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Cortical control of postural responses

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Summary This article reviews the evidence for cortical involvement in shaping postural responses evoked by external postural perturbations. Although responses to postural perturbations occur more quickly than the fastest voluntary movements, they have longer latencies than spinal stretch reflexes, suggesting greater potential for modification by the cortex. Postural responses include short, medium and long latency components of muscle activation with increasing involvement of the cerebral cortex as latencies increase. Evidence suggests that the cortex is also involved in changing postural responses with alterations in cognitive state, initial sensory-motor conditions, prior experience, and prior warning of a perturbation, all representing changes in "central set." Studies suggest that the cerebellar-cortical loop is responsible for adapting postural responses based on prior experience and the basal ganglia-cortical loop is responsible for pre-selecting and optimizing postural responses based on current context. Thus, the cerebral cortex likely influences longer latency postural responses both directly via corticospinal loops and shorter latency postural responses indirectly via communication with the brainstem centers that harbor the synergies for postural responses, thereby providing both speed and flexibility for pre-selecting and modifying environmentally appropriate responses to a loss of balance.

Keywords: Cerebral cortex, automatic postural responses, posture, balance

Postural responses are influenced by cortical function

To maintain postural equilibrium in daily life we often need to quickly respond to external perturbations, such as stumbling over obstacles; slipping on wet, icy or compliant surfaces; or pushing by an opponent during sport. The extent to which the fast, automatic postural responses used to recover postural equilibrium can be influenced by voluntary intention and by mental disease depends on the extent to which they are controlled by the cerebral cortex. Participation of the cortex in postural control is controversial and debated. In this article, we will review the evidence for cortical involvement in shaping postural responses that are evoked by external postural perturbations.

Historically, the neural control of automatic postural responses was thought to arise from brainstem and spinal circuits with little consideration for the role of the cerebral cortex (Magnus, 1926; Sherrington, 1910). The cortex was not considered essential for the control of posture because animals with transections at the midbrain (thus eliminating input from the cerebral cortex to lower neural centers) retain many "reflexes" that correct and maintain stance posture (Magnus, 1926; Sherrington, 1910); a point of view that was embodied by Magnus (1926) when he wrote, "the whole righting apparatus . . . is arranged sub-cortically in the brainstem, and in this way made independent of direct voluntary influences." In addition to these early reports, the idea that postural responses were regulated subcortically persisted with time, partly because postural responses are triggered automatically, without voluntary intent, and are initiated more quickly and with less variability than cued, voluntary movements (Diener et al., 1984; Keck et al., 1998).

Although responses to postural perturbations occur more quickly than the fastest cued, voluntary movements, the onset of postural responses occurs at longer latencies than those of spinal stretch reflexes (Chan et al., 1979; Matthews, 1991), suggesting that postural responses exhibit greater potential for modification by neural centers residing higher along the neural axis. Indeed, animals and humans with cortical lesions that spare the brainstem exhib-

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it abnormal postural responses to external perturbations (Bard, 1933; Brooks, 1933; Chan et al., 1979; Diener et al., 1985; Geurts et al., 2005; Magoun and Ranson, 1938; Rademaker, 1931), thereby supporting the notion that postural equilibrium is influenced by the cerebral cortex. In addition, unlike stretch reflexes, postural responses involve activation of muscle synergies throughout the entire body and are also more context-specific, flexible and adaptable than spinal proprioceptive reflexes (Horak and Macpherson, 1996).

