What If Low Back Pain Is the Most Prevalent Parkinsonism in the World?

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Low back pain (LBP) has a point prevalence of nearly 10% and ranks highest in global disease burden for years lived with disability; Parkinson's disease (PD) ranks in the top 100 most disabling health conditions for years lost and years lived with disability (1). Recent evidence suggests that people with chronic, recurrent LBP exhibit many postural impairments reminiscent of a neurological postural disorder such as PD. We compare and contrast postural impairments associated with LBP and PD in order to inform treatment strategies for both conditions. The literature suggests that both LBP and PD associate with impaired proprioceptive function, sensory orientation during standing balance, anticipatory postural adjustments, automatic postural responses, and striatal-cortical function. Although postural impairments are similar in nature for LBP and PD, the postural impairments with LBP appear more specific to the trunk than for PD. Likewise, although both health conditions associate with altered striatal-cortical function, the nature of the altered neural structure or function differ for PD and LBP. Due to the high prevalence of LBP associated with PD, focused treatment of LBP in people with PD may render benefit to their postural impairments and disabilities. In addition, LBP would likely benefit from being considered more than just a musculoskeletal injury; as such, clinicians should consider including approaches that address impairments of postural motor control.

Keywords: Parkinson's disease, low back pain, posture, anticipatory postural adjustment, postural response, balance

INTRODUCTION

Low back pain (LBP) represents one of the most prevalent health conditions worldwide, having a point prevalence of nearly 10% and ranking first in global disease burden for years lived with disability (1). Parkinson's disease (PD) also represents a significant health concern as the second-most prevalent neurodegenerative disease in older adults and ranking in the top 100 most disabling health conditions for years lost and years lived with disability (1). Although LBP is a musculoskeletal condition and PD is a neurodegenerative condition, both health conditions present with impairments of postural control and associated alterations of central neurophysiology. For both conditions, these postural impairments span multiple domains of postural control, including (a) reduced somatosensory perception and altered somatosensory integration for balance control; (b) excessive axial postural tone and stiffness; (c) delayed and non-specific anticipatory postural adjustments (APAs) to stabilize and facilitate voluntary movement; (d) non-specific and less effectual automatic postural responses (APRs) to external perturbations; and (e) slowness of walking and other activities. Further, altered structure and function of cortex and basal ganglia is evident for

both health conditions. The purpose of this perspective paper is to compare and contrast the postural impairments and related changes in neurophysiology associated with LBP and PD and to discuss the potential implications of their shared impairments on the treatment strategies for both health conditions.

SENSORY ACUITY, KINESTHESIA, AND DYNAMIC CENTRAL SENSORY INTEGRATION

Any type of physical activity optimally requires accurate sensation and perception of one's own position and movement (i.e., kinesthesia), and both PD and LBP associate with impaired kinesthesia. People with PD exhibit impaired tactile sensation and impaired kinesthesia to detect limb position during active motion as well as to detect passive limb and trunk rotation (2–8). People with LBP exhibit impaired two-point discrimination and can be unable to kinesthetically perceive their lumbar trunk based on body image traces (9). In contrast to the global somatosensory impairment exhibited by people with PD, the impaired tactile discrimination of people with LBP appears to be isolated to the area of the LBP (9). Impaired lumbosacral repositioning accuracy has also been reported for people with LBP (10). Further, similar to the impaired detection of trunk motion exhibited by people with PD, people with LBP exhibit increased thresholds for detecting passive trunk flexion and lateral bending (11). Thus, although the extent of impairment may differ between people with PD versus LBP, both health conditions associate with impaired tactile acuity and kinesthesia.

The act of maintaining standing balance requires integrating visual, somatosensory, and vestibular inputs. The central nervous system must also modulate each modality's influence on standing balance when transitioning to different sensory conditions. People with PD exhibit an impaired ability to limit postural sway during standing balance when somatosensory input is incongruent with visual and/or vestibular input (12, 13). Likewise, people with LBP also exhibit increased postural sway under conditions in which somatosensory input is incongruent with the other modalities (14). An enhanced use of ankle proprioception and the ankle strategy for postural sway, rather than a flexible control strategy to utilize trunk proprioception and hip motion under challenging conditions, has also been reported for people with LBP (15). These results suggest that, for people with LBP, the postural impairment may be localized to the processing of trunk proprioception and trunk control, with perhaps compensation through enhanced afferent processing and use of the distal limbs for the control of standing postural sway. Thus, people with LBP and PD alike exhibit an impaired ability to modulate the influence of surface somatosensory input in order to maintain standing balance.

