Pusher Syndrome- Stroke

Over 795,000 people experience a stroke each year in the United States1. A wide variety of neurological impairments come as a result with some more common than others. A less common deficit known as “pusher syndrome” or “contraversive pushing” occurs in about 10.4% of patients who experience stroke2,3,5. Most patients who demonstrate pushing behavior have a prolonged recovery time2,4. It is characterized by a postural deficit affecting the patient’s perceived midline which results in active pushing with the unaffected side toward the paretic side and refusal of weight acceptance on the unaffected side2. These patients do not attempt to correct loss of balance to the affected side which therefore highly increases their risk for falls and subsequent injury2,7. In a seated posture, the patient often pushes onto the wheelchair arm whereas in standing, the weaker side of the body is unable to control the body weight, leading to loss of balance and falls8. The natural response of a patient post-stroke is to shift their body weight toward the non-affected side in an effort to bias their body weight in a way that prepares them for potential loss of balance toward their affected side8. There is also a common occurrence known as the “listing phenomenon” in which patients appear to be pulled toward their hemiparetic side. These patients may also experience a loss of balance and fall toward their hemiparetic side; however, they are differentiated from those with ‘pusher syndrome’ by their absence of active and forceful abduction and extension of the unaffected side and resistance to any assistance to or across midline7,17. They also are aware of their loss of equilibrium and will learn to pull on objects to maintain balance instead of pushing3,7.

Pusher Syndrome is caused by a perceptual deficit of the orientation of one’s body in regards to the line of gravity. On average, these individuals perceive the vertical line of gravity to be about 18 degrees tilted from true vertical3,9. The pathophysiology behind this behavior is not fully known and currently debated throughout the available evidence. Researchers have been studying this population to determine if the impaired midline perception is a result of a visual perceptual deficit, vestibular impairment, single-sided neglect, anosognosia, aphasia, or apraxia. Karnath adopted the theory that humans have a second graviceptive system previously unknown to researchers9.

The relevant anatomy may help to enlighten practitioners to a better understanding of this phenomenon. Based on previous theories of thalamic involvement16, Karnath performed a study to evaluate the relationship between the anatomical thalamic region and pushing behavior. He examined 40 subjects with a diagnosis of right or left-sided thalamic stroke and found that there was a significant relationship between those with a large hemorrhagic stroke to the posterior thalamus and pushing behavior. 86% of those demonstrating Pushing had lesions in the posterior thalamus; whereas, only 23% of those without Pushers Syndrome had damage to this area6. This finding suggested that the posterior thalamus must be an area of the brain that helps to control body posture11. Those patients who did not present with pushing tended to have smaller ischemic infarcts in the anterior thalamus. The extent of weakness was also notably greater in patients with pusher’s syndrome than those without6. The region of injury also extended from the posterior thalamus into the posterior side of the internal capsule, which explains the severity of hemiparesis in patients with Pusher Syndrome16.The thalamus is the sensory relay system which provides information about visual, somatosensory, auditory, and taste to the cerebral hemispheres for processing8. Another anatomical region of interest was explained by Johannsen in her journal article that aimed to address those patients who demonstrated pushing behaviors without a thalamic stroke20. She examined 45 subjects who had experienced a cortical lesion that was not in the thalamus. Those patients who demonstrated pushing showed damage in portions of the postcentral gyrus and the insular cortex20.

Based on these anatomical findings, one would suppose that the hemorrhagic damage would occur along a pathway that included all three of these brain structures in such a way that brings clarity to the pathophysiology of this phenomenon. Unfortunately, the association is not that simple. It appears that sensory information does travel through the afferent fibers into the relay center at the thalamus and continue onto the region of the postcentral gyrus where the somatic sensory cortex is located; however, the insular cortex is uninvolved in this pathway8. Instead, sensory information also travels from the posterior thalamus into the insular cortex through a separate pathway8. We can conclude that each of these three regions of the brain have some participation in postural perception; however, a clear pathway between the three areas has not yet been discovered to my knowledge. Furthermore, these anatomical findings still leave unanswered the question of which area of the sensory cortex leads to this presentation and why?

