Special Series

Postural Perturbations: New Insights for Treatment of Balance Disorders

This article reviews the neural control of posture as understood through studies of automatic responses to mechanical perturbations. Recent studies of responses to postural perturbations have provided a new view of how postural stability is controlled, and this view has profound implications for physical therapy practice. We discuss the implications for rehabilitation of balance disorders and demonstrate how an understanding of the specific systems underlying postural control can help to focus and enrich our therapeutic approaches. By understanding the basic systems underlying control of balance, such as strategy selection, rapid latencies, coordinated temporal spatial patterns, force control, and context-specific adaptations, therapists can focus their treatment on each patient's specific impairments. Research on postural responses to surface translations has shown that balance is not based on a fixed set of equilibrium reflexes but on a flexible, functional motor skill that can adapt with training and experience. More research is needed to determine the extent to which quantification of automatic postural responses has practical implications for predicting falls in patients with constraints in their postural control system. [Horak FB, Henry SM, Shumway-Cook A. Postural perturbations: new insights for treatment of balance disorders. *Phys Ther.* 1997;77:517–533.]

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Postural control, although poorly understood, is critical for the efficient and effective performance of all goal-directed activities. A useful experimental approach to understanding neural control of posture is the disrupting of stable equilibrium and the recording of behavioral reactions to these perturbations. Magnus,[1] Rademaker,[2] and Sherrington[3] described responses to disequilibrium in infants and animals almost 100 years ago. Over the last 20 years (see reviews by Horak and Macpherson,[4] Dietz,[5] and Massion[6]), however, the application of new technology to quantify the surface forces, muscle activations, movement patterns, and joint torques that characterize postural responses involving the entire body has provided many new insights into posture and movement control. Recent studies of responses to postural perturbations have provided insight into multijoint coordination and multisensory interaction in motor control in general, and not just specific to the task of postural stability. Studies of postural responses have helped lead the way from a "reflex/hierarchical" concept of motor control to a "systems" approach, which emphasizes a goal-directed neural organization of multiple, interacting systems.[7,8]

Recent studies of responses to postural perturbations have provided new views of how postural stability is controlled. Previously, balance was viewed as resulting from a distinct set of reflex-like equilibrium responses elicited by stimuli to a particular sensory system and neural balance center.[1,2] More recently, balance has been viewed as a skill that the nervous system learns to accomplish using many systems, including passive biomechanical elements, all available sensory systems and muscles, and many different parts of the brain. We believe that balance can no longer be viewed as a totally reactive response to sensory stimuli. Instead, results from platform studies have shown that equilibrium control is quite proactive, adaptive, and centrally organized based on prior experience and intention.[9]

Changes in our basic understanding of how the central nervous system (CNS) controls postural stability have implications for physical therapy practice. Therapists are no longer limited to retraining balance by facilitating a fixed set of equilibrium reflexes. By viewing balance as a fundamental motor skill that the nervous system learns, therapists can recognize the potential for applying concepts from motor learning such as practice, feedback, experience, and education to retraining balance.

The practical importance of studying responses to external perturbations is becoming more and more apparent to physical therapists. The majority of falls among elderly persons are thought to be due to inadequate responses to perturbations. About 50% of these falls are thought to be due to sudden motion of the base of support such as slips and trips, 35% are believed to be due to external displacement of the body's center of mass (COM), and only 10% can be attributed to spontaneous falls related to physiological episodes such as dizziness, seizures, or transient ischemic attacks.[10] Maki and colleagues[11] also found that quantification of postural responses to lateral surface translations was one of the best predictors of future falls among elderly persons. Quick, coordinated responses to environmental perturbations are a very important part of effective stability during stance and gait. Therefore, we believe that laboratory quantification of postural responses may be used to predict balance in functional activities and is useful in clinical assessments of balance.

This article focuses on insights into the neural control of posture achieved through studies of automatic responses to mechanical (primarily surface) perturbations. We will summarize what perturbation studies have taught us about specific control systems that are responsible for strategy selection, rapid latency, coordinated temporal-spatial patterns, force control, and context-specific adaptation of postural responses. We will then discuss the implications for rehabilitation of balance disorders and demonstrate how a better understanding of the specific systems underlying postural control can assist in developing therapeutic approaches.

Definition of Postural Control

A postural perturbation is a sudden change in conditions that displaces the body posture away from equilibrium. These perturbations could consist of sensory perturbations, such as vestibular perturbations that result from electrical stimulation,[12] visual perturbations caused by a moving room or moving visual images,[13] or somatosensory perturbations caused by vibration of muscle.[14] The postural reactions to these sensory perturbations may be in response to perceptions of instability rather than to actual disequilibrium.[4] In contrast, mechanical perturbations actually displace the position of body segments, which may lead to displacement of the total-body COM, or disequilibrium. Small displacements of a single body segment, such as the head, can result in very

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small muscle responses throughout the body,[15] but larger displacements of the total-body COM require large enough responses to exert directionally specific forces at contact surfaces to return the body's COM to equilibrium.[16] Mechanical postural perturbations can be applied to any body part, such as a push to the trunk,[17,18] head,[15] or limbs.[19] The most common experimental approach is to perturb the support surface, which displaces the base of support under the body's COM. These support-surface perturbations are similar to a slip, trip, surface irregularity, or acceleration or deceleration of a moving surface such as a bus in which an individual is balancing.

Postural equilibrium is the condition in which all the forces acting on the body are balanced such that the COM is controlled relative to the base of support, either in a particular position or during movements. Control of balance, or equilibrium, can be reactive, that is, in response to external forces displacing the COM, or proactive, as occurs in anticipation of internally generated, destabilizing forces imposed by the body's own movements. Both external forces, including gravity and forces related to interaction with the environment, and internal forces, which are generated during all body movements, even respiration, ultimately act to destabilize the body by accelerating its COM. The role of the nervous system is to detect and predict instability and produce the appropriate muscle forces that will complement and coordinate with all the other forces acting on the body so that the COM is well controlled and balance is maintained.

No response to an external postural perturbation is totally reactive. Platform perturbation studies indicate that although automatic postural responses to external displacements of the body's COM are shaped by the sensory characteristics of the perturbation, responses also are shaped by CNS mechanisms related to expectations, attention, experience, environmental context, and intention, as well as by preprogrammed muscle activation patterns called *synergies*.[9] Thus, carefully constructed studies of automatic responses to external perturbations may reveal the relative contribution of many different central neural mechanisms in coordinating the multisegmental task of maintaining postural equilibrium.

The clinical implications of recent postural perturbation research suggest that observing how a patient responds to external perturbations provides therapists with a window into (1) how effectively the patient's sensory and motor systems respond to a particular pattern of sensory stimuli, (2) how well the patient's nervous system is prepared for and adapts to perturbations under a variety of contexts, and (3) how well the patient learns and executes a preplanned, coordinated motor pattern. Evaluation of responses to perturbations may also predict the likelihood for falls in natural environments.[20,21] We believe, however, that therapists need to recognize that responses to perturbations reveal only one aspect of the postural control system. In order to fully characterize a patient's postural control, therapists should also consider mechanisms related to control of (1) stability and antigravity muscle tone in steady-state positions such as stance and sitting, (2) sensory interpretation for spatial orientation and body alignment, and (3) equilibrium control in anticipation of and during movement, locomotion, or changes in posture. This article focuses on postural responses to external perturbations and will not address these other aspects of postural control.

Postural Responses: What Have We Learned?

Synergies and Strategies

Studies of how we control equilibrium of the multisegmented body against gravity and environmental disturbances have led to two important motor control concepts: muscle synergy patterns and movement strategies.[22–24] Nashner[22,25] first described normal muscle synergies as stereotyped patterns of bursts of muscle activity in response to surface translations and rotations. Initially, these synergies were thought to be the result of hardwired, inflexible central pattern generators of the nervous system. Synergies were thought to be long loop extensions of the stretch reflex. Postural synergies were labeled "functional stretch reflexes" because they adapted to include activation of nonstretched muscles if required for the function of postural stability.[25] Over the last 20 years, however, the notion of muscle synergies has evolved toward a concept of "flexible" synergies, defined as centrally organized patterns of muscle activity that are responsive to initial conditions, perturbation characteristics, learning, and intention.[4,23,26] Understanding synergistic organization of multiple muscles for a common goal is useful to explain normal motor coordination, as well as disordered coordination such as occurs with brain injury.