Behavioral evidence also implicates the cerebral cortex as contributing to postural responses because they are modified by complex cognitive-motor processes thought to be mediated by the cerebral cortex, including: (1) changes in cognitive load and attention when performing concurrent tasks (Brauer et al., 2002; Brown et al., 1999; Carpenter et al., 2004; Maki et al., 2001; McIlroy et al., 1999; Norrie et al., 2002; Quant et al., 2004a; Zettel et al., 2005), (2) changes in a subject's intentions to respond with a specific strategy (Buchanan and Horak, 2003; Burleigh et al., 1994; Burleigh and Horak, 1996; McIlroy and Maki, 1993), (3) learning and modification of postural responses with prior experience (Diener et al., 1988; Horak and Nashner, 1986; Horak et al., 1989; Maki and Whitelaw, 1993; McIlroy and Maki, 1993; Quintern et al., 1985), and (4) with changes in initial conditions (Chong et al., 1999; Henry et al., 2001; Tjernstrom et al., 2002; Zettel et al., 2002a, b).

In addition, attention, mental calculation, and memory have been attributed to represent high-order cognitive functions, controlled by the cerebral cortex (Dehaene et al., 2004; Kaiser and Lutzenberger, 2005; Naghavi and Nyberg, 2005). Thus, interactions among mental performance and balance function suggest cortical involvement in postural equilibrium. For example, in cerebral stroke patients, it has been shown that the extent of their deficits in divided and sustained attention correlate with their fall history and balance function (Hyndman and Ashburn, 2003). In addition, in response to an imposed loss of balance, a secondary task depresses the amplitude of the perturbation-evoked cortical potentials (recorded by electroencephalography; EEG) and increases the amplitude of the perturbation-evoked postural sway (Brown et al., 1999; Quant et al., 2004a). The interference between a cognitive task and perturbation-evoked potentials demonstrates that the cortical representation of sensory feedback arising from perturbed posture becomes attenuated when performing other tasks, and that this attenuated cortical representation corresponds to impairments in the postural response (Quant et al., 2004a). In addition to attention, generalized cognitive function (as assessed by clinical exams of mental calculation, orientation, and memory) correlates with balance function (as assessed by dynamic posturography or by clinical tests of balance), and subjects with dementia are at an increased risk for falls (Buchner and Larson, 1987; Hauer et al., 2003; Kose et al., 2005). Thus, executive functions that are mediated by the cerebral cortex interact with postural control, thereby providing evidence that the activity of the cerebral cortex influences postural equilibrium. Therefore, contrary to Magnus (1926), the righting and equilibrium responses are definitely not independent of voluntary or cortical influences.

Cortical involvement in postural responses increases with increasing response latency

Whether postural responses involve long loops through the motor cortex has been debated and is still controversial (Beloozerova et al., 2005; Dimitrov et al., 1996; Keck et al., 1998; Solopova et al., 2003; Taube et al., 2006). Nevertheless, there is a general consensus that automatic postural responses involve short-latency (SL), medium-latency (ML) and long-latency (LL) components and that the likelihood of a transcortical loop contributing to the response increases with the latency of the response (Taube et al., 2006). The controversy comes with attempts to define the precise latencies for the 'medium' and 'long' latency components of the response, since the latencies depend on the conduction distance (height of subjects and whether the response is in an upper extremity or axial segment, versus in the lower extremity), characteristics of the perturbation (velocity, acceleration, direction, location, etc.), and initial conditions (background motor neuron and muscle activity, initial posture, etc; Horak and Macpherson, 1996). Because so many methodological factors affect the reporting of response latencies, it becomes difficult to define specific latencies that correspond to the SL, ML, and LL components across different studies. Thus, for this review, we will not attempt to define specific response latencies that correspond to cortical or sub-cortical response components, but will discuss in general terms (i.e., "initial" versus "late" phases) the evidence for cortical involvement in postural responses.