One of the earlier theories suspected that Pusher Syndrome developed as a symptom of left-sided neglect, a common presentation in those with right hemispheric injury. These researchers thought that perhaps the problem was a spatial neglect of the left side of one’s body. One study found that in a sample of 15 patients with Pusher Syndrome, 80% demonstrated spatial neglect and impairment of the somatosensory system16. Karnath and colleagues rejected the idea of this being a presentation of a right hemispheric syndrome due to their findings. In his study he found that 35% of the patients with pusher behaviors presented with left-sided brain damage3,16. Similarly, in a study done by Pedersen et al, about 50% of the patients with pushing behaviors presented with left-sided brain damage4. Of those patients in Karnath’s study, none of those with left-sided damage were positive for tests of spatial neglect; instead, they presented with aphasia16. Therefore, it is clear that Pusher Syndrome is not only a disorder of the right hemisphere and thus does not occur due to spatial neglect3. It seems as though those with injury to the right hemisphere often experience spatial neglect and those with a left hemisphere injury often experience aphasia, however, neither of these presentations are associated with Pusher Syndrome but instead may occur in conjunction with it3. This may give clues to the location of the areas of the brain responsible for upright body orientation since they must be close to the regions responsible for aphasia in the left side of the brain and spatial neglect in the right side2.

Others have given credit to sensory and vestibular impairments as the cause of pushing. Perennou found that the lateral tilting was more pronounced in those without intact vision11. Davies even suggested that there was a complete lack of perceived sensation on the hemiplegic side including loss of tactile, proprioceptive, visual, and auditory perception2. However, Karnath found that the “visual vertical” was unaffected in patients with pushing behavior, but their posture was tilted in relation to gravity3. Karnath tested the patients’ visual and vestibular function by placing them in a dark room. A glowing pole was introduced to the patient at an angle and the patient was asked to adjust the rod so that it was vertical. The patients with Pusher Syndrome were able to achieve correct vertical within the same margin for error as the control subjects. They were also able to correct their posture to line up vertically with a simulated visual environment filled with vertical objects for visual cueing. These two results show that the vestibular and visual systems are intact in patients diagnosed with Pusher Syndrome11. Vestibular function was also tested using a lateral tilt chair. Those patients with diagnosed vestibular dysfunction such as vertigo or vestibular neuritis still subjectively reported a perceived postural vertical that was equivalent with the true gravitational vertical. Patients with Pusher Syndrome, on the other hand, showed a distinct tilt in their perceived postural vertical in comparison to gravitational vertical11. Therefore, they must be in constant internal conflict between the vertical objects understood by their visual and vestibular systems and their body’s perception of postural vertical3.

The most widely accepted theory explaining this pushing phenomenon is “graviceptive neglect” which is defined by Karnath as “a severe misperception of body orientation in relation to gravity”3. These patients seem to have all sensory capabilities intact, but the pathology occurs in the perception of upright body posture. This means that when a patient is seated and pushing to the hemiplegic side, they feel upright but visually see that they are tilted2,3. Karnath suggests, based on this assumption, that humans may have a distinctive pathway for the perception of body position in comparison to gravity that is separate from the visual-vestibular system11. Because of the commonalities in MRI imaging in these individuals, he believes that this pathway must run through the posterolateral thalamus and into the cortical region responsible for the body’s orientation to gravity3.

Based on this theory, one would expect that the patient would push toward the direction in which their perceived vertical was tilted; however, this is not the case. Karnath found that the patient’s perceived postural vertical is actually 20 degrees toward the same side as the lesion, opposite the direction of their push11. There are a few theories on why this presentation exists; however, my personal opinion is different from these. In the few patients that I have observed with this diagnosis, I have noticed that there is movement of the trunk and pelvis. The pelvis is lifted on the unaffected side in order to create a tilt of the pelvis in the direction of the hemiplegic side. This is consistent with Perennou’s observation of a pelvic tilt12 and Roller’s observation of a correct vertical head orientation despite a lateral body lean2. Additionally, the trunk is shortened on the unaffected side. My theory is that the patient is tilting at the pelvis and pushing in an effort to correct their posture to match the visual vertical while also shortening the trunk in order to remain comfortably in line with their perceived vertical.

Performing an examination of a patient with Pushers Syndrome requires careful attention and preparation. There are three assessment tools that are currently available for diagnosis and continued assessment of pushing behavior14. The Scale for Contraversive Pushing (SCP) was created based on the first definition of Pushing Syndrome by Davies. This scale gives one point for each of the three criteria: abnormal posture occurs during both sitting and standing, active pushing occurs with the non-affected side of the body via abduction and extension of the extremities, and the patient resists passive correction to or across midline. A score of 3 indicates that the patient does have Pushers Syndrome2,14. The Modified Scale for Contraversive Pushing (mSCP) scores the patient on their ability to do the following 4 tasks and they are evaluated on the amount of pushing that occurs in each scenario on a scale of 0-2: feet on the floor when in a seated position, posture fully erect during standing, keeping hip flexion during transfer from bed to chair, and transferring using a stand pivot technique. Scores range from 0 to 8 and the cut-off score is 3. The Burke Lateropulsion Scale (BLS) measures the patient’s resistance to passive postural correction. The patient receives a score of 0-3 based on the amount of resistance to vertical alignment he or she gives during the following activities: supine rolling, sitting, standing, transferring, and walking. A score of 0 is indicative of zero resistance and 3= severe resistance14.