The concept of postural strategies emerged as investigators struggled with a way to describe general sensorimotor solutions to the control of posture, including not only muscle synergies but also movement patterns, joint torques, and contact forces. Nashner and McCollum[27] predicted and Horak and Nashner[24] described two distinct postural response "strategies," an ankle strategy and a hip strategy, that people could use to maintain equilibrium in response to anterior or posterior surface translations. Horak[5,24,28,29] also predicted a "stepping strategy," which has since been well characterized.

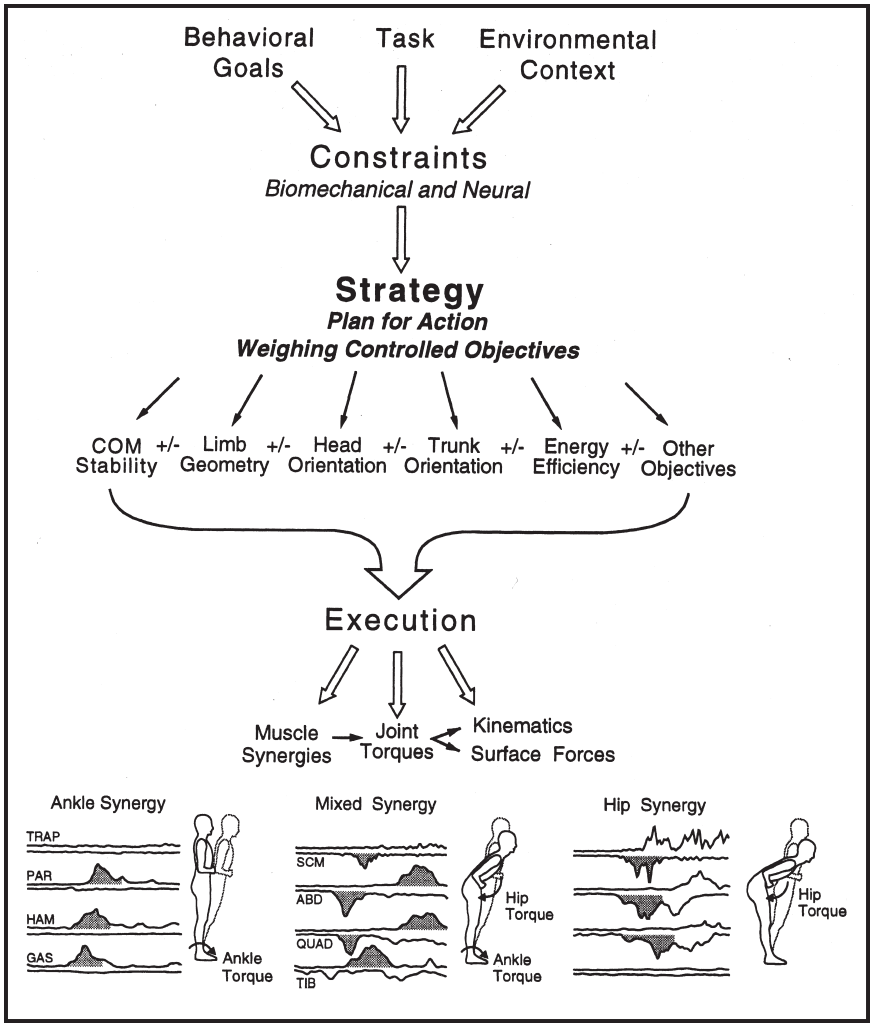

Figure 1.
A conceptual framework for the emergence of strategies that are plans for action. COM=center of mass, TRAP=trapezius muscle, SCM=sternocleidomastoid muscle, PAR=lumbar paraspinal muscles, ABD=rectus abdominis muscle, HAM=hamstring muscles, QUAD=rectus femoris muscle, GAS=gastrocnemius muscles, TIB=tibialis anterior muscle.

These strategies can be characterized by their different muscle synergies, kinematics, and joint torques.[30] The ankle strategy uses distal-to-proximal muscle activation, the hip strategy uses early proximal hip and trunk muscle activation, and the stepping strategy uses early activation of hip abductors and ankle co-contraction.[24,29] These strategies can be differentiated by the large angular trunk acceleration of the hip strategy and by the flexible inverted-pendulum style of the ankle strategy, which includes motions at the knee and hip as well as at the ankle.[31] The ankle strategy moves the body's COM with torques primarily at the ankle and the knee.[31] The hip strategy adds hip torque to the ankle and knee torque.[31] The stepping strategy is characterized by asymmetrical loading and unloading of the legs to move the base of support under the falling COM.[24,28,32]

Horak and Nashner[24] discovered that these strategies were not hardwired like reflexes, but could gradually be learned with experience in new environmental contexts. At first, these strategies were conceived as consisting of distinct, centrally programmed patterns that were combined to provide a continuum of "mixed" strategies.[24] More recently, the continuum of postural strategies from ankle to hip is considered to be similar to the continuum of locomotor strategies in which there are transitions from walking to galloping and trotting.[22] The stepping, or stumbling, strategy could, however, represent a truly independent strategy that is usually preferred to the hip strategy when there are no surface or instructional constraints. Thus, the strategy concept has been gradually changing to allow for the functional flexibility, specificity, and motor learning apparent in postural behavior.[4]

Horak[4,7–9] now hypothesizes that strategies are emergent neural control processes providing an overall "plan for action" based on the behavioral goals, environmental context, and particular task or activity (Fig. 1). The plan for action involves prioritizing many potential controlled variables of postural control such as control of the body's COM,[6,30,33] limb geometry,[34] stabilization of the head and visual fixation,[35] alignment of the trunk in space,[12,34] energy efficiency,[36] and contact forces.[37] Thus, what the nervous system is controlling in any particular postural situation, and how this is done, can vary depending on the individual's goals, the environmental context, and the task that the person is conducting. For example, when attempting to read a hand-held book while walking, stabilization of the head and gaze to the hand with the written word may be the highest priority, whereas when attempting to balance a full glass of water while walking, stabilization of the hand and glass with respect to gravity emerges as the highest priority in order to accomplish the task.[38]

Strategies that emerge in any situation are limited by both external constraints (ie, those imposed by the environment and the particular task) and internal constraints (ie, those imposed by an individual's biomechanical system and nervous system) (Fig. 1). Inter-

nal, biomechanical constraints, such as the number of limbs available, joint range of motion, and strength of muscles involved in the task, as well as internal, neural constraints, such as the extent to which attention is focused on the task, accuracy of sensory information, and force- and position-control mechanisms in the nervous system, will ultimately shape the emergent strategy.

Horak and Macpherson[4] and Horak and Shumway-Cook[32] hypothesize that postural strategies may be best differentiated by what the CNS is attempting to control. Computer models have shown that the hip strategy is optimal for quickly moving the COM, whereas the ankle strategy is optimal for maintaining a trunk vertical orientation while moving the COM.[32,36] However, these goals are not discrete and mutually exclusive. People use a whole continuum of strategies to control multiple neural objectives in the face of a variety of biomechanical and neural constraints, depending on the circumstances.