To characterize postural responses triggered by external perturbations in the laboratory, subjects are exposed to translations or rotations of the support surface, or the trunk is pulled or pushed, such that the body's center of mass is moved with respect to the base of foot support (Ackermann et al., 1991; Allum, 1983; Do et al., 1990; Horak and Nashner, 1986; Mille et al., 2003; Nardone et al., 1990; Nashner, 1977; Pidcoe and Rogers, 1998; Woollacott

et al., 1988). In response to these perturbations, a large group of muscles are quickly activated throughout the body (a postural synergy) to generate forces on support surfaces in contact with the body that counteract the forces imposed by the postural perturbation (Horak and Nashner, 1986; Ting and Macpherson, 2005). Even if the feet stay in place while postural responses move the center of body mass back over the base of foot support, the pattern of muscles used to counteract the perturbation depends on initial context (such as surface configuration, stance width, instructions, and emotional state), as well as prior experience (Horak, 1996; Horak and Macpherson, 1996). These feet-in-place automatic postural responses may also be accompanied by subsequent change-in-support responses, which include arm reaching or stepping (Maki and McIlroy, 2005). The change-in-support responses extend the base of support beyond the fall of the body's center of mass in order to reacquire equilibrium. Where and whether a subject steps or reaches in response to a perturbation can also be under voluntary control and is influenced by initial conditions, such as the location of safe step placement or hand rails (Ghafouri et al., 2004; Zettel et al., 2002a, b, 2005).

Figure 1 summarizes the potential neural loops involved in a postural response. Beginning at the spinal cord, movements of the support surface can elicit a short-latency activation of the distal leg muscles (Ackermann et al., 1991). Based on the activation latencies to electrically stimulate a monosynaptic spinal reflex from Ia afferents in these

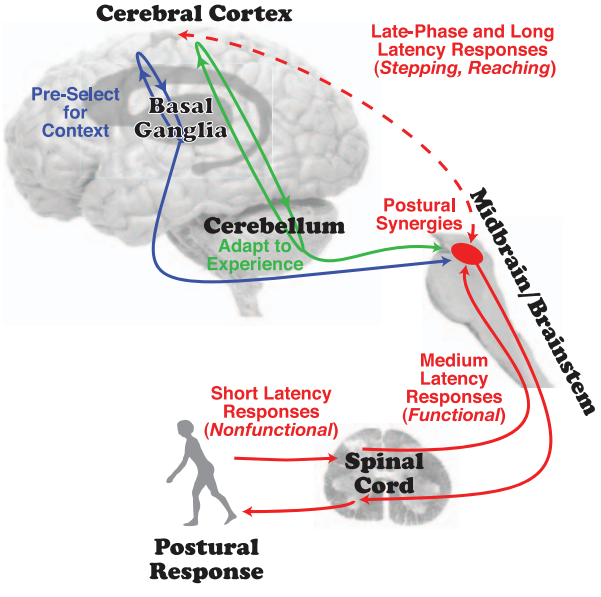

Fig. 1. A simple model of proposed neural pathways involved in cortical control of short, medium and long latency automatic postural responses to external perturbations

muscles (DeLisa and Mackenzie, 1982), the SL response likely represents the activation of a mono- or oligo-synaptic spinal, segmental circuit. Again, whether the SL response represents a mono- or oligo- synaptic spinal circuit depends on the initial conditions surrounding the perturbation (Ackermann et al., 1991). In isolation, the spinal cord's contribution to the postural response, however, is minimal because this SL response is too small to stabilize balance. In fact, although the spinal cord is sufficient to maintain antigravity muscle tone, cats with spinal transections and intact SL reflexes exhibit an inability to maintain unsupported stance or to maintain balance when exposed to postural perturbations (Fung and Macpherson, 1999; Macpherson et al., 1997).

Following the SL response, the feet-in-place postural response continues with functionally stabilizing muscle activations in whole body synergies (Horak and Nashner, 1986; Nashner, 1976), including the ML and LL responses. The onset of the functionally stabilizing response varies considerably with different perturbations and initial conditions (Ackermann et al., 1991; Chan et al., 1979; Horak and Macpherson, 1996; Horak and Nashner, 1986), and because of this variability, sometimes the ML response converges into the LL response. Because it is sometimes difficult to differentiate the ML response from the LL response, we will term these functional responses, the automatic postural response (excluding the non-functional, spinal-mediated SL response) allowing for a general discussion regarding cortical involvement in the neural control of postural responses, regardless of methodology.