MECHANICAL CONSTRAINT OF RIGIDITY

Rigidity (resistance to passive movement) is one of the cardinal symptoms of PD and can be evident across axial, proximal, and distal body segments. Although largely neural rather than peripheral in its generation (16), the rigidity associated with PD elicits a significant mechanical constraint that associates with impaired gait quality (17), turning (18), standing postural sway (19), and diminished quality of life (20). Direct measurement of axial rigidity by slow, passive trunk or hip rotation in stance demonstrates an increased rigidity with PD that correlates with clinical symptom scores (21) and with difficulty walking or rolling over (18). Thus, rigidity is a pervasive impairment in PD that influences mobility, balance, and daily life.

Axial or spinal-segmental rigidity is also common in people with LBP, and a change in LBP corresponds with a change in axial rigidity, but these results are not always consistent across studies (22). Although not the intent of a study by Cacciatore and colleagues (and therefore not powered to detect group differences), direct measurement of axial rigidity by slow, passive trunk and hip rotation during standing posture has been evaluated in people with and without LBP (23) using the same methods as those of Wright et al. (21) for people with PD. Cacciatore and colleagues reported nearly identical hip torques between a group of 8 people with LBP (mean ± SD = 3.06 ± 2.19) and a group of 15 control subjects without LBP (3.07 ± 1.66), but a statistically non-significant trend for increased trunk torque (6.26 ± 3.61 for LBP versus 5.00 ± 1.80 without LBP). Although requiring further study with a larger sample, the trend for increased mean rigidity with greater inter-individual variability in the group with LBP suggests that some, but not all, individuals with LBP exhibit axial rigidity (22). If the rigidity is evident, however, it is likely specific to the trunk. Therefore, although both LBP and PD have been associated with rigidity, this mechanical constraint is more consistent and pervasive for PD than for LBP.

ANTICIPATORY POSTURAL ADJUSTMENTS

Anticipatory postural adjustments represent learned, centrally programmed muscle activations of supporting body segments to counteract anticipated perturbing forces associated with voluntary movement in order to maintain posture and balance (24). Efficient movement thus depends upon appropriate movement-specific timing and amplitude of APAs.

For PD, impaired APAs appear evident across multiple tasks, such as step initiation and arm raising. During step initiation, for example, people with PD exhibit prolonged and diminished APAs that are poorly scaled to initial mechanical constraints (25, 26). During arm raises, people with PD exhibit APAs that can be delayed beyond a time window of anticipatory control prior to movement-related perturbation, and these delayed postural activations are not specific to the movement (27). Thus, PD associates with delayed, diminished, prolonged, and unspecified APAs across tasks that elicit APAs from axial or distal musculature.

For LBP, the primary impairment of the APA appears to be a delay in activation that can extend beyond a window of anticipatory control prior to movement-related perturbation (28, 29). Interestingly, similar to the findings on people with PD, people with LBP also exhibit a delayed APA that is not specific to the requirements of the movement (30). One notable difference,

however, is that the impairment is particularly limited to specific axial muscles and can actually be enhanced or earlier in onset at distal muscles (29). Thus, although delayed and contextually non-specific APAs are shared by both PD and LBP, prolonged duration and generalized impairment across body segments appears more evident with PD than with LBP.

AUTOMATIC POSTURAL RESPONSES

The ability to maintain balance and posture in response to an externally induced postural perturbation is also essential for efficiency and safety during daily activity. APRs represent rapid, automatic, but functionally specific responses to postural perturbations in order to maintain posture and balance.

For PD, the APR results in impaired stability marked by greater induced center-of-mass displacement and diminished corrective center-of-pressure displacement (31, 32). PD is also marked by impaired directional specificity of the APR, in which a non-specific stiffening strategy of antagonistic muscle co-contraction is evident (32, 33).