Researchers who investigate postural strategies in response to perturbations in different directions,[16,39] at different perturbation sites,[18] in different initial postural alignments,[40,41] and under different sensory conditions are searching for invariant neural objectives or "controlled variables." For example, postural strategies for recovery of stability following lateral surface perturbations may be similar to the so-called "ankle" strategy for anteroposterior perturbations. The goal of both types of strategies is to move the COM without compromising an erect trunk via loading and unloading of vertical surface force under the feet. The muscle synergies, kinematics, and joint torques used to implement the sagittal and lateral postural strategies, however, must be different to accommodate the different biomechanical constraints of sagittal-plane movement versus frontal-plane movement.[16,39,42] For example, correction for lateral perturbations involves early activation of the hip abductors rather than the ankle muscles.[39]

Thus, although a strategy may be identified by measuring kinematic, electromyographic (EMG), or force variables, these variables describe how the strategy is implemented and not, necessarily, the strategy itself (Fig. 1). A strategy is best described by what the CNS is attempting to control. Although it is often very difficult to determine what the nervous system is attempting to control, examining invariance in the way postural strategies are implemented can help researchers determine what the CNS is controlling. The implementation of many types of normal and abnormal postural strategies in response to disequilibrium have been well characterized in the literature.[15,22,24,29,43–50] This information can be very useful for improving therapists' understanding of the basis for instability and for designing effective interventions for improving balance that are specific to the underlying cause.[24,43,47,51,52]

Studies of postural strategies used by patients with sensory loss have shown that strategy selection depends not only on biomechanical constraints, but also on the sensory information available to the nervous system. Patients with complete vestibular loss are unable to utilize a hip strategy in recovery of their balance even when standing across a narrow beam where a hip strategy is required for efficient control of equilibrium.[43] In contrast, impaired somatosensory information from the lower limbs from peripheral neuropathy or ischemic leg cuffs results in an inability to use an ankle strategy effectively and reliance on a hip or stepping strategy.[43,51] This research suggests that utilization of the ankle strategy requires adequate surface somatosensory information and that utilization of the hip strategy requires adequate vestibular information. Thus, postural strategies that emerge in any situation are further constrained by the availability of sensory information inherent in the environment and perceived by the individual.

Postural Response Latencies

The earliest responses to surface perturbations are called "automatic postural responses" because postural latencies (70–180 milliseconds) are much longer than stretch reflex latencies (40–50 milliseconds), but shorter than voluntary reaction times (180–250 milliseconds).[19] Part of the delay in activating postural muscles comes from conduction delays. Muscles closer to the spinal cord, such as those of the neck or arm, are often, but not always, activated prior to muscles farther away, such as those crossing the ankles.[15,18] Results of platform perturbation studies suggest that centrally programmed synergies can delay activation of muscles more proximal to the spinal cord in order to obtain a particular functional spatiotemporal pattern. For example, in the ankle strategy, distal muscles at the ankle are activated well in advance of the trunk muscles, suggesting a central delay in activating the trunk muscles.[53]

Delays in the onset of a postural response can arise from (1) slowed sensory or motor conduction such as occurs with peripheral neuropathies,[51] (2) slowed spinal conduction such as can occur with multiple sclerosis,[54] or (3) delay in central processing such as occurs with Down syndrome,[44] cerebral palsy,[44] or aging.[45,55] Figure 2 illustrates a 45-millisecond delay in onset of ankle, knee, and hip muscle activity in response to a backward surface translation in a patient with diabetic peripheral neuropathy. Some persons compensate for delayed postural response latencies by increasing the magnitude of their responses and using more anticipatory control.[54] Apparent delays in postural responses as evaluated by body motion may not be related to an actual delay in onset of

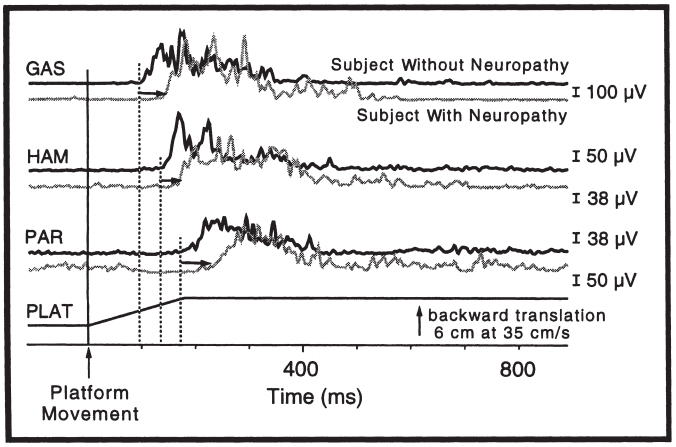

Figure 2.
Display of postural responses with impaired somatosensation for a representative subject with peripheral neuropothy (thin trace) are delayed compared with those for a subject without neuropathy (thick trace). Typical averaged electromyographic recordings from each subject activated in response to a backward platform translation of 6 cm at 35 cm/s. The three dotted vertical lines mark the burst onsets for each of the muscle groups for the subject without neuropathy. The muscle burst onsets for the subject with neuropathy are delayed. GAS=medial gastrocnemius muscle, HAM=biceps femoris muscle, PAR=paraspinal muscles, PLAT=platform. (Adapted from Inglis et al.[51])

EMG activity but to a slow rate of force production, a slow increase of muscle activity, or an abnormal spatiotemporal coordination of synergies.[47]

Spatiotemporal Coordination

Postural muscle synergies are organized in space and time to produce effective forces against support surfaces to move the body's COM and to control or prevent excessive motion at joints due to indirect, interactive torques. For example, the ankle synergy is implemented with early activation of ankle muscles to produce torques at the surface, followed by activation of knee and then hip muscles approximately 30 to 50 milliseconds later to control excessive knee and hip buckling due to the effect of ankle torques on other joints.[27,56]

Postural responses are not always initiated in the stretched muscles. For example, the first functional response to a toes-up tilt of the surface is in the shortened tibialis anterior muscle.[25] The earliest postural muscle activation may be at the neck[15] or arms,[57] instead of at the ankles, depending on the speed of perturbation and the particular context (eg, gaze instructions or presence of handrails).[57,58]

A muscle is not activated for only one direction of perturbation, but is activated over a small range of perturbation directions, with a maximum activation often in response to a diagonal direction of perturbation.[39] Figure 3A shows that activation of the left tensor fasciae latae muscle is maximal for a lateral perturbation to the right. It is also active for a range of anterolateral and posterolateral directions. When the magnitude of EMG responses to postural perturbations in 12 different directions is plotted as a polar plot, maximum muscle activation for most muscles tends to be in one of two main diagonals (Fig. 3B).[39] These results suggest that the nervous system constrains muscles to work together as synergies that are flexibly tuned to the specific biomechanical conditions, such as perturbation direction or initial stance width.

Patients with neurological impairments may show abnormal spatiotemporal coordination of automatic postural responses. Patients with stroke, head injury, or spastic cerebral palsy, as well as some elderly individuals, can show reversals in the normal distal-to-proximal temporal sequencing of postural muscle activation, resulting in excessive buckling or hyperextension of the knees and hips.[46,56,59] Abnormal spatiotemporal coordination of postural muscle responses has also been seen in patients with Parkinson's disease.[47,60] Some patients with Parkinson's disease show excessive coactivation of muscles in response to surface translations (Fig. 4).[47,60] Although patients with Parkinson's disease show normal latencies in the gastrocnemius, hamstring, and paraspinal muscles in response to forward sway, they often additionally add bursts in antagonist tibialis anterior, quadriceps femoris, and rectus abdominis muscles, which would increase stiffness but would be ineffective for direction-specific forces against the surface to preserve equilibrium. Abnormal spatiotemporal coordination of postural synergies could result from problems in creation of the synergies themselves, from interaction among simultaneous synergies, or from abnormal sensorimotor or biomechanical constraints.