The neurophysiology underlying the automatic postural response has been debated for decades to arise either from polysynaptic spinal loops (Ackermann et al., 1990; Berger et al., 1990; Dietz et al., 1984, 1985; Keck et al., 1998; Quintern et al., 1985) or from transcortical loops (Ackermann et al., 1986; Chan et al., 1979; Diener et al., 1985; Taube et al., 2006). A recent study suggests that the initial response likely arises from the brainstem instead of the cortex: decerebrate cats (despite many functional limitations) can maintain balance and exhibit intact, perturbation-specific muscular synergies when exposed to multiple directions of postural perturbations (Honeycutt and Nichols, 2006). As further evidence against a transcortical loop, in humans, changes due to repetition in the magnitude of the distal leg muscles' initial response do not correspond to changes in the perturbation-evoked cortical potentials that represent the sensory processing of the balance disturbance (Ackermann et al., 1990, 1991; Berger et al., 1990; Quintern et al., 1985). Further, while it has been argued that the latency of the initial response is sufficient for a transcorti-

cal loop (Chan et al., 1979), others have argued that the onset latency of the afferent perturbation-evoked cortical potential is only slightly shorter than that of the muscle response and, therefore, the efferent path of the initial postural response is not properly timed with the afferent cortical potential in order to signify a transcortical loop (Dietz et al., 1984, 1985).

Although the earliest part of the postural response may not involve a cortical loop, studies suggest that the cerebral cortex may become involved in shaping the postural response as the response progresses (that is, once latencies reach beyond the minimum sum conduction time of the afferent and efferent pathways of the cerebral cortex). For example, single-pulse transcranial magnetic stimuli (or conditioning repetitive stimuli) over the motor cortex increase the size of postural muscle responses and H-reflexes in the soleus muscle, but only when the probe stimulus occurs in the later phases of the response (Taube et al., 2006). In addition, a progressive increase in activation latency occurs when comparing postural responses from muscles in the arm, proximal leg, and distal leg, and this increase is too large to be attributed to differences in the lengths of the segmental spinal loops (Chan et al., 1979), suggesting that the postural response routes through supraspinal regions of the central nervous system. Further, intracranial recordings from standing cats and rabbits demonstrate that projection neurons and interneurons of the primary motor cortex modulate their activity in response to tilts of the support surface (Beloozerova et al., 2003, 2005).

Altogether, the literature suggests that a direct transcortical loop does not trigger the initial phase of postural responses to external perturbations, but it seems likely that the cerebral cortex becomes involved in later phases of the response. Thus, given that postural responses last for many hundreds of milliseconds, it may be that brainstem circuits initiate a response, and then the response subsequently becomes modified by cortical circuits during its later phases. Behaviorally, studies have found that performing a concurrent cognitive-motor task or altering the intention to step when responding to a postural perturbation (thought to represent cortical influence) only affects the later phases of the postural response (Burleigh and Horak, 1996; Norrie et al., 2002). To provide a specific example, we found that the response of the automatic postural response in the gastrocnemeus muscle to a backward surface translation could be completely inhibited when subjects intended to take a step in response to perturbations whose characteristics were predictable based on prior experience (Burleigh and Horak, 1996), whereas only the second 50-ms part of the muscle burst could be inhibited by voluntary intention when the perturbation velocities were randomized (Burleigh and Horak, 1996). These results suggest that prior intention to respond with a specific strategy (which we speculate involves cortical processes) to predictable perturbations enables modifications of the entire response, whereas responding to an unpredictable perturbation requires online response modification based on a subject's intentions (that is, online use of cortical influence), and this online cortical involvement is only capable of influencing the late phase of the postural response.