People with LBP exhibit remarkably similar impairments of the APR as people with PD, demonstrating increased center-of-mass displacements (34), muscle co-contraction (35, 36), and impaired directional specificity of the APR (37, 38). As previously described for both quiet stance and the control of the APA, however, people with LBP also exhibit a redistribution of control for the APR via compensation at distal body segments (36, 37, 39) that is not evident with PD. Thus, both health conditions exhibit diminished stability, co-contraction, and directionally non-specific APRs, but the impairment is more pervasive across body segments for PD, whereas the impairment appears localized to the trunk with compensation elsewhere for LBP.

BRADYKINESIA

Bradykinesia is a hallmark feature of parkinsonism. For people with PD, bradykinesia can span movements across body segments, such as finger tapping, smiling, and gait (40–42). For PD, bradykinetic gait is marked by slowed gait velocity, decreased step length, step asymmetries, and variability, and recent studies have also identified altered trunk coordination (42–44).

Interestingly, people with LBP also exhibit slowed gait velocity, decreased step length, step asymmetries, and altered trunk coordination (45–47). Further, as with PD, bradykinesia is not isolated to gait for people with LBP, as they also exhibit slowed trunk motion and lifting behaviors (48, 49). Although the extent of bradykinesia with PD appears greater than for people with LBP when evaluating differences compared to matched control subjects, both health conditions share similar characteristics of bradykinesia.

ASSOCIATED NEUROPATHOLOGY

Dysfunction of circuits involving the basal ganglia represents a hallmark pathophysiology associated with the development of motor symptoms in PD, which associates with clinical symptom severity, including postural instability and gait disturbance (50).

LBP also associates with pathology of the basal ganglia. The transition from acute to chronic LBP associates with diminished striatal gray matter across multiple nuclei as well as with increased functional connectivity between prefrontal cortex and the nucleus accumbens; this increased connectivity also correlated with reported pain intensity (51). In subjects with established chronic LBP, however, there are many associated changes in neural structures and functions that do not necessarily resemble those of PD (52), including non-overlapping regions of diminished cortical gray matter and increased striatal gray matter (53, 54). Thus, corticostriatal pathology may affect both PD and LBP, but the nature of the pathology is quite different.

Beyond the existence of corticostriatal pathology, more specific alterations of cortical neurophysiology during postural tasks are evident with both PD and LBP. As determined by repetitive transcranial magnetic stimulation, the prolonged APA durations of people with PD during step initiation associate with the function of circuits involving the supplementary motor area, and the influence of stimulation at the supplementary motor area on APA duration appears to increase with increasing disease severity (26). Further, prior to initiating an APA for step initiation, greater amplitudes of electroencephalographic (EEG) preparatory cortical potentials associate with increasing disease severity for people with PD (55). With regard to the APR, people with PD exhibit enhanced preparatory EEG potentials, and the modulation of these potentials associates with the extent of APR modulation between conditions of differing perturbation amplitudes (56). During walking, people with PD exhibit enhanced frontal lobe activity (57). In sum, the results suggest an enhanced influence of the cerebral cortex on postural control for people with PD.

People with LBP likewise exhibit evidence of an increased influence of the cerebral cortex on postural control. As determined by transcranial magnetic stimulation, larger areas of the transversus abdominus muscle's cortical representation correlate with the onset delay of that muscle's APA activation during an arm-raise task (58). People with LBP also exhibit increased preparatory EEG potentials (29) as well as an increased topographical area of the potentials (59) prior to arm raises that require an APA. Amplitudes of preparatory EEG potentials have also been reported to correlate with APA onset time for subjects with LBP when performing an arm-raise task (59). With regard to the APR, people with LBP exhibit increased amplitudes of evoked EEG potentials in response to postural perturbation, and the amplitude of these enhanced potentials correlated with evoked center-of-mass displacement as well as the subjects' reported pain-related disability and fear of physical activity (39). Therefore, people with PD and people with LBP exhibit altered cortical functions that significantly correlate with their postural behavior and clinical symptoms, and this altered cortical function suggests an increased influence of the cerebral cortex on postural control for both health conditions.