Force Control

Platform perturbation studies have indicated that individuals without neurological impairments proportionally scale the magnitude of their automatic postural responses to the magnitude of their disequilibrium.[61,62] This scaling is based on both direct sensory characteristics, such as the initial speed of perturbation, and anticipatory mechanisms based on prediction of displacement characteristics, such as the estimated displacement amplitude. Figure 5A shows normal scaling of the magnitude of gastrocnemius muscle activation and the resulting surface reactive torque in response to increasing amplitudes of backward surface translations. Because automatic postural responses are initiated at 100 milliseconds, the nervous system does not have sensory information available about the amplitude of displacements of longer duration and therefore must rely on predictive mechanisms based on prior experience.[62] The role of prediction can be illustrated by comparing the scaling of postural response to predict-

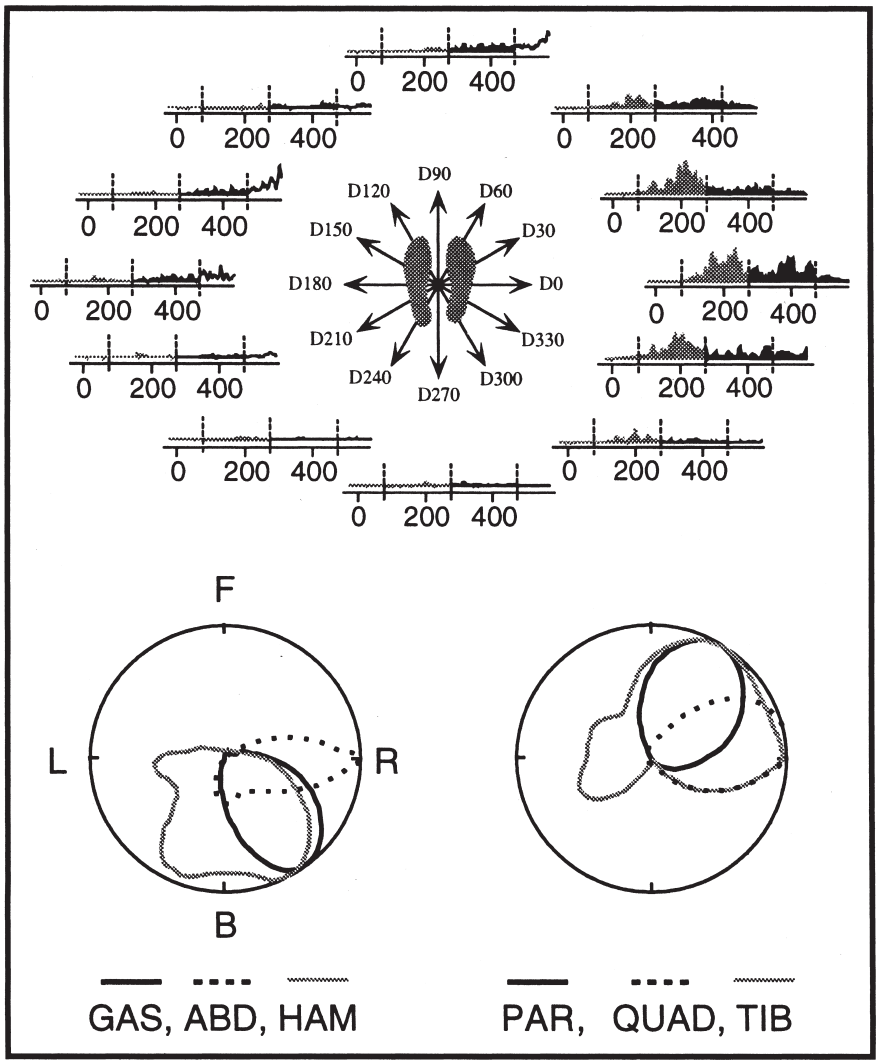

Figure 3.
The top portion of the figure shows electromyographic (EMG) activity from left tensor fasciae latae muscle that is averaged from five trials for each perturbation direction in narrow stance for one subject. The EMG integrals were calculated from individual trials using fixed windows (dotted lines) from 70 to 270 ms (early) and from 270 to 470 ms (late) after platform onset (0 ms). The bottom portion of the figure represents the six muscle amplitude changes with perturbation direction superimposed upon each other. These muscle "tuning curves" are created by normalizing the EMG integrals such that the maximum EMG amplitude reaches the circle radius. The tuning curves generally group into one of two diagonal regions. PAR=lumbar paraspinal muscles, ABD=rectus obdominis muscle, HAM=hamstring muscles, QUAD=rectus femoris muscle, GAS=gastrocnemius muscles, TIB=tibialis anterior muscle. (Adapted from Henry et al.[39])

able amplitudes and lack of scaling when the same amplitudes were unpredictable (Fig. 5C).

Many patients with neurological disorders show abnormally small (hypometric) or large (hypermetric) responses to postural displacements (Fig. 5B) despite normal latencies. For example, patients with Parkinson's disease often show hypometric postural responses related to slow buildup of EMG activation and coactivation. In contrast, patients with midline cerebellar disorders, such as anterior lobe atrophy, often show hypermetric postural responses in which each EMG burst is too large and too long, resulting in falls in the direction opposite to the direction of perturbation.[48] Patients with cerebellar dysfunction try to compensate for these hypermetric responses with large, reciprocal activation of antagonists. Despite the fact that persons with parkinsonism and cerebellar dysfunction show abnormal magnitudes of postural response, their ability to increase the magnitude of responses based on velocity feedback is intact.[60] In contrast to this ability to use sensory feedback for velocity modification, patients with cerebellar dysfunction show the unique problem of inability to adjust their postural responses based on prediction from prior experience (Fig. 5C).[60] Patients with parkinsonism adjust to predicted amplitudes when small forces are required, but they have difficulty generating the larger forces required to adjust responses to larger amplitudes (Fig. 5C). Thus, the balance problems of both persons with parkinsonism and those with cerebellar dysfunction are partly related to abnormal force control, but in very different ways, suggesting very different functional problems.

Adaptation of Postural Strategies

Context-specific adaptation. Results from platform perturbation studies have provided insight into the adaptability of the postural system. These studies have shown that automatic postural coordination is flexible and adapted to particular tasks and contexts based on the sensory information specific to each condition. The particular muscle synergies activated in response to an external perturbation depend on initial body position,[41,63] the initial support conditions,[19,64] and the location and characteristics of the sensory stimuli triggering the response.[61]

Experimental studies have shown that any group of muscles or any body segment can be used in a postural role, depending on the initial body positions and support conditions.[65] For example, responses to surface translations while maintaining bipedal versus quadrupedal stance results in a switch from using primarily ankle extensors to using primarily ventral hip flexors and co-contraction of arm muscles in both humans and cats

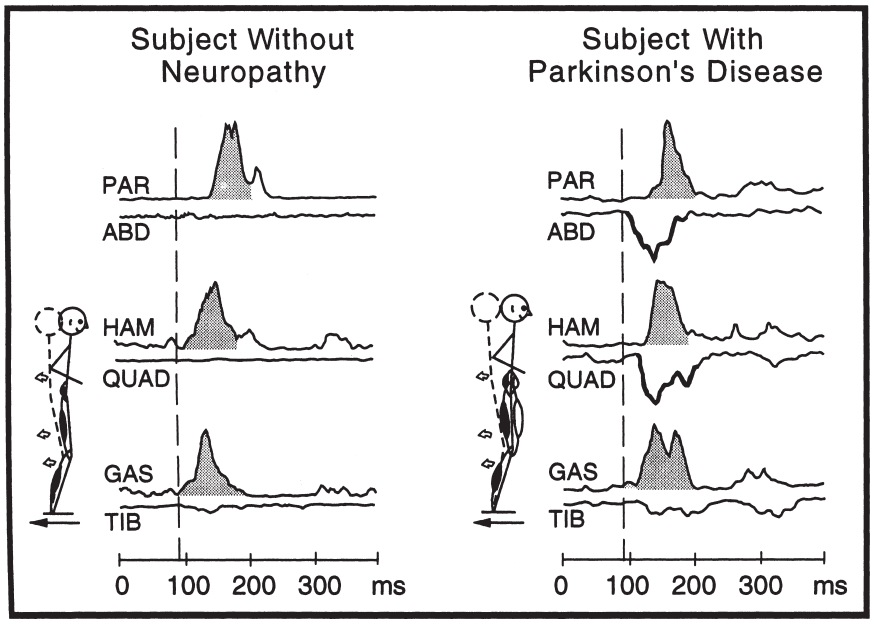

Figure 4.
Typical patterns of muscle activation in representative subject without neuropathy and subject with Parkinson's disease in response to backward surface translations. Dorsal electromyographic bursts associated with an ankle strategy are shaded, and ventral bursts associated with a hip strategy are thickened. The subject with Parkinson's disease showed normal latencies to activation of the gastrocnemius muscles (GAS), the hamstring muscles (HAM), and the paraspinal muscles (PAR), but with addition of coactivation of antagonists. Stick figures show muscles activated, and small arrows indicate direction of active correction for forward sway displacement. ABD=rectus abdominis muscle, QUAD=rectus femoris muscle, TIB=tibialis anterior muscle. (Adapted from Horak et al.[47])