Unlike initial feet-in-place responses that likely depend upon brainstem neural loops, change-in-support responses (such as compensatory stepping and reaching responses) likely include a transcortical loop through the motor cortex for their initiation (Fig. 1). Early studies showed that animals with lesions of the motor cortex fail to generate compensatory steps (Bard, 1933; Brooks, 1933; Magoun and Ranson, 1938; Rademaker, 1931). In addition, the latencies of the stepping responses in humans are well within the range of what would be necessary to activate transcortical pathways; the stepping responses occur after the feet-in-place response of the distal leg muscles (Burleigh et al., 1994; Maki and McIlroy, 2005). Similarly, for arm reaching, after moving the floor under the feet during stance, the proximal arm muscles activate at latencies that are consistent with a transcortical pathway (McIlroy and Maki, 1995; Quintern et al., 1985). In addition, the reaching response and cortical perturbation-evoked potentials both attenuate as a subject becomes practiced through repeated perturbations, whereas the initial feet-in-place response from the distal leg muscles do not attenuate to the same degree or with the same time course (Quintern et al., 1985). When responding to unpredictable perturbations or unexpected perturbation characteristics, the early components of these perturbation-evoked cortical potentials have also been shown to be larger than when responding to predictable perturbations and are, therefore, thought to indicate a cortical error signal of sensory-motor processing related to the perturbation stimulus (Adkin et al., 2006; Quintern et al., 1985). Thus, the change-in-support responses may represent transcortical protective responses to unexpected postural disturbances, whereas the initial feet-in-place responses that inevitably precede them represent sub-cortical compensations to initially attempt to correct the postural disturbance (Quintern et al., 1985).

Cortical control of stepping and reaching in response to external perturbations is consistent with the ability to voluntarily alter which limb to use and its intended trajectory during compensatory limb movements, such that the stabi-

lizing features of protective stepping and reaching reactions can be modulated to meet environmental constraints (Ghafouri et al., 2004; Jacobs and Horak, 2006a; Tripp et al., 2004; Zettel et al., 2002a, b, 2005). Recently, we demonstrated that subjects with Parkinson's disease can also alter the length and direction of their compensatory stepping responses when provided with a visual target before a perturbation (Jacobs and Horak, 2006a). This paradoxical stepping (Souques, 1921) is remarkably similar to a PD subject's ability to improve voluntary stepping with external sensory cues, which has been reported to be related to compensatory activity of a circuit that includes the parietal cortex, dorso-lateral premotor cortex, and cerebellum (Hanakawa et al., 1999). Thus, cortical centers may also influence compensatory steps, rendering it feasible that similar cortical-brainsteim circuits govern both compensatory and voluntary stepping. These similarities are supported by reports that repetitive training of externally triggered postural responses also improves voluntary gait in elderly and PD subjects with impaired balance (Jobges et al., 2004; Rogers et al., 2003b).

Thus, rather than viewing the generation of postural responses as being either spinal or brainstem or cortical in origin, we should view the generation of postural responses as resulting from a dynamic and context-dependent interplay among all levels of the neural axis. Because they need to be fast, the earliest phases are most automatic with peripheral sensory input triggering synergies pre-set in the brainstem, whereas the later phases of the same responses are less automatic and can be modified to accomplish goals involving cortical loops.

The cortex influences central sensorimotor set

In addition to transcortical loops governing the generation of the postural response, the cerebral cortex may also influence postural responses in a more indirect fashion, by altering the circuits that generate the postural response through anticipatory control, prior to a perturbation. Changes in postural responses with alterations in cognitive state, initial sensory-motor conditions, or with prior warning of a perturbation all represent adjustments in ''central set'', defined as a modified neuromotor state due to changes in initial contexts (Prochazka, 1989). These changes in central set may involve the cerebral cortex, in which the cortex acts to prime postural response synergies accommodated within the brainstem, thereby optimizing postural responses for a given environmental context, while still allowing for the early response latencies that are necessary to recover equilibrium. In support of this hypothesis, pyramidal tract neurons recorded in the cat modulate their activity during postural perturbations, and this perturbation-associated activity becomes altered with changes in the cats' initial postural alignment (a change in central set; Beloozerova et al., 2005).