The prognosis of Pusher Syndrome is encouraging. Most patients experience this type of behavior for a few months at most10,13. Danells findings showed that 79% of patients were unaffected after three months10 and Karnath found that almost all of his patients demonstrated no signs of pushing after 6 months13. Physical therapy may be beneficial to decrease this time until recovery. Unfortunately, patients with Pushers Syndrome usually have had a worse stroke than others without it as measured by lower neurological scores on the Scandinavian Stroke Scale and lower ability to perform ADLs according to the Barthex Index4,6. This is consistent with the statistic that demonstrating Pushers Syndrome prolongs recovery time by up to 63%.4 Bayber also found that those with pushing behaviors were more dependent at discharge and had a lower FIM score as compared to other patients with similar impairments15.

Due to the wide variety of theories about the causes and pathophysiology of this syndrome, there are several proposed methods of treatment. Paci and colleagues completed a case report on a 71 year old man with Pusher’s syndrome5. He received physical therapy for 2 hours twice a day for 3 weeks in which the therapist performed Bobath activities in the opposite directions of his lean. The therapist incorporated several types of sensory-involved treatment including tactile-somatosensory, auditory, and visual information in an effort to determine which treatment is most beneficial. The most beneficial results came from the sessions when the therapist gave visual or auditory feedback; however, there were no improvements shown after somatosensory feedback5. The earliest description of Pusher Syndrome was penned by Davies17. She recommends use of the Bobath concepts to passively provide manual adjustment to the patient’s posture to assume true vertical17. Karnath argues to say that visual feedback is most beneficial because the patient’s visual perceptions are not impaired so they are able to use their strength in vision to correct their weakness in other systems9. Carr and Shepherd suggest that passive positioning of the patient by the therapist may serve to further exacerbate their abnormal pushing behavior19. Davies’ suggested theory was also not beneficial for me in the clinic because the more I would push against the patient towards the correct midline posture, the harder the patient would push laterally in response.

Based on Karnath’s theory of graviceptive neglect, a therapist can take advantage of the fact that the patient has intact vision and encourage concentration on objects that demonstrate true vertical3. These activities should be done in an upright position such as sitting, standing, or walking but not supine3. For example, ask the patient to focus on chair legs and door frames and remind themselves of these objects being vertical. They can use these objects to compare what they see to what they perceive3,12. The goal is to increase the patient’s confidence by aligning their body’s vertical with their visual vertical while also noticing their safety and absence of loss of balance in this position18. The patient and therapist will work together to find the isolated movements necessary to assume the position that is equivalent with true vertical. Once this task is accomplished, the therapist can work with the patient on maintaining this position while completing other tasks in order to practice this posture during daily functional activities3. Similarly, Bohannon found success in teaching upright standing balance through practice and feedback18. The patient begins standing against a wall for balance assistance. Overtime, less assistance is given. He found that patients are able to achieve temporary independent standing without loss of balance after a 30 minute balance treatment18. Carr and Shepherd echo this suggestion, stating that intensive balance training has been anecdotally successful in the past19.

In a separate study done by Karnath, he found that though visual cueing was successful during treatment, the patients were unable to maintain the vertical position. According to this research article, visual cues alone are not enough to control upright body posture. Perhaps visual cue training PLUS something else such as cognitive retraining would be effective2. Perennou suggested the treatment option of transcutaneous electrical nerve stimulation (TENS) to the affected side neck to decrease one’s perception of incorrect vertical12. The appropriate settings are a short pulse duration and an intensity below motor threshold12.