(Fig. 6A).[66] Changing from a wide to a narrow stance also alters automatic postural response patterns by increasing the magnitude of responses and including more trunk activation (Fig. 6B). If individuals initially assume a leaning position prior to a perturbation, the trunk muscles rather than the ankle muscles may be activated first.[63]

When initial body positions or support conditions are changed, the same surface perturbation will result in activation of a different set of muscles on the first trial; muscles whose action is not effective in restoring equilibrium are not activated. For example, when persons support themselves by holding a handle during surface perturbations, automatic postural responses in the legs are suppressed and postural activity originates at the interface of the body and the stable surface (eg, hand, arm) (Fig. 6C).[67] Similarly, individuals who are perturbed while sitting on a stool with legs dangling use trunk, and not leg, muscle activation on the first trial.[47,68]

Practice. Perturbation studies have shown that postural strategies become more efficient and effective in response to repeated exposure to a destabilizing stimulus. When people are repeatedly exposed to a particular postural perturbation, their automatic postural responses are gradually reduced in magnitude and fewer, or different, muscles are recruited as these individuals change from a more vigorous to a less vigorous response.[62] Initial postural responses to unexpected disturbances are larger than necessary and inefficiently executed, with excessive muscle activation.[9]

Reduction in the magnitude of postural responses with repeated surface translations is shown in Figure 7A, which compares the first 10 and last 10 of 100 sequential responses. Although onset latency does not change with practice, the magnitude of responses is reduced, especially in antagonist muscles (Fig. 7B). Performance improves with less effort after practice because the time (t) it takes to reach a stable equilibrium position (sway in Fig. 7A) decreases as the torque response reduces with repetition. Although initial responses to unexpected perturbations bring the body's COM back near to its initial position, trials later in a session and over months of training show that people "pre-lean" in the direction of the predicted sway due to perturbation.[37,62] This type of "pre-leaning" can provide a mechanical advantage by increasing joint stiffness.[69]

Teaching voluntary postural responses. Voluntary responses to postural perturbations generally have a longer latency than automatically triggered postural responses.[19,49,70,71] The muscle activation patterns with voluntary sway or steps, however, may have a spatial and temporal organization that is similar to that of automatic postural responses. Although slower, these voluntary responses can be fast enough to be effective in preventing falls in patients whose automatic response latencies are delayed by sensory loss.[50] Attempts to alter automatically triggered postural responses in adults without neurological impairments by providing prior information regarding the size or direction of an impending perturbation have been unsuccessful,[72,73] although prior instruction to "resist" or "give" in response to an upcoming perturbation can alter response magnitude (Horak FA, unpublished observations).

Recent studies have shown that automatic postural responses can be suppressed by a person's intent to move, provided that the person can anticipate the characteristics of the upcoming perturbation.[70,74] When people are instructed to step, instead of remaining in

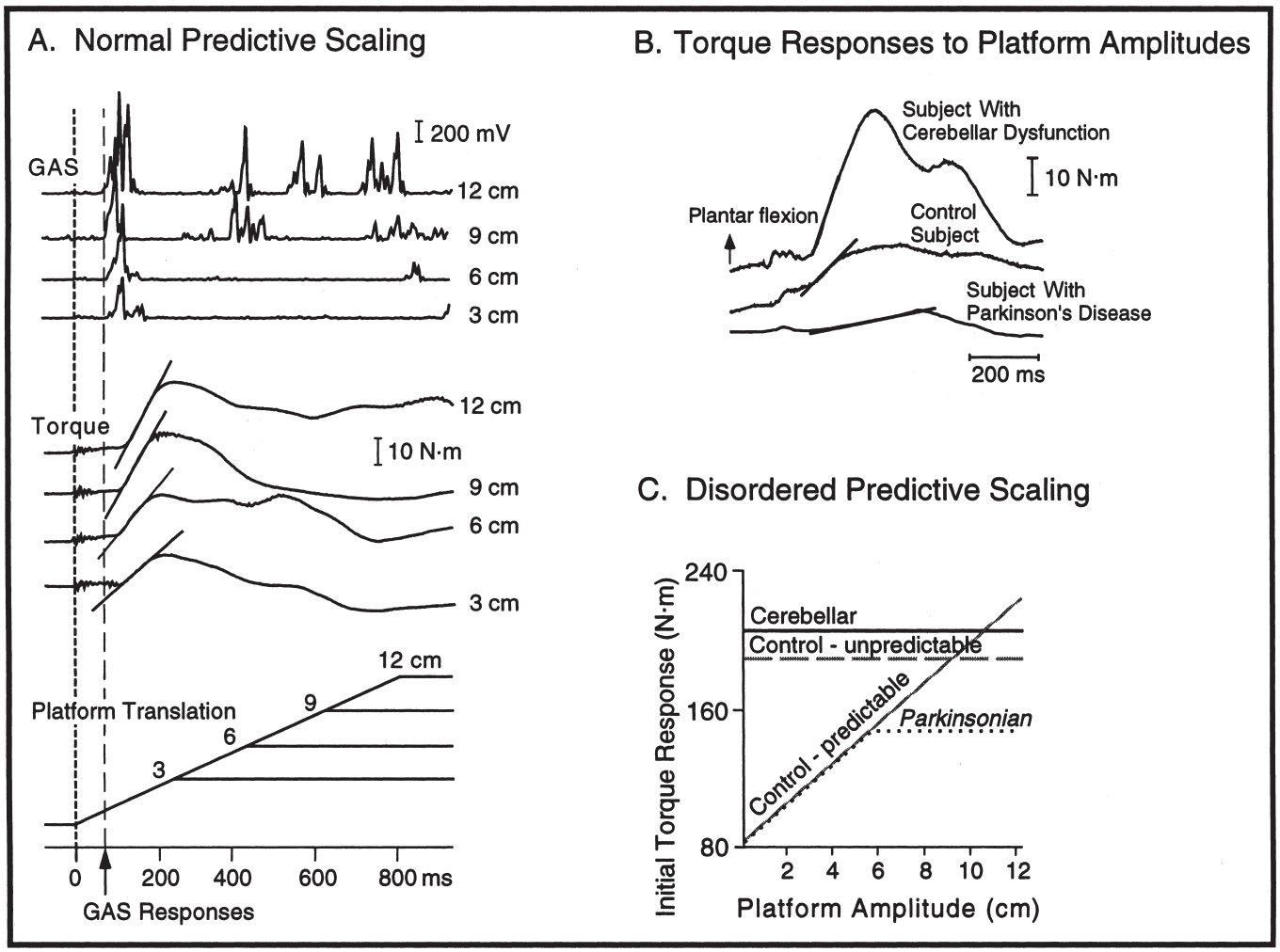

Figure 5.
(A) Initial gastrocnemius muscle (GAS) burst and resulting surface torque occur prior to completion of platform translations but are adjusted to platform amplitude when they are predictable (each amplitude is presented as a block of 5–10 like trials). (B) Effect of platform displacement amplitude (6 cm) on surface reactive torque responses in a representative subject with cerebellar dysfunction, a control subject, and a subject with Parkinson's disease. Rate of change of initial torque response (slope of regression during first 75 ms of active torque) is indicated by bold line superimposed on each torque response (average of 10 like trials). (C) Modification of postural response torque based on prediction of platform translation amplitude. Control subjects are able to modify their initial torque responses using prior experience if platform amplitude is predictable. The lack of adjustment of initial torque responses is seen when the same amplitudes were unpredictable. Persons with cerebellar dysfunction are unable to adjust their postural responses based on prediction from prior experience, whereas persons with Parkinson's disease adjust to predicted amplitudes only when small corrections forces are required. (Figs. 5A and 5C adapted from Horak[9]; Fig. 5B adapted from Horak and colleagues.[47,48])

place in response to a backward surface translation, they can suppress their automatic postural response and initiate a very rapid step. Figure 8 shows the suppression of the automatic soleus and gastrocnemius muscle activation in response to a backward surface translation with activation of the tibialis anterior muscle in order to move the COM forward in preparation for a step. The tibialis anterior muscle's EMG latency for intentional step initiation is longer than that for in-place automatic postural responses, suggesting that that voluntary, or cortical, mechanisms have substituted for the suppressed automatic postural mechanisms. This suppression of postural responses, however, is larger when individuals can predict the speed of the impending perturbation.[74] Thus, although functional, voluntary postural responses to external perturbations can be taught, patients cannot be expected to substitute fast, voluntary responses for automatic postural responses unless they are able to predict an upcoming perturbation.