In humans, we recently found changes in cortical excitability just prior to anticipated postural perturbations, thereby supporting the hypothesis that cortical activity

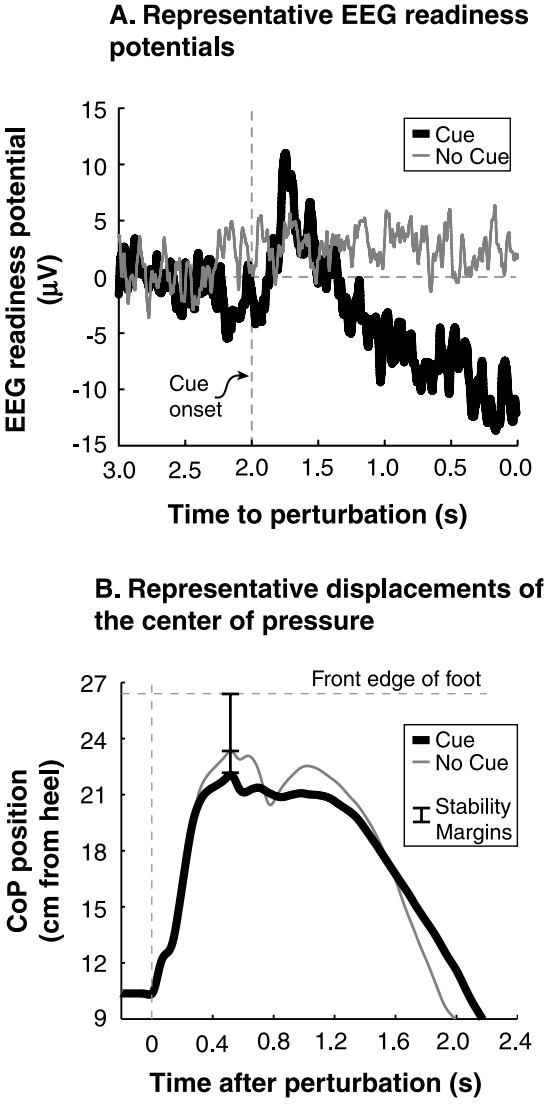

Fig. 2. Effects of prior knowledge of onset time of an upcoming surface perturbation on electroencephalographic readiness potentials and center of pressure responses from a representative healthy adult. **A** An EEG readiness potential shows slow negativity starting 500 ms after a visual cue and 1500 ms before a surface perturbation, seen only in the trials with a visual warning cue that turned on 2000 ms before the perturbation. **B** Center of pressure displacements show a larger distance between the maximum forward displacement of the center of pressure and the front edge of the foot (the stability margin) in trials with a cue compared to trials without a cue

can play a role in optimizing postural responses with changes in central set (Jacobs et al., 2007). Specifically, we found that, when subjects could anticipate the time of an upcoming perturbation, they exhibited a growing negative potential over their sensory-motor and supplementary motor cortex (Fig. 2), similar to ''readiness potentials'' that occur 1–2 sec prior to a voluntary postural movement (Saitou et al., 1996; Slobounov et al., 2005; Yazawa et al., 1997). Readiness potentials represent cortical activity related to movement planning and anticipation (van Boxtel and Brunia, 1994), and our results suggest that they serve as a cerebral correlate for response modifications mediated by changes in central set. Figure 2 illustrates, from a representative subject, an average EEG readiness potential from two conditions: (1) the Cue condition, in which the subjects could predict the time of the upcoming perturbation based on a light cue that turned on two seconds before translating the support surface, and (2) the No Cue condition, in which the subjects could not predict perturbation onset because no visual cue was provided and the time of perturbation onset was randomized. In addition, we found that changes in pre-perturbation cortical activity correlated with changes in postural stability margins (the difference between the peak displacement of the center of pressure and the location of the front edge of the foot), such that the subjects with the largest change in readiness potentials between the Cue and No Cue conditions showed the largest improvement in postural stability.