Given the complex systems that control posture and gait (60), these few neuropathological similarities do not demonstrate that they are necessary and sufficient to produce the shared motor behaviors of LBP and PD. Although isolated characteristics of LBP could also relate to isolated characteristics of other neurological

conditions to suggest other mechanisms of neuropathological involvement, we preliminarily argue that the similarities in overall presentation of posture and gait between LBP and PD are greater than for LBP with other neurological conditions (e.g., cerebellar, vestibular, peripheral neuropathy, stroke), which differ in sensory conditions of impaired standing balance, truncal rigidity, the contextual specificity, scaling, and timing of APAs and APRs, as well as parkinsonian versus ataxic, neuropathic, or hemiparetic gait patterns (61–64). Therefore, relating the control of posture and gait of LBP to that of PD appears more robust than other options.

LBP CONFOUNDS PD

Based on the above sections, many shared postural impairments exist for both PD and LBP. These shared impairments are so extensive that PD can be misdiagnosed as LBP (65). It is important to note, however, that LBP confounds PD, because LBP is often coincident with PD. In almost 30% of cases, LBP is an initial presenting symptom of PD (66, 67). In addition, the prevalence of LBP with PD is approximately 60–83% compared to approximately 25% in matched control subjects (68–70). Thus, it is possible that the postural impairments of PD are exacerbated

by the coexistence of LBP, and LBP may be exacerbated by the postural impairments of PD.

IMPLICATIONS FOR TREATMENT

Although multidisciplinary treatment strategies are espoused for both PD and LBP (71, 72), the conservative physical treatment of postural impairment differs considerably between these two conditions. First, despite the prevalence of pain with PD, pain is rarely a focus of treatment for people with PD (69, 70). The treatment of motor impairment, however, is more common for people with PD. For example, physical therapy is utilized by about 63% of cases with PD, and the majority of its use is focused on retraining gait, balance, and posture (73). In contrast, physical therapy is utilized by less than 20% of cases with LBP, and its use comprises approximately six visits that prioritize pain management, strength, and flexibility rather than gait, balance, and postural training (74, 75). Treatment outcomes for LBP with this approach have been variable, although the use of motor control retraining hasn't yet demonstrated superior treatment outcomes to general exercise (76). The lack of superior treatment outcomes for LBP with motor control retraining therapy, however, may be because the treatment does not adhere to principles of motor
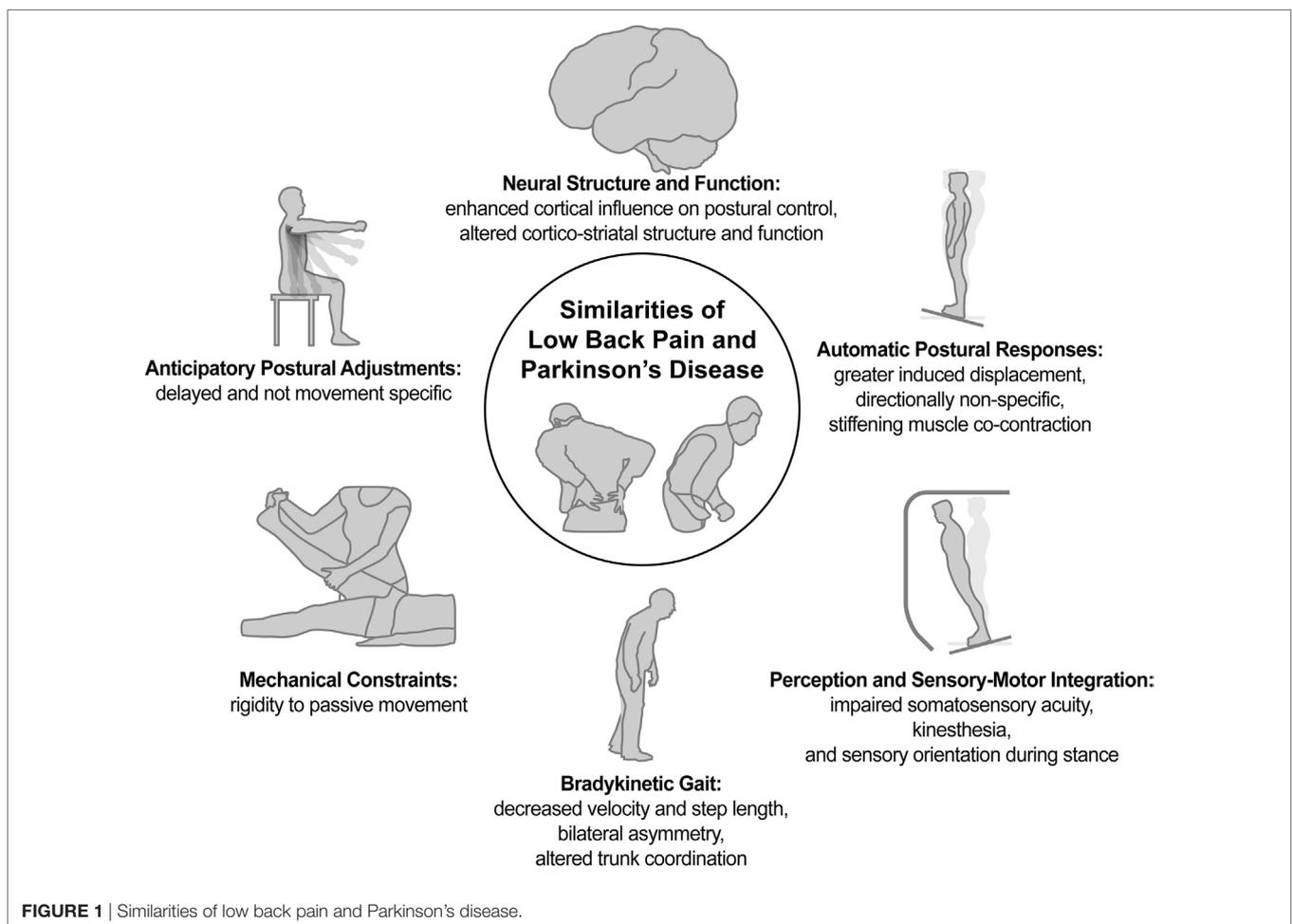