Summary of Studies

Studies of automatic responses to external surface perturbations have identified quick, coordinated, multisegmental strategies responsible for maintaining equilibrium. These strategies are organized to control a variety of postural objectives and are adapted to particular conditions, behavioral goals, and environmental contexts. These strategies become more efficient with practice. Studies have also shown that patients with neurologic impairments may show many different types of

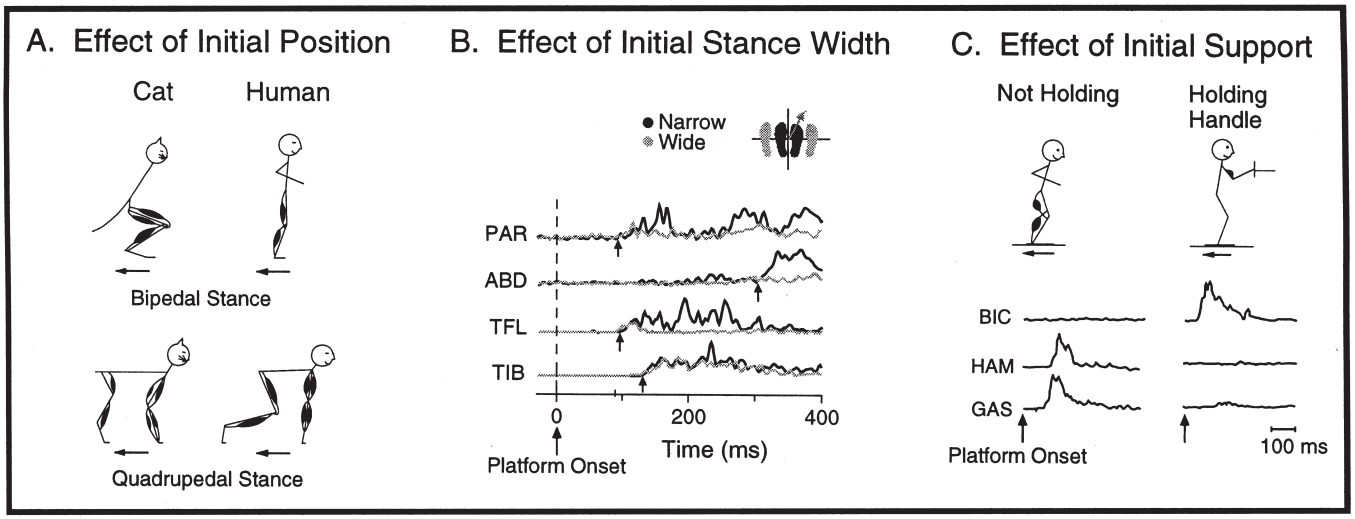

Figure 6.
(A) Cartoon shows which muscles responded to backward surface translations in bipedal and quadrupedal stance in cats and humans. (B) Average electromyographic responses from left muscles recorded during five horizontal translations that were forward and to the right in narrow (dark line) and wide (gray line) stance. Electromyographic amplitude decreases with wide stance, but latencies (arrows) do not change. (C) Muscle responses to backward surface translations during stance while not holding (on left) and while holding (on right) a handle. PAR=lumbar paraspinal muscles, ABD=rectus abdominis muscle, TFL=tensor fasciae latae muscle, TIB=tibialis anterior muscle, BIC=biceps muscle, HAM=hamstring muscles, GAS=gastrocnemius muscles. (Adapted from Henry et al,[39] Dunbar et al,[66] and Nashner.[67])

abnormalities in coordinating postural responses to disequilibrium, depending on the particular system involved and the underlying postural control impairments.

Clinical Implications of Perturbation Research

The Changing Face of Clinical Practice

Treatment success for patients with impaired balance depends on an understanding of (1) the systems controlling normal equilibrium, (2) the postural systems that are likely to be disordered by aging and pathology, and (3) a clinical framework for assessing and treating imbalance that is consistent with current research on postural control and relevant to the needs and problems of patients with impaired balance. Results from research characterizing equilibrium responses to external surface perturbations have provided insights into the contributions and interaction of the many systems that are important to normal balance. In addition, postural perturbation research has provided an approach for understanding the basis for instability during aging and in the patient with neurological pathology. This information is being used to develop a new clinical framework for assessing and treating instability, specifically by targeting the component problems that contribute to instability.[7,8,32,75]

Much has been written recently about changing theories of motor control and their effect on clinical practice.[7,8] Traditionally, balance has been conceptualized as resulting from a series of hierarchically organized reflexes and reactions.[7,8,75] This theoretical framework led to the development of assessment tools that measured the presence or absence of reflexes and interventions that focused on inhibiting primitive and pathological reflexes and facilitating the emergence of normal equilibrium reactions.[75]

Perturbation studies have shown that movement strategies for achieving balance are not the result of stereotyped reflexes, but emerge as the CNS learns to apply generalized rules for maintaining equilibrium in a variety of tasks and contexts. We suggest that balance can be viewed as a motor skill that emerges from the interaction of multiple systems that are organized to meet functional task goals and that are constrained by environmental context. Balance viewed as a motor skill suggests that, like any skill, balance can improve with practice. That is, postural motor coordination can be learned. This view of balance has led to the development of assessment tools that focus on measuring functional capacity of the patient and on quantifying underlying impairments that constrain functional performance. Therapeutic intervention is directed at changing impairments and improving functional performance, including the capacity to adapt performance to changing task and environmental demands.[8,75]

In the remaining section of this article, we discuss some of the clinical applications that have been developed in response to insights gained from postural control research. The development of new clinical approaches based on emerging research related to postural control is just beginning. Many of the new approaches discussed in the next section have yet to undergo experimental

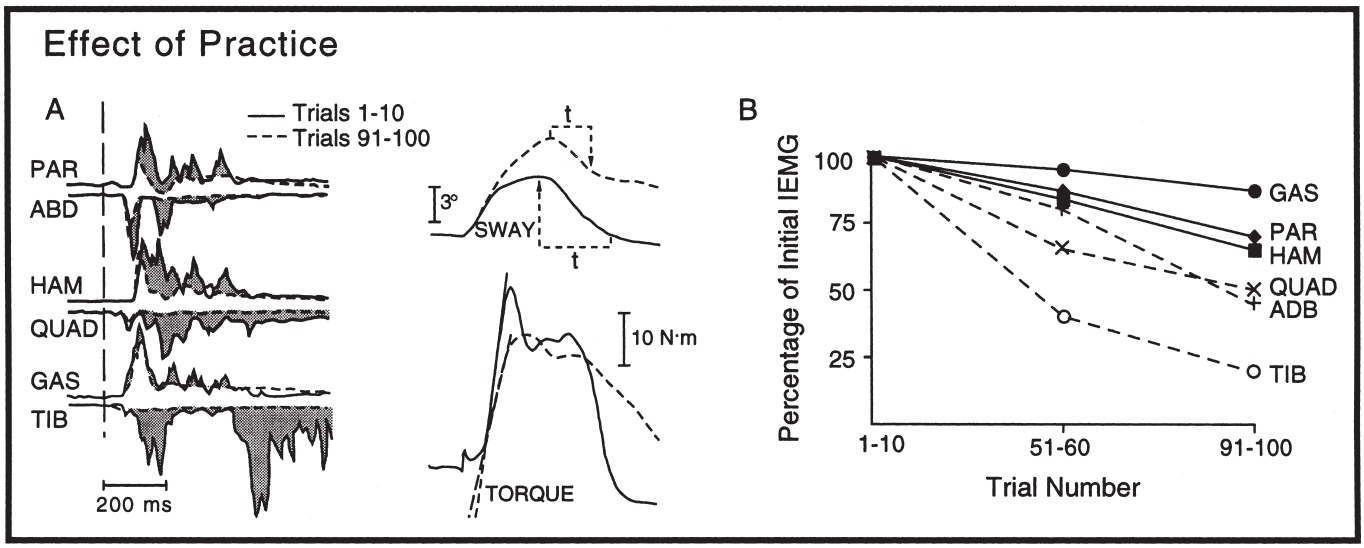

Figure 7.
Reduction of electromyographic (EMG) activity and forces with practice. (A) Reduction of EMG response, torque, and time to achieve equilibrium (t) with repetition of forward sway induced by a backward surface perturbation. Solid lines show average response of trials 1 to 10, and dashed lines show average response of trials 91 to 100. (B) Graph shows average reduction of integrated EMG (IEMG) activity (first 100 ms) in trials 51 to 60 and in trials 91 to 100 as a percentage of initial trails 1 to 10. Trial-to-trial variability was less than 10% of the average values. PAR=lumbar paraspinal muscles, ABD=rectus abdominis muscle, HAM=hamstring muscles, QUAD=rectus femoris muscle, GAS=gastrocnemius muscles, TIB=tibialis anterior muscle. (Adapted from Horak et al.[62])

validation. Nevertheless, the face of clinical practice appears to be changing in response to many factors, including recent research in postural control.