Thus, feet-in-place responses appeared to be optimized to maximize postural stability based on changes in central set. Although we did not allow subjects to modify their initial position in anticipation of an upcoming perturbation, we cannot completely rule out the possibility that the cortex sent a command (or efference copy) to stiffen or otherwise prepare muscle activation or that feedback from this motor command contributed to the recorded ''readiness potential''.

The cortex can influence central set for postural responses via two main loops, one including the cerebellum and one including the basal ganglia (Fig. 1). Studies suggest that the cerebellar-cortical loop is responsible for adapting postural responses based on prior experience and the basal ganglia is responsible for pre-selecting and optimizing postural responses based on current context.

The cerebellum is involved in adapting response magnitude and in tuning the coordination of postural responses based on practice and knowledge of results, just as it participates in the adaptation and coordination of all movement (Thach and Bastiaan, 2004). The cerebellum ensures that the magnitude of postural response is scaled appropriately, not only to current perturbation characteristics, but also based on the anticipated characteristics of an upcoming perturbation. Unlike healthy subjects, patients with cerebellar lesions are unable to scale the magnitude of their postural responses to predicable amplitudes of surface translations (Horak and Diener, 1994; Timmann and Horak, 1997) and, therefore, the cerebellum may be involved in the cortico-brainstem circuit responsible for modifying postural responses with changes in central set.

The basal ganglia are also likely included in the cortico-brainstem pathway that is activated by changes in central set. Indeed, dysfunction of basal ganglia due to Parkinson's disease leads to an inability to alter postural responses with changes in (1) initial support conditions, (2) the intention to respond with different strategies, or (3) perturbation direction (Beckley et al., 1993; Bloem et al., 1995; Chong et al., 2000; Nardone et al., 1990; Horak et al., 1992, 2005). For example, whereas healthy subjects change postural synergies immediately, in the first trial (when using their hands for support, when intending not to resist the perturbation, or when the direction of a perturbation changes from a linear translation to a rotation), postural synergies in subjects with Parkinson's disease do not change immediately, but require prior experience across several trials to be modified appropriately (Bloem et al., 1995; Chong et al., 2000; Horak et al., 1992). These results suggest that the basal ganglia-cortical loop is critical for pre-selecting a brainstem response synergy optimal for initial conditions, so an appropriate response can be rapidly triggered. This concept is consistent with our recent study suggesting that healthy people appear to select a stepping limb and step trajectory in advance of unpredictable surface perturbations, whereas subjects with basal ganglia deficits due to Parkinson's disease more often utilize online response selection because of an impaired ability to execute a pre-selected response strategy (Jacobs et al., 2005; Jacobs and Horak, 2006b). Therefore, the basal ganglia likely act as an intermediary between the cerebral cortex and brainstem for automating the selection and execution of a context-specific postural response (Grillner et al., 2005; Takakusaki et al., 2004).

In summary, the nervous system (including the cerebral cortex) normally makes a ''best guess'' about an anticipated postural perturbation and primes a contextually appropriate and experience appropriate postural response (located within the meso-pontine regions of the brainstem) before the response occurs. Then, if a perturbation does occur, and the response is initially inadequate to recover postural equilibrium, the cerebral cortex is again recruited during the late phases of the postural response in order to provide additional (more voluntary) postural adjustments.