Neural Structure and Function:
enhanced cortical influence on postural control,
altered cortico-striatal structure and function

Anticipatory Postural Adjustments:
delayed and not movement specific

Similarities of Low Back Pain and Parkinson's Disease

Automatic Postural Responses:
greater induced displacement,
directionally non-specific,
stiffening muscle co-contraction

Mechanical Constraints:
rigidity to passive movement

Perception and Sensory-Motor Integration:
impaired somatosensory acuity,
kinesthesia,
and sensory orientation during stance

Bradykinetic Gait:
decreased velocity and step length,
bilateral asymmetry,
altered trunk coordination

FIGURE 1 | Similarities of low back pain and Parkinson's disease.

rehabilitation that have been more thoroughly researched and considered in practice for neurological rehabilitation (77).

Therefore, despite the many shared postural impairments between PD and LBP, as well as the high prevalence of LBP in people with PD, the treatment approaches of these two health conditions are highly divergent. The coexistence of LBP with PD suggests focused management of pain, strength, and flexibility could potentially, at least partially, help alleviate postural impairment with PD. Similarly, the shared motor impairments of LBP to PD suggests that the management of LBP could optimally include a postural motor retraining approach that is of sufficient focus and training exposure that motor patterns can be modified across multiple domains of postural control. Although important to substantiate mechanisms of pathology associated with LBP as an axial parkinsonism of postural tone and dynamic control, that substantiation does not preclude exploring postural motor retraining for LBP as a potential treatment to improve patient outcomes.

SUMMARY

Review of the literature indicates that both PD and LBP exhibit many shared impairments in postural control as well as some similar changes in neural pathology or function (**Figure 1**). Notably, for LBP (a) the impairments appear less pervasive and more localized to the trunk, (b) the impairments seem less consistent across individuals, and (c) despite some shared characteristics, the neural pathology is holistically of a different nature than for PD. Nevertheless, motor impairments seem more alike than different, suggesting that treatment strategies for LBP could benefit from those provided for PD, and treatment strategies that ameliorate LBP have the potential to benefit the treatment of motor dysfunction and lumbar pain in people with PD. Overall, the similarities of LBP and PD in postural impairment and associated neurophysiology suggest it may not be so implausible to consider LBP as an axial parkinsonism, rendering it the most prevalent parkinsonism in the world.

AUTHOR CONTRIBUTIONS

All authors contributed to generating the scientific arguments of this perspective paper. First draft was generated by JJ, and critical review and editing was provided by all other authors.

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