Synergies and Strategies

The selection of movement strategies that are effective in controlling the body's COM depends on numerous factors, including the biomechanical, sensory, and neuromuscular constraints within the individual, as well as on the affordances of the environment and task. Helping patients to develop effective and efficient movement strategies for postural control depends on understanding the postural system disorders that result in constraints limiting the availability and selection of movement strategies for balance.

Biomechanical constraints. Biomechanical constraints affect the development and selection of movement strategies used for balance. The development of coordinated multijoint movement strategies during balance retraining involves helping patients to develop efficient and effective ways to control their body's COM despite biomechanical constraints imposed by their musculoskeletal impairments or disabilities. For example, a musculoskeletal impairment such as limited range of motion at the ankles due to shortening of the gastrocnemius and soleus muscles may limit a patient's ability to generate forces against the surface to control the COM. Treatments aimed at lengthening these muscles may allow the person to resume use of postural movements relying on force generation at the surface. In cases in which impairments are permanent, such as may occur with an arthritic ankle, alternative postural strategies that are effective in controlling the COM must be developed. In these circumstances, use of a movement strategy that controls COM position largely through movements at the trunk, hips, and arms, or alternatively taking a step, may be appropriate.

Sensory information available. Strategy selection is also influenced by the sensory information that is available. For example, a permanent loss of sensory information may result in a restricted range of movement strategies available for postural control. In this case, patients may need to be educated to identify and avoid dangerous tasks and environments or to develop compensatory strategies using assistive devices.[76,77]

To improve balance control in patient populations where one or more senses may be impaired or not used effectively by the nervous system, therapists can develop approaches to help patients learn effective ways to organize and structure remaining senses to achieve postural control. Suggested treatment strategies designed to affect sensory systems that are important to postural control have focused both on resolution of underlying sensory impairments and on improving the organization and adaptation of sensory information for postural control.[78] For example, patients with partial vestibular loss can be taught to utilize ("tune up") the residual vestibular information that is available.[76] If the patients are unable to do this, the therapist can teach them to recognize dangerous environmental contexts and to use alternative visual and somatosensory informa-

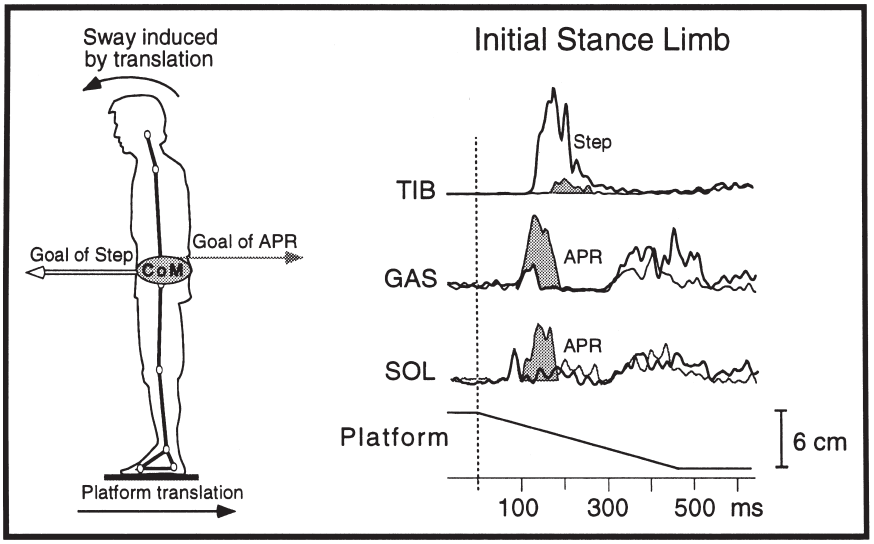

Figure 8.
Intent to step alters postural response. With intent to step, the body's center of mass (COM) moves forward with tibialis anterior muscle (TIB) activation, whereas in response to forward sway induced by a backward platform translation, the COM moves backward via the automatic postural response (APR) in the gastrocnemius muscles (GAS) and the soleus muscle (SOL) (shaded electromyographic response). With simultaneous backward platform perturbation and step initiation (open electromyographic response), the GAS and SOL postural responses are suppressed. (Adapted from Burleigh et al.[70])

tion for postural control. Initially, this type of patient should be treated barefoot on a firm surface and encouraged to attend to the sensation from the feet. Then, the information from the support surface can be made less accurate by having the patient stand on foam, which will encourage the use of vision and remaining vestibular information for orientation. Additional challenges that encourage the use of remaining vestibular information can be introduced by making the available visual information gradually more inaccurate as an orientation reference.[76,78]

Practice sessions for retraining balance can be designed to structure opportunities so that patients have a chance to become efficient in the use of given postural strategies. By initially providing a situation with accurate sensory information and progressing to more complex sensory environments, patient have an opportunity to practice while maintaining a specific level of function in increasingly difficult contexts.

Use of assistive devices. Physical therapists may choose to use assistive devices as part of their therapeutic plans. The use of a cane, for example, will alter the strategy selected based on the changes in the initial biomechanical conditions and based on the available sensory information. A study is currently underway to determine whether sensory information from light touch of the fingertip with a surface can effectively substitute for absent or impaired sensory information from the lower extremities in people with peripheral neuropathies (Horak and colleagues, unpublished research). Cutaneous information from fingertip contact with a stable surface can be more powerful than vision in stabilizing sway in stance.[79] Thus, assistive devices change both the biomechanical and sensory constraints for posture.

Improving Response Latencies

Delayed onsets of muscle responses to surface perturbations can result in increased sway and, if sufficiently delayed, an inability to regain equilibrium using an in-place movement strategy. If patients are slow in taking a step to recover balance, a fall will occur. Slowed responses can result from delays in sensory or motor conduction or from central processing problems. In most clinical settings, it is difficult to identify the problem of delayed onset latencies without force-plate and EMG recordings. Suggested treatments for shortening onset latencies include the use of sensory stimulation such as ice, sweeping, tapping, vibration, and stretching over the postural muscles themselves to increase the excitability of the motoneurons and bring them closer to firing. Other treatments designed to improve the effective recruitment of a muscle for balance include the use of electrical stimulation in conjunction with a footswitch and the use of single-channel or multichannel EMG biofeedback.[75]

Interventions aimed at improving the organization and interpretation of sensory information for postural control have been shown to have the additional effect of decreasing onset latencies of muscle responses to platform perturbations.[80,81] In a study by Hu and Woollacott,[80,81] elderly subjects practiced maintaining stance balance under altered surface and visual conditions over a period of weeks. At the end of the training period, the elderly subjects had improved their ability to stand on a compliant surfaces, and they demonstrated shortened latencies to postural responses to surface displacements.

If delayed onset latencies are due to a decreased ability to generate and sustain forces, as occurs with Parkinson's disease, interventions designed to improve force production could, in theory, affect how quickly effective movements for recovery of balance are generated. Context-specific strengthening exercises and biofeedback may also be effective treatments for improving recruitment of muscles and the rate of force development in muscles needed for postural control.