Cortical loci involved in externally triggered postural responses

The specific loci of the cerebral cortex involved in externally triggered postural responses are still unclear and require further investigation. Studies investigating the effects of human cerebral lesions on posture suggest that perception of the visual vertical involves the insula (Brandt et al., 1994), and perceived gravitational vertical requires healthy function of the thalamus (Karnath et al., 2000, 2005), superior parietal cortex (Blanke et al., 2000; Johannsen et al., 2006), and insula (Johannsen et al., 2006). In addition, lesions of the temporal-parietal junction (a region of multimodal sensory integration) lead to poor equilibrium control on an unstable support (Perennou et al., 2000). These studies, however, primarily demonstrate that these regions of thalamus and cortex integrate sensory input for postural tasks, but do not focus on loci involved in motor output during externally triggered postural responses. It has been suggested, however, that vestibular and somatosensory input may be integrated within a distributed cortical network (including the temporal-parietal cortex, supplementary motor area, and prefrontal cortex) in order to process input related to self-motion and counteract a loss of balance (de Waele et al., 2001).

EEG potentials associated with postural perturbations in humans show a small, positive response over the primary sensory cortex at 40–50 ms after a perturbation, thought to be a primary sensory signal. The large, negative potential that arises 100–200 ms after any unpredictable, but not predictable, postural perturbation is largest over frontocentral regions, suggesting that the SMA and cingulate cortex are involved, probably with the cerebellum, in generating an ''error signal'' between expected and actual sensory information regarding postural status. The role and cortical sources of later, more variable, cortical potentials are largely unknown. When explicitly testing externally triggered postural responses in animals, lesioning the motor cortex of cats hinders compensatory stepping responses (Bard, 1933), and intra-cranial neural recordings in rabbit also suggest involvement of the primary motor cortex during feet-in-place postural responses (Beloozerova et al., 2003). In humans, transcranial magnetic stimulation of the primary motor cortex alters the late phase of feet-in-place postural responses (Taube et al., 2006). In addition, EEG readiness potentials that precede external postural perturbations (Jacobs et al., 2007) and perturbation-evoked potentials exhibit maximal amplitude at Cz (Dimitrov et al., 1996; Duckrow et al., 1999; Quant et al., 2004b), suggesting involvement of primary sensory-motor and supplementary motor cortex, but more explicit tests are required in order to localize the sources of these potentials. Together, these studies suggest that the primary motor cortex is likely involved in the generation of the late-phase, feet-in-place and compensatory stepping postural responses, whereas parietal, temporal and insula cortex are likely essential for sensory integration during postural tasks.

Conclusions

The cerebral cortex likely influences postural responses both directly via corticospinal loops and indirectly via communication with the brainstem centers that harbor the synergies for postural responses, thereby providing both speed and flexibility for pre-selecting environmentally appropriate responses to a loss of balance. The influence of the cerebral cortex on postural responses is still largely untested, and its influence may vary with context. While anticipated losses of balance allow for the pre-selection and optimization of postural responses (Ackermann et al., 1991; Ghafouri et al., 2004; Horak et al., 1996; Jacobs and Horak, 2006b; Zettel et al., 2005), the extent to which cortical preselection of postural responses also applies to entirely unexpected situations is unknown. For a truly unexpected loss of balance, the influence of the cerebral cortex may include either online activation for selecting and optimizing an appropriate response, or pre-selection to allow for optimized responses based on prior experience and current context. The occurrence of either of these options may further depend on the balance capability of a subject (e.g., a person with impaired balance may be incapable of rapidly selecting a context-appropriate response based on central set and, instead, may depend on using cortical loops during the late phases of the response in order to shape the postural response to environmental demands). Thus, in addition to the basic physiological question of whether or not the cerebral cortex contributes to postural equilibrium, further research is required to understand the role of the cerebral cortex in varying contexts: such as any changes that occur with dual tasking, while altering the predictability of postural perturbations and/or the intentions of the subject, and with age or disease.

We additionally propose that, in order to answer these questions, experiments should be directed to both animal and human models, with direct recordings of the activity of the cerebral cortex during postural tasks. Altogether, our understanding of the physiology that underlies postural equilibrium is still in its infancy, particularly with regard to the role of the cerebral cortex. Thus, with the current advances in cellular recording and neural imaging techni-

ques, more attention should be paid to this topic in order to better direct physical, pharmacological, and surgical therapies for those with impaired balance.

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