & Horak, 1996). Thus, even the normal elderly may have some difficulty adapting postural responses to changes in conditions. Therefore, assessments of balance function in the elderly should include an assessment of the ability to adapt postural control to different conditions.

Assessment tools can include (a) changing support conditions by observing postural control during free stance and when using a handheld or other support versus stance with support; (b) changing instructions, by having subjects either try to resist a gentle postural perturbation or to allow their bodies to move; and (c) determining whether subjects can modify their responses with practice. The fact the ability to adapt may be present but may require a longer learning period in the elderly also suggests that
training interventions may be of limited usefulness; subjects do not have the luxury of practicing postural responses to balance perturbations outside the clinic. The limited ability of subjects with Parkinson’s disease to adapt to a change in support conditions further suggests that even canes and walkers may be of limited use in this population. In these cases, education about fall prevention and environmental modification may be critical to preventing injurious falls.

Underlying Postural Control Deficits

Although the majority of elderly individuals do not have Parkinson’s disease or profound loss of somatosensation in the feet, some will be at high risk for a fall because of deteriorating basal ganglia function, and others, because of reduced sensory function. The implications of these studies for exercise interventions and fall prevention in the elderly are clear. First, the studies make clear that the sources of postural dyscontrol can be quite heterogeneous even if their results are not. Regardless of whether response times are delayed or postural responses are badly matched in amplitude to a balance disturbance or the postural control system has become inflexible, falls can result.

This concept is illustrated schematically in Figure 6, which shows a typical curve describing the increase in falling in the elderly and illustrates some of the many causes that can, singly or in combination, result in an increased tendency to fall. These results also imply that generalized balance assessments may not be successful in predicting which elderly individuals may fall, even when these assessments are fairly comprehensive (see Martin & Grabiner, 1999). They also suggest that generalized interventions may be of limited usefulness. For example, strength training may not prevent falls.
related to improper scaling of postural responses due to a sensory or central nervous system disorder. Finally, they suggest that a successful falls intervention program will need to include an education component, an exploration of the benefits of assistive aids, and modification of home environments, as well as a program of balance training and strengthening exercises.

References