Temporal and Spatial Dyscoordination

Studies recording EMG activity in response to surface perturbations have provided considerable insight into spatial and temporal disruptions to multijoint muscle synergies essential to the recovery of balance. Although clinicians are able to observe the behavioral outcomes of dyscoordinated movements, such as buckling of the knees, excessive lateral sway, or rotation of the body, the exact nature of the dyscoordination can be determined only with the use of technology such as recording of EMG, kinematic, and kinetic patterns. This inability to identify the source of dyscoordination can hinder the development of interventions designed to improve the spatial and temporal coupling of multijoint muscle activation for balance.

Therapeutic interventions designed to improve coordination problems affecting the ability to maintain stability can begin with treatments aimed at eliminating any biomechanical constraints that may be contributing to the dyscoordination. Developing efficient and coordinated movement strategies for balance may be facilitated by having the patient practice performance of a task that requires the desired strategy. For example, working with the patient on an inclined surface such that the COM of the body is forward so the knee is less likely to buckle would allow a patient to practice the task of moving the COM without buckling of the knee.

Another potential approach to dyscoordination problems is to focus on the synergy level; that is, give patients feedback about muscle recruitment patterns. It has been well documented that to recover from instability in the backward direction, the tibialis anterior muscle fires first, followed by firing of the quadriceps femoris muscle about 30 milliseconds later.[22] In children with diplegic cerebral palsy[56] and some adults with stroke,[46] the recruitment pattern may be abnormal, with the quadriceps femoris muscle firing in advance of the tibialis anterior muscle, resulting in knee bucking. Multichannel electrical stimulation based on EMG monitoring has been used to stimulate the quadriceps femoris muscle, based on the timing of the tibialis anterior muscle activation during backward sway, to improve the distal-to-proximal temporal coordination of muscle activation to recover equilibrium.[75]

Force Control

Modifying forces appropriate to the speed and amplitude of body sway is a critical aspect of effective postural control. Generating too much force is as detrimental to the recovery of equilibrium as insufficient force generation.[48] Both hypermetric and hypometric responses result in the inability to bring the COM back to a point of stability with respect to the base of support following a perturbation.[48]

Proportional control of postural responses is based on characteristics of the perturbation. A wide variety of amplitudes and speeds of displacement, therefore, provides patients with the best opportunity to learn to modify their responses appropriately. Because predictive aspects of postural control are important to learning proportional force control, using identical perturbations that are repeated sequentially can help patients utilize prediction in formulating their postural responses. If perturbation characteristics are altered randomly, patients must rely more on reactive control.[9,62]

Numerous techniques have been developed to help patients improve the modification of forces used to predictively control postural movements. Appropriate modification of forces is needed for voluntary COM movement and for anticipatory postural adjustments when the amount of force needed to control the COM can be predicted, as well for responses to external perturbations. For voluntary postural movements, force-plate retraining systems have been designed to provide patients with visual feedback regarding movement of the center of pressure (COP) at the feet. The capacity to move the COP to designated targets placed in different quadrants on the computer monitor requires effective proactive modification of forces. This technology has been useful when retraining hypometric, as well as hypermetric, force responses (Shumway-Cook, unpublished observations). We have found that people with Parkinson's disease who demonstrate hypometric force responses respond best to a treatment progression that begins with small, slow movements of the COP and moves to large, fast movements of the COP.[61] In contrast, people with cerebellar disorders who demonstrate hypermetric force responses may need to start with large, fast movements of the COP and progress to small, slow movements of the COP.[75] The degree to which training on force-plate systems transfers to other functional tasks such as walking or responding to external perturbations has yet to be determined.[82] There is some evidence to suggest that force-plate retraining in patients with stroke does not transfer well to functional tasks such as gait.[6,81] Further research in this area is needed.

Adaptation of Postural Strategies

Context-specific adaptation. Recovery of stability requires the adaptation of effective and efficient sensorimotor strategies for postural control in the face of constraints imposed by pathology. In addition, patients must be able to adapt sensory and motor strategies to changes in task and environmental demands. We propose that the capacity to adapt is a sign of good potential for rehabilitation because it suggests the potential for change in the face of existing impairments and constraints. Research using surface perturbation paradigms

indicates that under normal conditions, the strategies used to control the COM change quickly to accommodate the task and conditions.[39,66,67]

Helping people with balance disorders learn context-specific adaptation involves facilitating the development of effective strategies for controlling the COM in a wide variety of tasks and contexts. We believe that treatment should not be limited to training a single or limited set of strategies, but should include helping patients to develop strategies appropriate for each motor problem and to learn in which situations and contexts to deploy these strategies. For example, if a therapist has a patient hold on to parallel bars (or to the therapist's hands), the initial biomechanical and sensory information has changed considerably. In this context, the therapist should expect to see activation of the patient's arm muscles first during the balance response. If a patient assumes a quadrupedal position, COM is controlled primarily with hip and shoulder muscles, unlike in stance. If the patient assumes a wide stance, he or she will need to activate lower-limb muscles to a lesser degree than if standing in a very narrow stance, because the wider stance is inherently more stable.[39] By exposing patients to a multitude of environments, they are afforded the opportunity to learn how to develop context-specific strategies for solving the problems of maintaining equilibrium.

Teaching voluntary responses. People can be taught to suppress their automatic postural responses if the responses are not functional for their intended goals, especially if they can predict an upcoming perturbation.[70,74] Therapists can exploit this finding by having patients practice suppressing ineffective postural responses during functional activities. For example, patients with Parkinson's disease often have difficulty initiating postural adjustments in preparation for stepping. Therapists can assist the stepping by first moving the patient diagonally over the anticipated swing leg, then forward to the stance leg, thereby displacing the COM in preparation for a step. Initially, the patient may elicit an equilibrium response in response to the push, but with practice the therapist can teach the patient to suppress the unwanted automatic postural response and, instead, initiate a step. Such external perturbations have been shown to assist step initiation in patients with Parkinson's disease.[74]

Limitations of Postural Perturbation Research

Although studies of automatic responses to surface perturbations has increased our understanding of the underlying mechanisms involved in postural control, therapists should not limit their evaluation of balance to descriptions of responses to perturbations. Because balance control is multidimensional, involving proactive as well as reactive control, tonic as well as phasic control, and sensory as well as motor control, it is not realistic to think that any one type of postural task will be useful to analyze all of these aspects of balance control effectively. Responses to perturbation, for example, do not attempt to test the mechanisms involved in orientating the body to various sensory reference frames, steady-state stance posture, or anticipatory postural coordination with voluntary movements. Responses to perturbations of the support surface in a standing subject cannot necessarily be used to predict how the subject will adapt his or her responses to other positions, such as when sitting or kneeling. Responses to external perturbations during stance do not provide information on mechanisms needed to control balance during different tasks (eg, transferring positions), during locomotion, and while stepping over obstacles.[83] In addition, characterization of equilibrium responses to postural perturbations does not necessarily allow for the prediction of how a patient will perform functionally in activities of daily living, although it may allow for the prediction of how well the patient will respond to similar situations such as reacting to surface perturbations induced by accelerations and decelerations of a bus or subway train, a jostle in a crowd, or a pull by a pet on a leash.[84] Additional research is needed to determine the predictive value of responses to postural perturbations.

Conclusions

The ability to effectively treat patients with balance disorders will be enhanced by a clearer understanding of the problems underlying imbalance. Identifying limitations in functional performance, such as inability to stand or walk independently, does not provide information on the underlying impairments, such as prolonged latencies, poor coordination, inadequate force, or inability to adapt postural responses that may be constraining functional performance. More research is needed to develop clinical assessment tools that are effective in identifying specific problems in systems that are essential to postural control. In addition, further research is needed to determine the relative efficacy of different treatment approaches to balance disorders and the extent to which patients are able to learn effective strategies for controlling equilibrium despite the presence of underlying impairments. By understanding the basic systems underlying control of posture, therapists may be able to focus their treatments on deficits that are specific for each patient. Quantification of postural responses via new technology has the advantage of measuring changes in postural behavior that are too small to observe, of differentiating among several potential disordered systems, and of documenting progress related to rehabilitation.

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