Based on my speculated theory of the reason patients push, I think that a suitable treatment approach would be to encourage neutral pelvis positioning and lengthening of the shortened ipsilesional trunk. This can be accomplished by completing functional task-oriented activities that require centralization of body weight. Carr and Shepherd note that most treatment methods for pushers do not carry over into long-term learned behavior unless practiced in a task specific manner19. For example, the patient could reach across midline with their hemiparetic arm to grab a dish, transfer it to the other hand, and then reach up to a high shelf on the ipsilesional side to put it away. This accomplishes three benefits in one activity: reaching across midline to grab the plate shifts the weight back to center, bringing the pelvis down so that both ischial tuberosities are in contact with the seat; reaching up with the unaffected arm allows lengthening of the trunk on that side; and placing the plate on the highest shelf requires a contraction of the abdominal and oblique muscles on the paretic side to get the plate up high enough. Each of these actions are working together to re-establish balance and upright posture. Carr and Shepherd resonate with this idea as they mention a similar suggestion to use reaching with visual and verbal feedback19.

References

1. The Internet Stroke Center. Stroke Statistics. Stroke Center. Updated 2014. Accessed 2/4/14 at http://www.strokecenter.org/patients/about-stroke/stroke-statistics/.

2. Roller, ML. The ‘Pusher Syndrome’. Journal of Neurologic Physical Therapy. 2004;28(1):29

3. Karnath H, Broetz D. Understanding and Treating “Pusher Syndrome”. Journal of the American Physical Therapy Association. 2003;83(12):1119-1125.

4. Pedersen PM, Wandel A, Jorgensen HS, et al. Ipsilateral pushing in stroke: incidence, relation to neuropsychological symptoms, and impact on rehabilitation- the Copenhagen stroke study. Archives of Physical Medicine and Rehabilitation. 1996;77:22-28.

5. Paci M, Nannetti L. Physiotherapy for Pusher Behavior in a Patient with Post-Stroke Hemiplegia. Journal of Rehabilitation and Medicine. 2004;36:183-185.

6. Karnath HO, Johannsen L, Broetz D, Kuker W. Posterior Thalamic Hemorrhage Induces “Pusher Syndrome”. Neurology. 2005;65(6):819.

7. Chong D. Stop Pushing! Sharing Treatment Ideas for ‘Pusher Syndrome’. Physical Thearpy and Rehab Medicine. 2006;17(26):32.

8. O’Sullivan SB, Schmitz TJ. Physical Rehabilitation. 5th ed. Philadelphia: F.A. Davis Company; 2007: 722.

9. Karnath H, Ferber S, Dichgans J. The Origin of Contraversive Pushing: Evidence for a second graviceptive system in humans. Neurology. 2000;55:1298.

10. Danells CJ, Black SE, Gladstone DJ, McIlroy WE. Poststroke “Pushing”. Stroke. 2004; 35:2873-2878.

11. Karnath H. Pusher Syndrome- a frequent but little-known disturbance of body orientation perception. Journal of Neurology. 2007;254:514-424.

12. Perennou DA, Amblard B, Laassel EM, et al. Understanding the pusher behavior of some stroke patients with spatial deficit: a pilot study. Arch Phys Med Rehabil.2002 ;83:570–575.

13. Karnath H-O, Johannsen L, Broetz D,et al. (2002) Prognosis of contraversive pushing. J Neurol 249:1250–1253.

14. Santos-Pontelli T, Pontex-Neto OM, Leite JP. New Insights for a Better Understanding of the Pusher Behavior: From Clinical to Neuroimaging Features. Neuroimaging for Clinicians- Combining Research and Practice, Dr. Julio F. P. Peres (Ed.), ISBN: 978-953-307-450-4, inTech, accessed 2/4/14 at http://www.intechopen.com/download/get/type/pdfs/id/24729

15. Babyar, S. R., Peterson, M. G., Bohannon, R., Perennou, D., & Reding, M. (2009). Clinical examination tools for lateropulsion or pusher syndrome following stroke: a systematic review of the literature. Clin Rehabil, 23(7), 639-650.

16. Karnath HO, Ferber S, Dichgans J. The neural representation of postural control in humans. Proceedings of the National Academy of Sciences of the United States of America. 2000;97:13931-13936.

17. Davies PM. Steps to Follow: The Comprehensive Treatment of Patients with Hemiplegia. 2nd ed. New York: Springer-Verlag; 2000.

18. Bohannon RW. Ipsilateral pushing in stroke. Arch Phys Med Rehabil. 1996;77:524-525.

19. Carr J, Shepherd R. Stroke Rehabilitation Guidelines for Exercise and Training to Optimize Motor Skill. United Kingdom: Butterworth- Heinemann; 2008: 230-231.

20. Johannsen L, Broetz D, Naegele T, Karnath H. “Pusher Syndrome” Following Cortical Lesions that Spare the Thalamus. Journal of Neurology. 2006;253(4):455-463.